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2 Editorial

Abdul Abyad

Original Contribution / Clinical Investigation

3 Iran

Acupuncture in the management of multiple sclerosis - an experience from the field

Ebrahim Khoshraftar, Mahnaz Khatiban, Zahra Amini

6 Bangladesh

Cord prolapse: experience in a tertiary care hospital of Peshawar

Tehniyat Ishaq Khattak, Bilquis Afridi, Jamila Javaid Shah

12 Yemen

Prevalence of Metabolic Syndrome in Patients with Chronic Hepatitis C (CHC), Aden

Salem A Bin Selm

Clinical Research and Methods

16 Qatar

Treatment of refractory varicose vein ulceration by means of quadruple therapy (silver cell-hydro alginate , compressive bandaging, micronized purified flavonoid fraction and modest weight loss)

Dr. Hashim Mohamed, Mohamed H., AL-Maseeh F., Al-Lenjawi B., Al-Kozaaei D, Al-Bader A., Abdeen J.

Medicine and Society

21 Nigeria

Assessment of factors and conditions that influence HIV Positive Women's Rights to family resources in Abia State of Nigeria

Enwerej, E. E., Enwereji, K.O.

Case Report

28 Saudi Arabia

Endorphins and diabetes mellitus

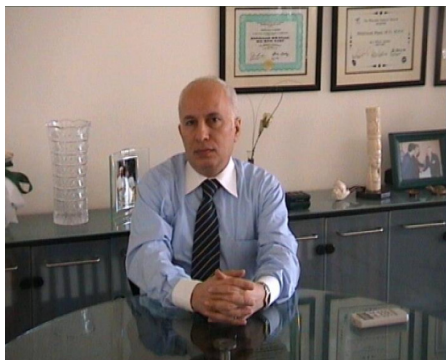
Almoutaz Alkhier Ahmed **47 Jordan**

33 Jordan

Warfarin-Induced Skin Necrosis: A rare but serious complication

Maher Hashem Al-Khateeb, Mohammed Nayef Al-Bdour, Waleed Ziad Haddadin

From the Editor



Abdulrazak Abyad
(Chief Editor)

This is the eighth year of the journal and the first issue this year. We appreciate all the people who have supported the journal over the years and who have made this journal one of the most read journals in the region and in a large part of the world. We receive papers from all over the World and this is why the journal name was changed to World Family Medicine and we look forward to more growth. The philosophy of the journal was and still is to help new authors and to open the door for developing countries in particular to voice their concerns regarding community and family medicine issues. We are indebted to the production team headed by Lesley for their support, in addition to the editorial board. Together we look forward to making the journal number one in the World.

In this issue a paper from Yemen looked at the Prevalence of Metabolic Syndrome in Patients with Chronic Hepatitis C (CHC). A total of seventy one patients with CHC were prospectively studied. The author noted that MS was found in 61.97% of cases. HOMA-IR was significantly higher in patients with CHC and MS vs those without MS. He concluded that CHC with MS was associated with a higher insulin resistance, and chronic hepatitis C has many features which suggest that this disease must be viewed not only as a viral disease, but also as a metabolic liver disease.

A paper from Nigeria looked at assessment of factors and conditions that influence HIV Positive Women's Rights to family resources. The authors stated that in developing countries, including Nigeria, cultural practices favour males in economic ventures, more than females. The authors studied

98 HIV positive women in a network of people living with HIV and AIDS and also 5 traditional rulers in charge of the communities studied. Finding showed that factors like widowhood inheritance, subordinate roles of women, breadwinner roles of men, terming women as visitors, and seeing women as responsible for the death of loved ones, negatively influenced rights to family resources of HIV positive women.

As high as 85(86.7%) of the women studied were denied rights to family resources. The authors recommended that regular seminars and/or workshops should be organized to educate the traditional rulers and others on the need to accord HIV positive women access to family resources so as to enable them cope with their health, economic and social needs and those of their children.

A prospective study from Bangladesh evaluated the frequency outcome and management of cord prolapse in a two year hospital based study. A total of 25 cases of umbilical cord prolapse were identified. The incidence of cord prolapse was 0.46% i.e. 1.6 per 300 deliveries. The authors concluded that cord prolapse is a major cause of perinatal morbidity and mortality. It can be reduced by regular antenatal checkups, early antepartum diagnosis of high risk cases, counselling during antenatal period for hospital delivery and short diagnosis delivery interval .

A paper from Iran looked at the effect of Acupuncture in the management of multiple sclerosis - an experience from the field. The authors report on one case with marked improvement in symptoms after treatment. The authors stressed that whilst the treatment did not cure the patient, it appears to have facilitated her movement and markedly improved her symptoms.

A paper from Hamad Medical Corporation in Qatar looked at treatment of refractory varicose vein ulceration by means of quadruple therapy (silver cell-hydro alginate, compressive bandaging, micronized purified flavonoid fraction and modest weight loss). The authors reported on treatment of an obese but otherwise healthy 38-year-old Egyptian male who presented with chronic superficial varicose vein ulceration of his right leg that had not responded to treatment over six years. An obese but

otherwise healthy 38-year-old Egyptian male presented with chronic superficial varicose vein ulceration of his right leg that had not responded to treatment over six years. After cleaning and light debridement the ulceration was treated with Nugel (Johnson & Johnson) and a silver cell dressing under three-layer bandaging including a carefully applied compression bandage. The dressing was changed every three days and there was complete resolution of the ulceration within four weeks. Complementary therapy involved initial bed rest with the limb elevated, counseling on necessary weight loss, and oral micronized purified flavonoid fraction (MPFF). The patient recovered gradually. The authors concluded that treatment of recalcitrant varicose vein ulcer is possible at primary care level .

A case report from Saudi Arabia looked at a case report on the use of a new device which arrived at the local anti-smoking clinic, called Silver Spike Point (SSP) that increases endorphins and helps people stop smoking.

A case report from Jordan looked at Warfarin induced skin necrosis which is a rare but serious complication of treatment with anticoagulants. The authors presented two cases and stressed that physicians should consider this reaction when suspicious skin lesions appear, regardless of the manner in which warfarin treatment was initiated. Early detection and proper management are essential.

Acupuncture in the management of multiple sclerosis - an experience from the field

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ABSTRACT

This experience from the field describes the use of acupuncture in the management of multiple sclerosis (MS) symptoms in a 36 year old female with 15 years history of MS. This condition is particularly difficult to treat whether using usual or complementary therapy. She reported rapid and significant improvement in her symptoms during a course of acupuncture treatment. Whilst the treatment did not cure the patient, it appears to have facilitated her movement and markedly improved her symptoms. There are few publications on acupuncture treatment in this condition. This experience from the field suggests that acupuncture may be a useful option in these patients.

Key words Multiple sclerosis; acupuncture

Introduction

This case concerns a 36 year-old female who first presented in 1993 with a severe common cold followed by diplopia, vertigo and nausea. Then, she also complained of right leg lameness, difficult in right hand grasp such as writing or holding things, and right arm launch. A progressive and chronic MS diagnosis was confirmed by Magnetic Resonance Imaging (MRI). Her medications before acupuncture were: Avonex, IVIG-g, Backlofen, Gabapentin, Clonazepam, Lorazepam, Q-10 Coenzyme, Metocarbamol in lumbar muscle spasm and chlordiazepoxide, botox treatment. The electric stimulations were used during her acute attacks.

At the same time different alternative treatments were pursued by the patient such as energy-therapy and Chinese electro-acupuncture. But she found them ineffectual. She and her family wished to avoid increasing her symptoms. All medications were maintained at the same level in the primary course of acupuncture treatment.

Physical Examination

She experienced a variety of severe symptoms despite her medications. Her symptoms included paraplegia with strict muscle weakness in legs, vertigo, visual problems, numbness and weakness with spasm in left hand, resistant spasm and clonus, Lhermitte's sign, frequency and polyuria and instability in the sitting position and some medication side effects. She remained severely fatigued with all persistent symptoms.

Treatment

The option of acupuncture was discussed with this patient and her family. They all consented. She reported being nervous about needles but was keen to try anything that might reduce her symptoms. The Korean acupuncture method (SU JOK) was selected for its simplicity, safety and efficiency.

The first treatment (in November 2005) consisted of needling at the brain and spinal cord meridians and the lumbar corresponding parts on hands. This treatment was repeated for all the subsequent treatments. The patient received a course of 12- 15 treatments with a 3 day interval over a period of 2.5 years with initial treatments being more closely spaced. All points were needed for 30 minutes using Chinese stainless steel sterile needles, 0.20mm diameter and 3 cm length. The needle appropriate length insertions depended on the place and purpose with no manual or electrical stimulation.

Results

There was an excellent response after the eighth treatment: her symptoms improved and the spasm of her left hand was gone. Her medicine was decreased after the eighth treatment. The improvement was sustained until the fourteenth treatment (January 2006), when she experienced leg muscle tonicity and felt well enough to restart work with her hands. She had extended her mobility because her instability in the sitting position decreased. She has been able to stand up with help of a hand and remain standing for 45 minutes with a walker stick without the knee brace. Then, she stopped

taking medications. Her symptoms and medical side effects decreased. Despite her history of paraplegia for 7 years, she also found movement in her little left toe.

Discussion

MS is a chronic, inflammatory, demyelinating disease that affects the central nervous system. Disease onset usually occurs in young adults, is more common in women, and has a prevalence that ranges between 2 and 150 per 100,000[1]. MS likely occurs as a result of some combination of both environmental and genetic factors [2]. MS affects the areas of the brain and spinal cord known as the white matter then it results in a thinning or complete loss of myelin. These lesions cause some of the neurological symptoms. Between attacks, symptoms may go away completely, but permanent neurological problems often persist [3]. The course of MS is difficult to predict and the disease may at times either lie dormant or progress steadily. In 1996 the United States National Multiple Sclerosis Society standardized the following four subtypes or patterns of progression definitions: relapsing-remitting, secondary progressive, primary progressive and progressive relapsing [4]. The prognosis of an individual patient is unpredictable [2].

The disease does not have a cure, but several therapies have proven helpful. Treatments attempt to return function after an attack, prevent new attacks, and prevent disability. During symptomatic attacks administration of high doses of intravenous corticosteroids is effective [5]. The treatment with interferons during an initial attack can decrease MS development [6]. As with any treatment, medications have several adverse effects.

Different alternative treatments are pursued by many patients. Examples are dietary regimens [7], herbal medicine [8] and general exercise [9]. Although, there are few publications on alternative treatment in MS, the acupuncture approach used here has not been reported previously.

Korean Su Jok acupuncture therapy is a new system of acupuncture using only the hands and feet to effect the same results as body acupuncture. Su Jok means "hand and foot". They represent a small mirror image of the anatomy of the human body (Fig 1). Su Jok Acupuncture is a general term describing this new system.

Su Jok Acupuncture is a two-dimensional system. The first dimension is physical treatment to give simple stimuli to the points in the hands or feet corresponding to the affected body parts. The second dimension draws on classical acupuncture. The classical 12 Main Meridians, the eight Extra Meridians, and their attendant points are represented on the hands and feet.

It is very difficult to predict an exact expected length of treatment. It depends on the duration of the disease. In Su Jok Acupuncture, response to treatment is usually immediate [10].

The experiences from the field, by their nature are anecdotal and improvement after treatment may be due to coincidental spontaneous improvement or expectation rather than the treatment. There are, however, several factors in this case that favor causality rather than coincidence. Firstly, in experience of this condition, spontaneous improvement or resolution is rare. The second is that this patient had definite symptoms with CNS affecting her movements and muscles tone particularly in her hands, legs and lumbar areas. So, it was attempted to needle in the brain, spinal cord and lumbar meridians. Most importantly, the patient remains well after putting by her medications.

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Korean Su Jok acupuncture therapy may offer benefits to chronic MS sufferers without other conventional and complementary therapies. It may provide an additional treatment option for patients unable to follow or maintain a common medical program. Nurses or paramedics can easily learn and apply the correspondence system of hand and foot without any side effects. In and

highly trained medical practitioners, Su Jok Acupuncture offers one of the best, most affordable and effective solutions.

References

1. Rosati G. The prevalence of multiple sclerosis in the world: an update. *Neurol Sci.* 2001; 22 (2): 117-39.
2. The Royal College of Physicians. Multiple Sclerosis. National clinical guideline for diagnosis and management in primary and secondary care. Salisbury, Wiltshire: Sarum ColourView Group. ISBN 1 86016 182 0. Free full text. (2004-08-13). Retrieved on 2008-03-15.
3. Brunner LS, Smeltzer SC, Suddarth DS, Bare BG. *Brunner and Suddarth's Textbook of Medical-Surgical Nursing.* 10th ed. Philadelphia: Lippincott Williams & Wilkins, 2003.
4. Lublin FD & Reingold SC. Defining the clinical course of multiple sclerosis: results of an international survey. National Multiple Sclerosis Society (USA) Advisory Committee on Clinical Trials of New Agents in Multiple Sclerosis. *Neurology* 1996; 46(4):907-11.
5. Brusaferrri F, Candelise L. Steroids for multiple sclerosis and optic neuritis: a meta-analysis of randomized controlled clinical trials. *J Neurol* 2000; 247 (6): 435-42.
6. Comi G, Filippi M, Barkhof F, et al. (2001). Effect of early interferon treatment on conversion to definite multiple sclerosis: a randomized study. *Lancet*; 357 (9268): 1576-82.
7. Farinotti M, Simi S, Di Pietrantonj C, et al. Dietary interventions for multiple sclerosis. *Cochrane database of systematic reviews (Online)* 2007; (1): CD004192.oi:10.1002/14651858.CD004192.pub2
8. Chong MS, Wolff K, Wise K, Tanton C, Winstock A, Silber E. Cannabis use in patients with multiple sclerosis. *Mult Scler* 2006; 12 (5): 646-51.
9. Oken BS, Kishiyama S, Zajdel D, et al. Randomized controlled trial of yoga and exercise in multiple sclerosis. *Neurology* 2004; 62 (11): 2058-64.
10. Jae PW. *The Six Energy Theory, the Illustrated Handbook,* Su Jok Academy, 2005

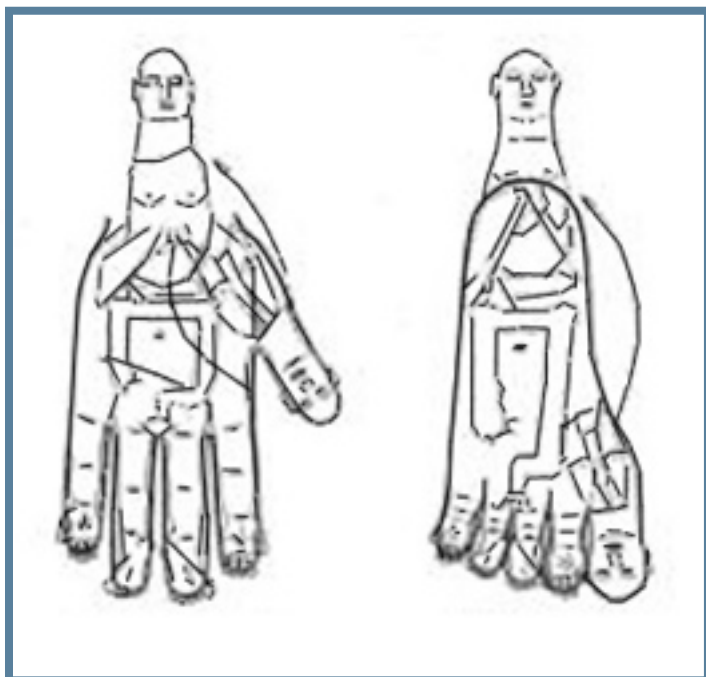


Figure 1: Su Jok



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Cord prolapse: experience in a tertiary care hospital of Peshawar

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ABSTRACT

Objective:

To evaluate the frequency outcome and management of cord prolapse in a two year hospital based study.

Study

Design, setting and duration: Prospective observational study was conducted; in Khyber Teaching Hospital, Peshawar from June 1995 to June 1997.

Materials and methods:

In this study 25 cases of umbilical cord prolapse were identified. The total number of deliveries were 4,650.

Patients presenting with cord prolapse were 25 cases. All the patients, booked or emergency, admitted with cord prolapse or developed cord prolapse after admission, whether overt or occult, irrespective of age and parity, term or preterm pregnancy, are included in the study. Patients with cord presentation are not included in the study. Data regarding age, parity, socio-demographic characteristics, booking status, referral source etc was collected on structured proformas and analysed with statistical software, SPSS version 13.

Results: Patients with cord prolapse were 25 giving an incidence of 0.46%. ie 1.6 per 300 deliveries. The majority of the patients in this series were non booked (92%). Only 8% patients were booked. There were 2 times as many multigravida and grand multigravida having cord prolapse as primigravida. 72% of the patients came in labour and the majority of them in second stage of labour. Frequency of cord prolapse was significantly higher in patients with abnormal lie (32%) followed by abnormal presentations e.g. breech (20%). 48% of patients were admitted with fetal distress. There were 6 stillbirths and 5 neonatal deaths.

In 68% we had to resort to emergency lower segment caesarian section to save the fetus and 12% had normal vaginal delivery. Outlet forceps were applied on 8%, 12% were delivered as assisted breech and only 4% had vacuum extraction.

Conclusion: We conclude that cord prolapse is a major cause of perinatal morbidity and mortality. It can be reduced by regular antenatal checkups, early antepartum diagnosis of high risk cases, counseling during the antenatal period for hospital delivery and short diagnosis delivery interval.

Key words: Umbilical cord prolapse, perinatal morbidity, perinatal mortality, short diagnosis delivery interval.

Materials and methods

This prospective descriptive study was conducted in Gynaecology and Obstetrics unit B of Khyber Teaching Hospital Peshawar from June 1995 to June 1997. All the patients (25) who were treated for umbilical cord prolapse during this period were enrolled in the study.

Diagnosis was made on the basis of clinical findings. The patients were treated according to the condition of the fetus and cervical dilatation and effacement. If the cord was found to be prolapsed and lying outside the introitus, it was checked for pulsations, was gently replaced into the vagina and dilatation of cervix was determined. If cervix was fully dilated and vaginal delivery was considered safe, it was done. If cervix was not fully dilated, immediate caesarian section was done, keeping the presenting part of the fetus off the cord until delivery of the fetus. Unnecessary handling of the cord was avoided to prevent spasm of the vessels and fetal distress.

If the fetus was dead, labour was allowed to continue unless contraindicated. If amniotomy was to be done, extreme care was taken to avoid dislodging the fetal head by applying a little fundal pressure. Fundal height was assessed and fetal heart sound recorded prior to and immediately after the procedure. Patient was discharged with advice to have an early antenatal booking and regular antenatal check ups in the next pregnancy and to have hospital admission in early labour to have a short diagnosis delivery interval.

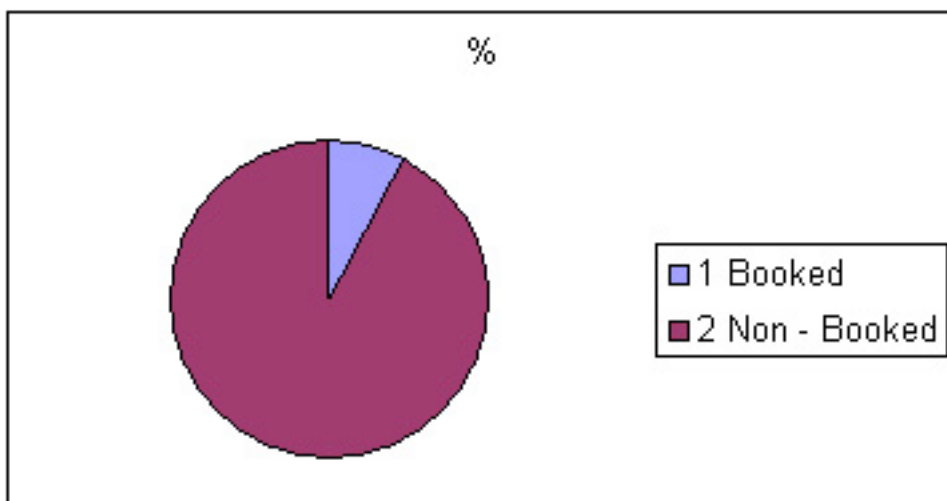
S. NO	Description	Number	%
01	Total Number of Obstetric Admissions	5,322	
02	Total Number of Deliveries	4,650	
03	Total Number of Umbilical Cord prolapse	25	0.46%

Table 1: NUMBER OF CORD PROLAPSE IN THE STUDY GROUP

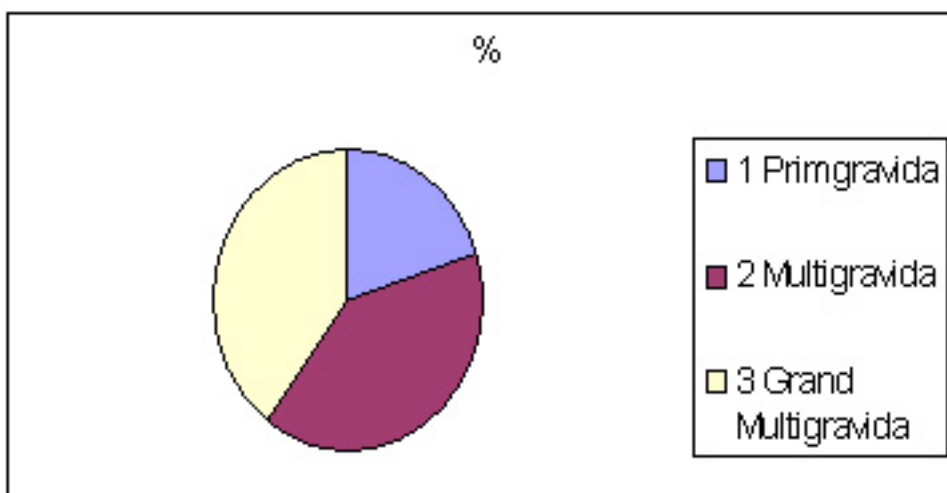
The majority of the patients in this series were non booked. Only 2 patients were booked out of 25 patients (8%). The non-booked patients were received via emergency or referred by the peripheral hospitals. Most of these patients were in labour at the time of admission. Among those who came with cord prolapse, the highest percentage was of those with overt compared with occult prolapse; the highest percentage of cord prolapse was noted in multi and grand multi gravidas (80%) compared with primary gravidas (20%).

In this series the most common associated factor was abnormal lie (32%). Out of these 87.5% of the patients had transverse lie and 12.5% presented with oblique lie. The second highest incidence was in premature labour. Five out of twenty five patients presented with abnormal presentation (3 as breech and 2 as compound presentation); flex breech was more commonly seen as compared with extended breech. Three out of twenty five patients had major degree placenta previa. Two patients had twins. In one patient cord prolapse was followed by amniotomy for induction of labour. (Table 2)

INCIDENCE IN PRIMIGRAVIDA AS COMPARED WITH MULTI & GRAND MULTIGRAVIDA

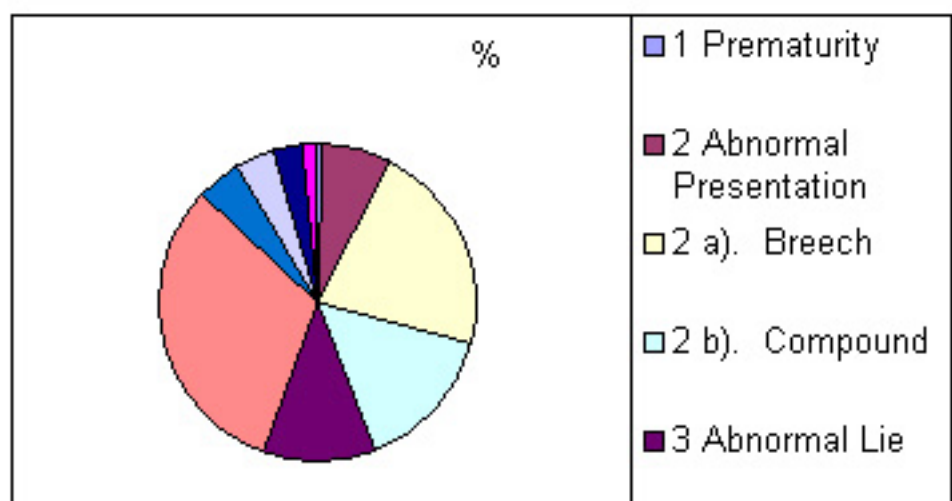


BOOKING STATUS OF THE PATIENTS



S. No.		Number	%
01	Prematurity	6	2
02	Abnormal Presentation	6	20%
	a) Breech	4	70%
	b) Compound	2	30%
03	Abnormal Lie	8	32%
	a) Transverse lie	7	87.5%
	b) Oblique lie	1	12.5%
04	Placenta Previa	3	12%
05	Twins	2	8%
06	Amniotomy	1	4%

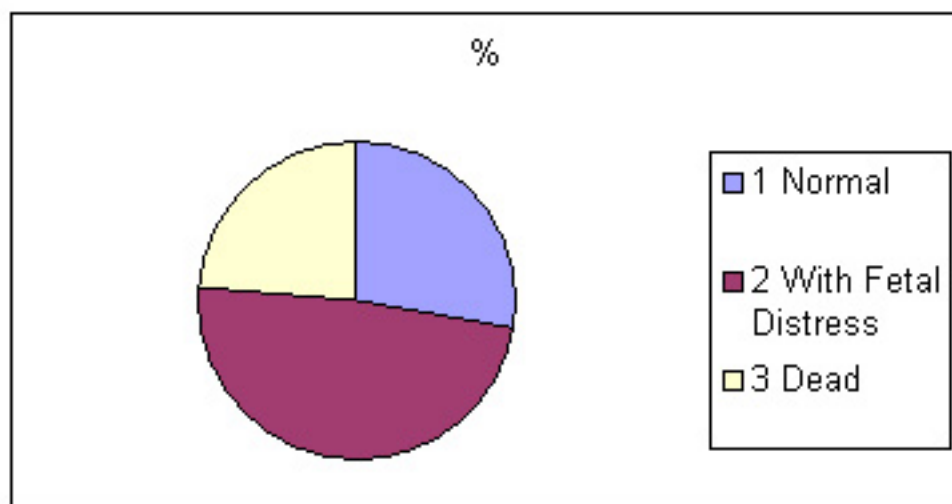
Table 2: PERCENTAGE OF PREDISPOSING FACTORS



As shown in Table 3, twelve out of twenty five (48%) fetuses came distressed. Although pulsation in the prolapsed cord was positive the fetal heart showed abnormal tachycardia or bradycardia. Seven out of twenty five showed normal heart rate and six were dead.

S. No.	Condition at Admission	Number	%
01	Normal	7	28%
02	With Fetal Distress	12	48%
03	Dead	6	24%

Table 3: CONDITION OF THE FETUS AT THE TIME OF ADMISSION



S. No.	Mode of Delivery	Number	%
01	Caesarean Section	16	64%
02	Normal Vaginal Delivery	3	12%
03	Forceps Delivery	2	8%
04	Vacuum Extraction	1	4%
05	Assisted Breech Delivery	3	12%

Table 4: MODE OF DELIVERY

Twenty eight percent (7 out of 25) when they came in with cord prolapse were not in labour. Fifty two percent (13 patients) came in the first stage of labour and twenty percent (5 patients) were in the second stage of labour at the time of admission.

Condition of the patients at time of admission

Sixteen patients were managed by caesarian section. Three out of 25 had normal vaginal delivery. Outlet forceps were applied on two patients and three were delivered as assisted breech and only one patient had vacuum extraction as shown in Table 4

Table 5 and 6 (next page) show the Apgar score of the delivered fetuses and perinatal deaths respectively. The majority of the fetuses that died, were stillborns. Five out of eleven died during the first week of life due to neonatal complications like neonatal sepsis and birth asphyxia. The majority of the stillborns were premature and prematurity itself is a major cause of perinatal morbidity and mortality.

Discussion

Umbilical cord prolapse is an obstetrical emergency during pregnancy or labour that endangers the life of the fetus. This is associated with anything that prevents the presenting part from fitting closely into the lower uterine segment and thus shutting off the fore waters from the hind waters. Such cases include transverse lie, breech presentation especially with flexed legs, when the risk is trebled³. In one study in over 53% of cases a malpresentation was present⁷. Umbilical cord prolapse demands immediate attention. Delay in management is associated with significant perinatal morbidity and mortality⁸ due mainly to prematurity and birth asphyxia and occasionally congenital anomalies⁹. Four fifths of the cases occurred in multiparous patients especially in higher parities. Forewater amniotomy or manual rotation prior to forceps extraction has been responsible for up to 20% of umbilical cord prolapse in various series but in large series, risk of amniotomy appears small.¹⁰ It occurs when the baby's umbilical cord falls into the birth canal ahead of the baby's head or other parts of the baby's body.

When the cord is compressed or squeezed e.g. between the baby and the wall of the uterus or vagina, the baby's supply of blood and oxygen is cut off which can lead to severe brain damage or death if the problem is not taken care of within minutes.

If the accident occurred outside the hospital, many babies would be dead or severely asphyxiated upon arrival in the hospital. Previous reports suggested that even if the neonates were delivered immediately after cord prolapse, the complication rate remained elevated. This is related to the fact that fetal prematurity and congenital anomalies are major contributory factors. In our series 6 babies were delivered before 37 completed weeks of pregnancy.

Some epidemiological studies have shown that the incidence of cord prolapse has remained stable through the years with the quoted rate of between 1 in 200 and 1 in 700. Our rate of 1.6 in 300 deliveries is in this quoted range. There is however conflicting evidence as to whether the

S. No.	APGAR Score	Number - 25	%
01	At 1 minute		
	0 - 3	11	44%
	4 - 7	11	44%
	8 - 10	3	12%
02	At 5 minutes		
	0 - 3	8	32%
	4 - 7	12	48%
	8 - 10	5	20%

Table 5: APGAR SCORE OF THE FETUSES DELIVERED

S. No.	Type of Death	Number	%
01	Still birth	6	54.6%
02	Neonatal Death in 1st week of life	5	45.4%

Table 6: TYPE OF PERINATAL DEATH

fetal outcome is actually improved with better obstetric care 11,12,13 . We believe that the neonatal outcome is improved with the practice of immediate caesarian section. In our study there were 5 stillbirths and 6 babies died during the first week of life in the neonatal care unit; the majority of them due to prematurity and low level of neonatal care facilities. The majority of the stillbirths were due to referrals from remote areas either mishandled by unqualified birth attendants or reached late and the fetal demise already occurred in utero or the fetus was severely asphyxiated.

The immediate management of umbilical cord prolapse is determined by 3 factors: fetal viability, fetal maturity and presence of any lethal fetal anomalies. Emergency delivery is recommended for a normally formed and sufficiently mature fetus. In the first stage of labour, a caesarian section is the only way to achieve early delivery, however with a completely dilated cervix, the obstetrician has a choice between instrumental vaginal delivery and caesarian section. Several studies have quoted more favourable outcomes with caesarian section even in the second stage of labour. 14

Upon diagnosis of umbilical cord prolapse, various manoeuvres have been advocated to alleviate pressure on the prolapsed cord. We found that digitally elevating the presenting part was quicker and the most important component in addition to other methods described in the literature such as urinary bladder distension with saline, pelvic elevation or tocolysis.¹⁵

The German Society of Gynaecology and Obstetrics recommends a decision to delivery time of less than 20 minutes. The American College of Obstetricians and Gynaecologists believes a decision to incision time of 30 minutes is appropriate. We believe that this rapid decision to delivery interval contributes to reducing the morbidity of the cord prolapse. In our study of 25 cases, the main cause for delay was a logistic problem in preparing an operating theatre.

In our series, a predisposing factor was present in the vast majority of cases as seen in the table above. These are abnormal lie, malpresentation, prematurity, multiple pregnancy, polyhydramnios.¹⁶ There is a lack of consensus as to whether obstetric interventions are associated with higher risk of cord prolapse ^{17,18} In our series only

4% of the patients had cord prolapse following an amniotomy.

When a patient has spontaneous rupture of membranes or an ominous cardiotocographic tracing, immediate vaginal examination enables umbilical cord prolapse to be diagnosed.

In addition patients should be educated on the early signs of labour or pre-labour rupture of membranes so that they come to the hospital early for supervised delivery, as early delivery can make a difference between life and death for the baby.

Conclusion

Prolapse of umbilical cord is an obstetrical emergency with a well documented grave fetal prognosis in the literature. A high index of suspicion and recognition of predisposing factors may allow for early detection and timely delivery, thereby minimizing perinatal morbidity and mortality. More and more stress on regular antenatal checkups and supervised hospital delivery is also mandatory. A multidisciplinary approach to the organization of an emergency caesarian section is essential to allow the rapid and safe conduct of an emergency caesarian section to minimize maternal and fetal risks in such an

emergency that threatens the life and well being of the fetus and indirectly of the mother. Immediate delivery is the ideal if the fetus is alive and sufficiently mature.

References

- 1) Lin MG. Umbilical cord prolapse. *Obstet Gynecol S URV* 2006;61:269-77.
- 2) Dufour P, Vinatier D, Bennani S , Tordjeman N, Fondras C, Monnier JC et al .Cord prolapse. .Review of the literature. A series of 50 cases. *J Gynaecol Obstet Biol Reprod (Paris)*1996;25(8):841-5
- 3) Murphy DJ,MacKenzie IZ.The mortality and morbidity associated with umbilical cord prolapse.*Br J Obstet Gynaecol* 1995Oct;102(10):826-30.
- 4) Savage E.W.Kohl S.G. and Wunn R.M (1970) Prolapse of the umbilical cord. *Obstet Gynaecol NY* 36,502-9.
- 5) Pahak UN. Presentation and prolapse of the umbilical cord. *Am.J.Obstet Gynecol* 1968;101:401-5.
- 6) Clark D.O, Copeland W.and Ullery J.c.(1968).Prolapse of the umbilical cord. *Am J Obstet Gynecol* 101,84-90.
- 7) Jacobson T,Madsen H. Unexpected survival after conservative management of cord prolapse I two very preterm babies. *Acta Obstet Gynaecol Scand* 1990;69:663-4.
- 8) Dutour P, Vinatier D, Bennani S, Tordjeman N, Fondras C, Monnier JC et al .Cord Prolapse. Review of the literature. A Series of 50 cases. *J Gynecol Obstet Biol Reprod (Paris)*1996;25(8): 841-5.
- 9) Murphy DJ,MacKenzie IZ. The mortality and morbidity associated with umbilical cord prolapse.*Br J Obstet Gynecol* 1995 Oct,102(10):826-30
- 10) Ekwepu CC. Cord prolapse through a fenestration in a caesarian section scar .*East Afr Med J* 1977;54:692
- 11) Y la-Outinen A, Keinonen PK, Tuimala R. Predisposing and risk factors of umbilical cord prolapse . *Acta Obstet Gynecol Scand* 1985;64(7):567-70
- 12) Prabulous AM, Philipson EH. Umbilical cord prolapse. Is the time from diagnosis to delivery critical ? *J Reprod Med* 1998 Feb;43(2):129-32
- 13) Koonings PP,Paul RH,Campbell K. Umbilical cord prolapse. A contemporary look. *J Reprod Med* 1990 Jul;35(7):690-2
- 14) Critchlow CW, Leet TL, Benedetti TJ, Daling JR. Risk factors and infant outcomes associated with umbilical cord prolapse: A population-based case-control study among births in Washington State. *Am J Obstet Gynecol* 1994 Feb;170(2):613-8
- 15) Katz Z, Shoham Z, Lancet M , Blickstein I, Mogilner BM, Zalel Y. Management of labor with umbilical cord prolapse: A 5-year study. *Obstet Gynecol* 1988 Aug;72(2):278-81.
- 16) Migliorini GD, Pepperell RJ. Prolapse of the umbilical cord: a study of 69 cases. *Med J Aust* 1977 Oct 15 ;2(16):522-4.
- 17) Usta IM , Mercer BM, Sibai BM. Current obstetrical practice and umbilical cord prolapse. *Am J Perinatol* 1999;16(9):479-84.
- 18) Roberts WE, Martin RW, Roach HH, Perry KG Jr, Martin JN Jr, Morrison JC. Are obstetric interventions such as cervical ripening, induction of labour, amnioinfusion, or amniotomy associated with umbilical cord prolapse? *Am J Obstet Gynecol* 1997 Jun;176(6):1181-3.

Prevalence of Metabolic Syndrome in Patients with Chronic Hepatitis C (CHC), Aden

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ABSTRACT

BACKGROUND AND OBJECTIVES:

Several investigators have suggested insulin resistance overload as a possible explanation for the increased prevalence of metabolic syndrome among patients with chronic hepatitis C virus (CHC) infection. Therefore, I performed this study to explore the relationship between CHC and the metabolic syndrome and evaluate the value of insulin resistance as a marker for risk factors in patients with chronic hepatitis C (CHC), according to the presence or absence of metabolic syndrome (MS).

PATIENTS and METHODS:

Seventy one patients with CHC were prospectively studied. Parameters of MS according to the IDF criteria were evaluated. Insulin resistance (IR) was established by homeostasis model assessment (HOMA-IR). An index 2.0 was designated as IR.

RESULTS:

MS was found in 61.97% of cases. HOMA-IR was significantly higher in patients with CHC and MS vs those without MS (7.88 ± 1.11 vs 4.29 ± 0.5 , $p=0.023$).

CONCLUSIONS:

CHC with MS associated with a higher insulin resistance, and chronic hepatitis C has many features which suggest that this disease must be viewed not only as a viral disease, but also as a metabolic liver disease.

Key words:

Chronic hepatitis C - metabolic syndrome - insulin resistance - Aden.

Introduction

Hepatitis C virus (HCV) infection is not confined to the liver, but can induce disturbances in many other organs and systems 1, 2. Chronic hepatitis C has many features which suggest that this disease must be viewed not only as a viral disease, but also as a metabolic liver disease which implies: insulin resistance (IR) 3, high prevalence of steatosis 4, increased prevalence of impaired glucose tolerance 5, type 2 diabetes mellitus 6, and changes in lipid metabolism 7. These findings together suggest that chronic HCV infection is closely related to the metabolic syndrome (MS). Accordingly, CHC should be divided into CHC with and CHC without MS. Metabolic steatosis occurs in non-3 genotype HCV infection and is associated with host metabolic factors: elevated body mass index (BMI) and central adiposity 8. Insulin resistance is the main feature of the MS. In CHC, there is a close association between IR [9], hepatic steatosis 3, 8, 9, progression of fibrosis 10 and a lower rate of sustained virological response 11,12. The pathogenetic mechanisms of metabolic steatosis are the IR induced by direct action of HCV on the insulin signaling pathways 1, 2 as well as the host factors, especially obesity 9. The IDF consensus worldwide definition of the MS was used. It implies the presence of the central obesity (defined as waist circumference ≥ 94 cm for men and ≥ 80 cm for women) plus two of the following four features: raised triglyceride levels ≥ 150 mg/dl; reduced HDL-cholesterol < 40 mg/dl in males and < 50 mg/dl in females; raised blood pressure: systolic ≥ 130 or diastolic ≥ 85 mmHg; raised fasting plasma glucose ≥ 100 mg/dl 13. To my knowledge there are no studies from Aden who have reported on this subject up to now, so in this direction I tried to determine the prevalence of metabolic syndrome among patients with chronic hepatitis C in Aden.

Methods

A number of 71 consecutive patients with CHC were prospectively evaluated. CHC infection was defined by the presence of anti-HCV for at least 6 months and a positive HCV-viremia. Patients with other etiology of chronic liver disease: hepatitis B, autoimmune liver disease, Wilson disease, hemochromatosis, α 1-antitripsin deficiency, patients with a history of hepatotoxic- or steatosis-inducing drug use. Patients with chronic alcohol consumption, as well as those with a history of diabetes mellitus were excluded from the study. All patients underwent a complete clinical and anthropometric evaluation, and an ultrasound scan of the liver, with a HS 2000 device, using a 3.5 MHz convex probe, and the presence of fatty liver was defined as the increased echogenicity with a bright pattern of the hepatic parenchyma and posterior attenuation. The five components of the MS were searched for in all patients, and subjects having 3 or more of the following criteria were labeled as MS: central obesity (waist circumference ≥ 94 cm for men and ≥ 80 cm for women) or body mass index (BMI) (weight in kilograms divided by the square of height in meters) was considered as obesity (BMI ≥ 30), plus any two of the following four factors: triglyceride levels >150 mg/dl or current use of fibrates; HDL-cholesterol < 40 mg/dl (men) and < 50 mg/dl (women); arterial pressure $\geq 130/85$ mmHg or pharmacologically treated; fasting glucose ≥ 100 mg/dl. The laboratory evaluation included measurement of the fasting blood glucose, fasting serum triglycerides, high-density lipoprotein cholesterol (HDL-C) levels, alaninaminotransferase (ALT) and aspartate aminotransferase (AST). Serum glucose, triglycerides, ALT, AST and HDL-C were measured by enzymatic colorimetric methods, and insulin resistance was established by homeostasis model assessment (HOMA-IR), by the formula:

fasting insulin level (mUI/l) x fasting glucose level (mg/dl) / 405. A HOMA-IR index value of more than 2.0 was considered as the criterion of insulin resistance

Viral markers HBsAg and anti-HCV were assessed using second-generation enzyme-linked immunosorbent assay (ELISA) tests.

Ethically a written informed consent was obtained from each patient.

Statistical Analysis

Comparison between groups was performed using Student's t-test for continuous variables and χ^2 test for categorical variables. The odds ratio (OR), the 95% confidence intervals (CI), and p values were calculated. A p value < 0.05 was considered significant.

Results

According to the presence or absence of MS the patients were divided in two groups for comparison (Table 1). In the univariate analysis, 9 variables were significantly related to the Metabolic Syndrome associated with CHC: female gender, increased BMI, visceral obesity, serum triglycerides, fasting glucose, HOMA-IR, and presence of fatty liver (NAFLD), and the prevalence of metabolic syndrome, obtained was 61.97% (44/71 cases), as illustrated in Table 1 (next page).

Discussion

Hepatitis C and Metabolic Syndrome are common conditions worldwide and both have IR as a key pathogenetic factor 14. In this study we found that Metabolic Syndrome, according to the IFD definition was present in 61.97% (44 out of 71) patients with CHC. All of them had visceral obesity, evaluated by waist circumference and a significantly higher BMI as compared with patients without Metabolic Syndrome. Low HDL-cholesterol level (68.4%), raised plasma glucose (59.8%), elevated blood pressure (48%) and high triglyceride levels (30.2%) were also present in these patients.

Chronic hepatitis C and Metabolic Syndrome may coexist in the same individual 15, but chronic HCV infection can also generate by itself some metabolic abnormalities characteristic for the Metabolic Syndrome.

Insulin resistance in chronic HCV infection could be caused by interplay between viral and host factors 16. HCV infection per se generates multiple defects in hepatic insulin signaling pathways 17, 18, 19. In this study insulin resistance was higher in patients with CHC and Metabolic Syndrome than in those without it, and in the univariate analysis HOMA-IR was correlated with BMI and visceral obesity. Visceral obesity estimated by waist circumference is viewed as the phenotypic expression of IR 20 and we found that HOMA-IR was almost two-fold higher in patients with CHC and MS than in those with CHC alone.

Our study revealed a positive correlation between IR and activity. Most investigators have demonstrated that IR has developed before the stage of cirrhosis and that it is higher in patients with CHC 21, 22, and suggested the link between IR and hepatic steatosis. In concordance with these studies, we found that Metabolic Syndrome correlated with NAFLD in large number. This might be explained by a higher contribution of the metabolic versus viral factors in this study, as in other studies 23. Another unexpected finding of our study was the correlation of IR with the necroinflammatory activity. This is not a singular finding. Another study found an association between IR, high serum viral load and necroinflammation in patients with CHC infected especially with genotype 1 or 4 24. Despite the major role played by HCV in the development of IR and hepatic steatosis, host metabolic factors might have a great contribution in chronic HCV infection. A significant number of our patients had Metabolic Syndrome, and visceral obesity was the constant criterion for the definition of Metabolic Syndrome. The adipose tissue is no longer considered only as a storage organ, but rather a very active neuroendocrine organ, that produces and secretes a large number of active peptides, collectively named adipocytokines or adipokines 25, 26, 27, with significant implications in several metabolic processes. Among these cytokines, the role of adiponectin in NAFLD and CHC has been largely studied. These findings support the hypothesis that IR is not only the result of a direct action of the virus, but also of an imbalance of adipocytokines, mainly in patients with Metabolic Syndrome, confirming the role of the metabolic factors in modulating insulin sensitivity 21,22. Our study revealed a positive correlation between presence of metabolic syndrome and NAFLD, in

percentage of 63% among patients with CHC, and this result might explain the role played by CHC in the development of fatty liver.

In conclusion CHC with Metabolic Syndrome was associated with a higher insulin resistance, and these findings together suggest that chronic HCV infection is closely related to the metabolic syndrome.

References

1. Koike K. Hepatitis C as a metabolic disease: Implication for the pathogenesis of NASH. *Hepato Res* 2005; 33: 145-150.
2. Narita R, Abe S, Kihara Y, Akiyama T, Tabaru A, Otsuki M. Insulin resistance and insulin secretion in chronic hepatitis C virus infection. *J Hepatol* 2004; 41: 132-138.
3. Fartoux L, Poujol-Robert A, Guécho J, Wendum D, Poupon R, Serfaty L. Insulin resistance is a cause of steatosis and fibrosis progression in chronic hepatitis C. *Gut* 2005; 54: 1003-1008.
4. Adinolfi LE, Durante-Mangoni E, Zampino R, Ruggiero G. Review article: hepatitis C virus-associated steatosis - pathogenic mechanisms and clinical implications. *Aliment Pharmacol Ther* 2005; 22(suppl 2): 52-55.
5. Lecube A, Hernandez C, Simó R, Esteban JI, Genesca J. Glucose abnormalities are an independent risk factor for non-response to antiviral treatment in chronic hepatitis C. *Am J Gastroenterol* 2007; 102: 2189-2195.
6. Mehta SH, Brancati FL, Sulkowski MS, Strathdee SA, Szklo M, Thomas DL. Prevalence of type 2 diabetes mellitus among persons with hepatitis C virus infection in the United States. *Ann Intern Med* 2000; 133: 592-599.
7. Perlemuter G, Sabile A, Letteron P, et al. Hepatitis C virus core protein inhibits microsomal triglyceride transfer protein activity and very low density lipoprotein secretion: a model of viral-related steatosis. *FASEB J* 2002; 16: 185-194.
8. Adinolfi LE, Gambardella M, Andreana A, Tripodi MF, Utili R, Ruggiero G. Steatosis accelerates the progression of liver damage of chronic hepatitis C patients and correlates with specific HCV genotype and visceral obesity. *Hepatology* 2001; 33: 1358-1364.

Table I. Characteristics of CHC patients according to the presence or absence of metabolic syndrome

Variables	Metabolic syndrome		
	Absence	Presence	p
Number	27 (38.03%)	44 (61.97%)	0.000
Age (years)	47.09 ± 1.37	48.26 ± 0.77)	0.237
Gender			
M (n, %)	12 (54.5%)	10 (45.5%)	0.26
F (n, %)	15 (30.7%)	34 (69.3%)	0.006
BMI (kg/m ²)	25.28 ± 1.73	28.69 ± 0.67	0.036
Visceral obesity			
Presence	4 (9%)	40 (91%)	
Absence	27 (100)	0 (0)	0.000
Blood pressure >130/85 mmHg (n, %)	13 (24.2)	22 (62.8)	0.000
ALT (IU/l)	96.17 ± 8.18	90.97 ± 8.11	0.670
AST (IU/l)	66.26 ± 5.54	59.74 ± 3.85	0.332
GGT (IU/l)	89.56 ± 14.32	72.64 ± 5.26	0.196
HDL- cholesterol (F) (mg/dl)	62.5 ± 2.76	55.5 ± 2.28	0.080
HDL- cholesterol (M) (mg/dl)	50.61 ± 2.93	47.5 ± 3.45	0.491
Serum triglycerides (mg/dl)	107.38 ± 5.53	133.26 ± 7.42	0.014
Fasting glucose (mg/dl)	98.09 ± 2.53	115.06 ± 4.08	0.003
Fasting insulin (mU/l)	17.97 ± 2.05	26.93 ± 3.74	0.119
HOMA-IR	4.29 ± 0.5	7.88 ± 1.11	0.023
HOMA-IR ≥ 2 (n, %)	16 (59.2)	35 (79.5)	0.000
NAFLD	0.00	45 (63%)	0.000

Abbrev: BMI (body mass index); ALT (alanine aminotransferase); AST (aspartate aminotransferase); GGT (gamma glutamyl transpeptidase); HDL (high density lipoproteine); HOMA-IR (homeostasis model assessment for insulin resistance); NAFLD (non-alcoholic fatty liver disease)

9. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985; 28: 412-419.

10. Camma C, Bruno S, DiMarco V, et al. Insulin resistance is associated with steatosis in non diabetic patients with genotype 1 chronic hepatitis C. *Hepatology* 2006; 43: 64-71.

11. Conjeevaram HS, Kleiner DE, Everhart JE, et al. Race, insulin resistance and hepatic steatosis in chronic hepatitis C. *Hepatology* 2007; 45: 80-87.

12. Conjeevaram HS, Kleiner DE, Everhart JE, et al. Race, insulin resistance and hepatic steatosis in chronic hepatitis C. *Hepatology* 2007; 45: 80-87.

13. International Diabetes Federation. The IDF consensus worldwide definition of the metabolic syndrome. <http://www.idf.org>. May 2005.

14. Romero-Gómez M. Insulin resistance and hepatitis C. *World J Gastroenterol* 2006; 12: 7075-7080.
15. Petit JM, Bour JB, Galland-Jos C, et al. Risk factors for diabetes mellitus and early insulin resistance in chronic hepatitis C. *J Hepatol* 2001; 35: 279-283.
16. Sanyal AJ. Review article: non-alcoholic fatty liver disease and hepatitis C - risk factors and clinical implications. *Aliment Pharmacol Ther* 2005; 22(suppl 2): 48-51.
17. Ratziu V, Munteanu M, Charlotte F, Bonyhay L, Poynard T; LIDO Study Group. Fibrogenic impact of high serum glucose in chronic hepatitis C. *J Hepatol* 2003; 39: 1049-1055.
18. Shaheen M, Echeverry D, Oblad MG, Montoya MI, Teklehaimanot S, Akhtar AJ. Hepatitis C, metabolic syndrome, and inflammatory markers: results from the Third National Health and Nutrition Examination Survey (NHANES III). *Diabetes Res Clin Pract* 2007; 75: 320-326.
19. Aytug S, Reich D, Sapiro LE, Bernstein D, Begum N. Impaired IRS-1/PI3-kinase signaling in patients with HCV: a mechanism for increased prevalence of type 2 diabetes. *Hepatology* 2003; 38: 1384-1392.
20. Shintani Y, Fujie H, Miyoshi H, et al. Hepatitis C virus and diabetes: direct involvement of the virus in the development of insulin resistance. *Gastroenterology* 2004; 126: 840-848.
21. Bugianesi E, McCullough AJ, Marchesini G. Insulin resistance: a metabolic pathway to chronic liver disease. *Hepatology* 2005; 42: 987-1000.
22. Tilg H, Hotamisligil GS. Nonalcoholic fatty liver disease: Cytokine-adipokine interplay and regulation of insulin resistance. *Gastroenterology* 2006; 131: 934-945.
23. Liu CJ, Jeng YM, Chen PJ, et al. Influence of metabolic syndrome, viral genotype and antiviral therapy on superimposed fatty liver disease in chronic hepatitis C. *Antivir Ther* 2005; 10: 405-415.
24. Iannucci CV, Capoccia D, Calabria M, Leonetti F. Metabolic syndrome and adipose tissue: new clinical aspects and therapeutic targets. *Curr Pharm Des* 2007; 13: 2148-2168.
25. Guerre-Millo M. Adiponectin: an update. *Diabetes Metab* 2008; 34: 12-18.
26. Pittas AG, Joseph NA, Greenberg AS. Adipocytokines and insulin resistance. *J Clin Endocrinol Metab* 2004; 89: 447-452.
27. Xu A, Wang Y, Keshaw H, Xu LY, Lam KS, Cooper GJ. The fat-derived hormone adiponectin alleviates alcoholic and nonalcoholic fatty liver disease in mice. *J Clin Invest* 2003; 112: 91-100.

Treatment of refractory varicose vein ulceration by means of quadruple therapy (silver cell-hydro alginate, compressive bandaging, micronized purified flavonoid fraction and modest weight loss)

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ABSTRACT

Objective:

Varicose veins ulcers are extremely difficult to treat conservatively at primary care level. We report a novel approach using quadruple therapy in the successful management of resistant varicose vein ulcer.

Methods :

The case is discussed in relation to various modalities targeting varicose vein ulceration in the literature.

Result :

An obese but otherwise healthy 38-year-old Egyptian male presented with chronic superficial varicose vein ulceration of his right leg that had not responded to treatment over six years. After cleaning and light debridement the ulceration was treated with Nugel (Johnson & Johnson) and a silver cell dressing under three-layer bandaging including a carefully applied compression bandage. The dressing was changed every three days and there was complete resolution of the ulceration within four weeks.

Complementary therapy involved initial bed rest with the limb elevated, counseling on necessary weight loss, and oral micronized purified flavonoid fraction (MPFF).

Conclusion:

Treatment of recalcitrant varicose vein ulcer is possible at primary care level.

Keywords:

Varicose vein, quadruple therapy, primary care.

Introduction

Varicose ulceration has a significant prevalence and morbidity and places a considerable burden on health resources internationally (1,2). Chronic lower limb ulceration is common and may have a protracted course when, despite the best available treatments, some ulcers fail to heal.(3) Although there is no racial predilection, women seem more likely to develop venous ulcers than men (4) with a peak prevalence between 60 and 80 years of age (5,6) but 22% of people develop venous ulcers by 40 years of age leading to a substantial reduction in work productivity (7, 8). Consequently accurate diagnosis and optimal management are essential to promote speedy recovery and to prevent relapses.

The mechanisms by which venous hypertension, a prerequisite for venous ulcers, plays a role in the development of venous ulceration remains unclear although recent data suggest the involvement of pericapillary fibrin cuff deposition, fibrinolytic system dysregulation, entrapment of growth factors by macromolecules in the dermis and leukocyte plugging in the venous system of the lower limbs.(9-11).

Venous ulcers are painful with as many as three-quarters of patients reporting adverse effects on their quality of life (12,13). Several risk factors for venous ulceration have been proposed including leg injury (14), obesity, family history of varicose veins, phlebitis, occupations requiring standing for long periods, and previous surgery for varicose veins (15,16). They are characteristically located over the

medial malleolus or gaiter area. The ulcer bed tends to be shallow with fibrinous material and granulation tissue. Venous disease of long duration causes indurations and fibrosis of the dermis and subcutaneous layer and when coupled with lower limb edema, an inverted bottle appearance results (16).

Case report

In February 2008, an otherwise healthy 38-year-old Egyptian male presented with a large varicose ulcer over the medial aspect of the right lower limb (Figure 1) that had persisted in spite of intensive therapy for the previous six years. He had no medical history apart from varicose veins in both lower limbs but there was a family history of venous insufficiency.

He was obese with a BMI of 32; phlebologic examination showed chronic venous insufficiency. There was a superficial painful ulcer measuring 4 x 7 cm on an edematous lower half of the leg, above and around the ankle, distal to the medial malleolus (gaiter area). The edges of the ulcer were irregular; the base was superficial with an exudate and slough. The surrounding skin was erythematous with increased warmth.

Palpation of peripheral pulses was difficult due to edema with normal sensation using 10 g monofilament. Initially the ulcer was cleaned with normal saline using a 20-gauge needle for irrigation; light debridement removed the slough and non-vital tissues and was followed by the application of Nu-gel, (Johnson & Johnson), a hydrogel consisting of a matrix of insoluble polymers with up to 90% water content enabling the donation of water molecules to the wound surface thereby transmitting vapour and oxygen. This is claimed to promote wound debridement by rehydration of non-viable tissue and to facilitate natural autolysis in the management of sloughing or necrotic wounds (55-58). Silvercel hydro-alginate dressing is a



Figure (3)



Figure (1)



Figure (4)



Figure (2)

controversial treatment for varicose ulcers that is discussed further below. In this case it was used and covered with 3-layer bandaging including one compression bandage applied in a 50% overlapping fashion to exert a gradual pressure greatest distal to the toes and reducing progressively to the anterior tibial tuberosity. Systemic treatment included oral micronized purified flavonoid fraction (Daflon: Servier) 500mg twice daily. The patient was instructed to rest in bed with the limb elevated for the first three days. Bandages and dressing with silver cell were changed every three days. Re-epithalization started at the wound edges and, later, islands of epithelium could be seen in the middle of the ulcer that represented keratinocyte out-growth from the hair follicles (44,45) producing complete resolution within one month. (Figures 2,3,4). The patient was also counseled regarding an average weight loss of 5 kg using the portion control (one dahoo-plate) method. He

refused prophylactic compressive treatment, although some authors believe that compressive therapy constitutes the most important part of the conservative therapy of chronic venous insufficiency (43), but agreed to maintenance Daflon therapy. Follow up of the patient at three and six months showed intact skin (Figure 4).

Discussion

Recognised modifiable risk factors for varicose vein disease include occupations involving long periods of standing, and obesity (59,60) since it is thought that obesity leads to an increase in intra-abdominal pressure that impedes venous return from the lower extremities (61). Sugerman and colleagues demonstrated that weight loss is associated with correction of venous stasis in almost all patients (62).

Suggested modalities for the treatment of venous ulceration include elevating the legs above the heart (19) and compression therapy to improve ulcer healing and prevent recurrence (20-22) with a recent meta-analysis suggesting that multilayer compression therapy is superior to single-layer bandaging (23). Other options that have been studied with various degrees of success include compression sclerotherapy, echo sclerotherapy (31), ultrasound-guided foam sclerotherapy (32), skin grafting (33), superficial venous surgery (34), and sub-fascial endoscopic perforator surgery (35) and sub-fascial endoscopic perforator surgery (36) although endovenous laser therapy and vein surgery with or without skin grafting should be considered only as a final option when all other measures have failed (17). Tissue-engineered skin equivalent (recently approved by the U.S. Food and Drug Administration) is an exciting development in the treatment of venous

ulcer (37) and granulocyte-macrophage colony stimulating factor (GM-CSF), has proved to be effective both intraliesionally and topically in two randomized, double-blind, placebo-controlled studies (38-41). Currently, keratinocyte growth factor-2 (KGF-2) is under investigation to assess its safety and efficacy in humans (42). Medication with aspirin (24), pentoxifylline (25-28) and a methylxanthine derivative has been found effective (29). Flavonoid drugs have been used in the management of venous disease and their effects upon microcirculation studied. Micronized purified flavonoid fraction (MPFF) has been used in animal models and has shown efficacy in modulating leukocyte adhesion and preventing endothelial damage. (30) Human patients with venous disease have shown similar biochemical effects which may explain the efficacy of this novel treatment in the management of symptoms, edema and modification of venous leg ulcer healing (18). MPFF compounds are believed to act in the macrocirculation, improving venous tone as well as in the microcirculation decreasing capillary hyperpermeability (46). This inhibition is linked to a significant decrease in plasma levels of endothelial adhesion molecules (VCAM-1 and ICAM-1). The lymphatic system is improved also due to the lymphagogue activity (47) modulating leukocyte adhesion and preventing endothelial damage, thereby improving symptoms of chronic venous ulceration (48,49). Silvercel, a hydro-alginate is a highly-absorbent material that maintains an optimal antimicrobial and moist wound healing environment in medium to heavy exuding wounds (50,51). When in contact there is an exchange of sodium ions from the wound fluid with calcium ions on the alginate. This action gives the alginate its high absorbent properties, superior to a hydrofibre dressing (52,53), creating a warm, moist environment for wound healing and allowing non-traumatic removal of the dressing (54), although some (personal comment) feel that the silver dressing is unsuitable for painful ulcers and that the excessively moist environment produced by it and by Nugel actually delays healing by increasing maceration. However, that this one reported case healed in four weeks after failures over six years does suggest that the Silvercel/Nugel combination probably played at least some part in the success of the quadruple treatment.

Conclusion

Quadruple therapy is relatively expensive but the cost-effectiveness is governed by other considerations. The healing time of venous lower limb ulcers is significantly reduced with quadruple therapy and for this reason combination therapy is recommended for non-healing chronic venous ulcers that are resistant to other classic treatments.

References

- (1) Paul R. weaver: A varicose ulcer healed by non surgical varicose vein treatment using ultrasound guided foam sclerotherapy. *Nzfp*, vol 35, No.1, Feb 2008 p 32-33.
- (2) Valencia I C, Falabella A, Kirsner RS, Eaglstein W H : Chronic venous insufficiency and venous leg ulceration. *J Am Acad Dermatol*. 2001; 44: 401-21; quiz 422-4 [PMID : 11209109][Medline]
- (3) S. Abisi, J Tan and K.G, Burnard: Excision and meshed skin grafting for leg ulcers resistant to compression therapy. *British J of surgery* 2007; 94: 194-197.
- (4) Nelzen O, Bergqvist O, Lindhagen A. Venous and non-venous leg ulcers: clinical history and appearance in a population study *Br J Surg*. 1994;81 :182-7. [PMID: 8156328J] [Medline]
- (5) Callam MJ, Harper DR, Dale JJ, Ruckley CV. Chronic ulcer of the leg: clinical history *Br Med J (Clin Res Ed)*. 1987;294: 1389-91. [PMID: 3109669].
- (6) Bergqvist O, Lindholm C, Nelzen O. Chronic leg ulcers: the impact of venous disease. *J Vasc Surg*. 1999;29:752-5. [PMID: 10194512][Medline].
- (7) Callam MJ, Ruckley CV, Harper DR, Dale JJ. Chronic ulceration of the leg: extent of the problem and provision of care *Br Med J (Clin Res Ed)*. 1985;290: 1855-6. [PMID: 3924283].
- (8) Ruckley CV. Socioeconomic impact of chronic venous insufficiency and leg ulcers *Angiology*. 1997;48:67-9. [PMID: 8995346].
- (9) Falanga v, Eaglstein WH. The "trap" hypothesis of venous ulceration. *Lancet*. 1993;341: 1 006-8. [PMID: 7682272]. [Medline].
- (10) Browse NL, Burnand KG. The

cause of venous ulceration *Lancet*. 1982;2:243-5. [PMID: 6124673] [Medline]

- (11) Thomas PR, Nash GB, Dormandy JA. White cell accumulation in dependent legs of patients with venous hypertension: a possible mechanism for trophic changes in the skin *Br Med J (Clin Res Ed)*. 1988;296:1693-5. [PMID: 3135881].
- (12) Phillips T, Stanton B, Provan A, Lew R. A study of the impact of leg ulcers on quality of life: financial, social, and psychologic implications *J Am Acad Dermatol*. 1994;31:49-53. [PMID: 8021371][Medline]
- (13) Friedman SA. The diagnosis and medical management of vascular ulcers. *Clin Dermatol*. 1990;8:30-9. [PMID: 2129948][Medline]
- (14) Scott TE, LaMorte WW, Gorin DR, Menzoian JO. Risk factors for chronic venous insufficiency: a dual case-control study *J Vase Surg*. 1995;22:622-8. [PMID: 7494366]. [Medline]
- (15) Nelzen O, Bergqvist O, Lindhagen A. Leg ulcer etiology-a cross sectional population study *J Vase Surg*. 1991 ;14:557-64. [PMID: 1920653]. [Medline]
- (16) Browse NL, Clemenson G, Thomas ML. Is the postphlebotic leg always postphlebotic? Relation between phlebographic appearances of deep-vein thrombosis and late sequelae *Br Med J*. 1980;281 :1167-70. [PMID: 7427621].
- (17) Warburg FE, Danielsen L, Madsen S M, Raaschon H O, Munkvad S, Jensen R et al: Vein surgery with or without skin grafting Versus conservative treatment for leg ulcers. A randomized prospective study. *Acta derm venereol* 1994; 74: 307-309.
- (18) Coleridge. Smith P D: From skin disorders to venous leg ulcers: Pathophysiology and efficacy of Daflon 500mg in ulcer healing. *Angiology*, 2003, Jul-aug; 54 Suppl 1: S 45-50.
- (19) Abu-own A, Scurr JH, Coleridge Smith PD. Effect of leg elevation on the skin microcirculation in chronic venous insufficiency *J Vase Surg*. 1994;20:705-10. [PMID: 7966805]. [Medline].
- (20) Erickson CA, Lanza DJ, Karp DL, Edwards JW, Seabrook GR, Cambria RA, et al. Healing of venous ulcers in an ambulatory care program: the roles of chronic venous insuf-

- iciency and patient compliance J Vase Surg. 1995;22:62936. [PMID: 7494367]. [Medline]
- (21) Blair SD, Wright DD, Backhouse CM, Riddle E, McCollum CN. Sustained compression and healing of chronic venous ulcers BMJ. 1988;297:1159-61. [PMID: 3144330].
- (22) Partsch H. Compression therapy of the legs. A review J Dermatol Surg Oncol. 1991;17:799-805. [PMID: 1918586] [Medline].
- (23) Fletcher A, Cullum N, Sheldon TA. A systematic review of compression treatment for venous leg ulcers BMJ. 1997;315:576-80. [PMID: 9302954].
- (24) Weithmann KU. The influence of pentoxifylline on interactions between blood vessel wall and platelets IRCS Medical Science [microform]. 1980;8:293-4.
- (25) Colgan MP, Dormandy JA, Jones PW, Schraibman IG, Shanik DG, Young RA. Oxpentifylline treatment of venous ulcers of the leg BMJ. 1990;300:972-5. [PMID: 2256974].
- (26) Dale JJ, Ruckley CV, Harper DR, Gibson B, Nelson EA, Prescott RJ. Randomised, double blind placebo controlled trial of pentoxifylline in the treatment of venous leg ulcers BMJ. 1999;319:875-8. [PMID: 10506039 H=]
- (27) Falanga V, Fujitani RM, Diaz C, Hunter G, Jorizzo J, Lawrence PF, et al. Systemic treatment of venous leg ulcers with high doses of pentoxifylline: efficacy in a randomized, placebo-controlled trial Wound Repair Regen. 1999;7:208-13. [PMID: 10781212]. [Medline].
- (28) Jull AB, Waters J, Arroll B. Pentoxifylline for treating venous leg ulcers. Cochrane-Database svst Rev. 2002. (1). CD001733. Review. (PMID: 11869606).
- (29) Guilhou JJ, Dereure O, Marzin L, Ouvry P, Zuccarelli F, Debure C, et al. Efficacy of Daflon 500 mg in venous leg ulcer healing: a double-blind, randomized, controlled versus placebo trial in 107 patients Angiology. 1997;48:77-85. [PMID: 8995348].
- (30) Compression sclerotherapy is useful in v. vein : AYu. Krylov, AM.Shulutko, E.C.Najovitzyn, MV.Safonov . J Ang Vasc surg Vol 6.1/2000 ; P:54
- (31) Paul R . Weaver . Avaricose ulcer healed by non surgical varicose vein treatment using ultrasound guided to am sclerotherapy nzfp, vol 35, No. 1, Feb 2008, P 32-33..
- (32) Douglas WS, Simpson NB. Guidelines for the management of chronic venous leg ulceration. Report of a multidisciplinary workshop. British Association of Dermatologists and the Research Unit of the Royal College of Physicians Br J Dermatol. 1995;132:446-52. [PMID: 7718464]. [Medline].
- (33) Olivencia JA Subfascial endoscopic ligation of perforator veins (SEPS) in the treatment of venous ulcers Int Surg. 2000;85:266-9. [PMID: 11325008]. [Medline]
- (34) Dunn RM, Fudem GM, Walton RL, Anderson FA Jr, Malhotra R. Free flap valvular transplantation for refractory venous ulceration J Vasc Surg. 1994;19:525-31. [PMID: 8126867] [Medline].
- (35) Falanga V, Margolis D, Alvarez O, Auletta M, Maggiacomo F, Altman M, et al. Rapid healing of venous ulcers and lack of clinical rejection with an allogeneic cultured human skin equivalent. Human Skin Equivalent Investigators Group Arch Dermatol. 1998; 134:293-300. [PMID: 9521027]
- (36) Compression sclera therapy is useful in Vivien .Ref: A. Yu. Krylov, A.M . Shulutko, E.C. Nagovitzyn, M.V. Safonov. Jang Vasc Jugr Vol . 6.1/2000; P: 54
- (37) Marques da Costa R, Jesus FM, Aniceto C, Mendes M. Double-blind randomized placebo-controlled trial of the use of granulocyte-macrophage colony stimulating factor in chronic leg ulcers Am J Surg. 1997;173:165-8. [PMID: 9124619]. [Medline].
- (38) Halabe A, Ingber A, Hodak E, David M. Granulocyte-macrophage colony-stimulating factor--a novel therapy in the healing of chronic ulcerative lesions. Med Sci Res. 1995;23:65-6.
- (39) Pojda Z, Struzyna J. Treatment of non-healing ulcers with rhGM-CSF and skin grafts [Letter] Lancet. 1994;343:1100 [PMID: 7909116] [Medline]
- (40) Jaschke E, Zabernigg A, Gatringer C. Recombinant human granulocytemacrophage colony-stimulating factor applied locally in low doses enhances healing and prevents recurrence of chronic venous ulcers Int J Dermatol. 1999;38:380-6. [PMID: 10369552] [Medline].
- (41) Da Costa RM, Ribeiro Jesus FM, Aniceto C, Mendes M. Randomized, double-blind, placebo-controlled, dose-ranging study of granulocyte-macrophage colony stimulating factor in patients with chronic venous leg ulcers Wound Repair Regen. 1999;7:17-25. [PMID: 10231502] [Medline].
- (42) Robson MC, Phillips T J, Falanga V, Odenheimer OJ, Parish LC, Jensen JL, et al. Randomized trial of topically applied repifermin (recombinant human keratinocyte growth factor-2) to accelerate wound healing in venous ulcers Wound Repair Regen. 2001 ;9:347-52. [PMID: 11896977]. [Medline].
- (43) V. SLOhkova, Z . Navartilova, V . Semradova and J.Adler . Successful treatment of chronic venous leg ulcer with hyoplilized cultured epidermal allografts. Acta Dermatoven APA Vol 13, 2004, No.4 Page 119-123.
- (44) Scondotto G, Aloisi D, Ferrai P, Martini L . Treatment of venous leg ulcers with sulodexide Angiology . 1999 ; 50:883-9.[PMID:10580352].
- (45) Zacur H, Kirsner RS . Debridement : rationale and therapeutic options Wounds . 2002 ;14(Suppl F):2ER-7E.
- (46) Behar A, Lagrue G, cohen - Boulakia F, Baillet J, capillavy filtration in idiopathic cyclic edema- effects of Daflon 500 mg. Nuklearnedizin . 1998;27:105-7.
- (47) Al bert - Adrien .Ramelet MD, pharmacologic Aspects of phototropic drug in CVI - Associated edema . Angiology, Vol 51, No 1, 19-23, (200).
- (48) Phillip D. Coleridge Smith . Micro-nized & urified falconoid fraction and the treatment of chronic venous insufficiency : micro circulatory mechanisms . Micro microcirculation, volume 7, issue 6 supplement 1, Dec 2000.
- (49) Nicolaidis AN. from symptoms to leg edema : efficacy of Daflon 500 mg. Angiology.2003: 54: S33-S44.
- (50) Morgan DA . Alginate dressing . part 2: product guide . I bid 1997;7:9-14.

- stimulating factor applied locally in low doses enhances healing and prevents recurrence of chronic venous ulcers *Int J Dermatol.* 1999;38:380-6. [PMID: 10369552] [Medline].
- (41) Da Costa RM, Ribeiro Jesus FM, Aniceto C, Mendes M. Randomized, double-blind, placebo-controlled, dose- ranging study of granulocyte-macrophage colony stimulating factor in patients with chronic venous leg ulcers *Wound Repair Regen.* 1999;7:17-25. [PMID: 10231502] [Medline].
- (42) Robson MC, Phillips T J, Falanga V, Odenheimer OJ, Parish LC, Jensen JL, et al. Randomized trial of topically applied repifermin (recombinant human keratinocyte growth factor-2) to accelerate wound healing in venous ulcers *Wound Repair Regen.* 2001 ;9:347-52. [PMID: 11896977]. [Medline].
- (43) V. SLOHKOVA, Z . Navartilova, V . Semradova and J.Adler . Successful treatment of chronic venous leg ulcer with hyoplilized cultured epidermal allografts. *Acta Dermatoven APA Vol 13, 2004, No.4 Page 119-123.*
- (44) Scondotto G, Aloisi D, Ferrai P, Martini L . Treatment of venous leg ulcers with sulodexide *Angiology .* 1999 ; 50:883-9.[PMID:10580352].
- (45) Zacur H, Kirsner RS . Debridement : rationale and therapeutic options *Wounds .* 2002 ;14(Suppl F):2ER-7E.
- (46) Behar A, Lagrue G, cohen - Boula- kia F, Baillet J, capillary filtration in idiopathic cyclic edema- effects of Daflon 500 mg. *Nuklearnedizin .* 1998;27:105-7.
- (47) Al bert - Adrien .Ramelet MD, pharmacologic Aspects of phototropic drug in CVI - Associated edema . *Angiology, Vol 51, No 1, 19-23, (200).*
- (48) Phillip D. Coleridge Smith . Micro- nized & urified falconoid fraction and the treatment of chronic venous insuf- ficiency : micro circulatory mechanisms . *Micro microcirculation, volume 7, issue 6 supplement 1, Dec 2000.*
- (49) Nicolaidides AN. from symptoms to leg edema : efficacy of Daflon 500 mg. *Angiology.*2003: 54: S33-S44.
- (50) Morgan DA . Alginate dressing . part 2: product guide . *I bid 1997;7:9-14.*
- (51) Morgan DA Alginate dressing . part 2: product guide . *I bid 1997 ;7:9-14.*
- (52) SMTL - Surgical material testing laboratory / JJWM (2003) Data on file report RD 675(SMTL 03/1610/01).
- (53) Luciana Patricia Fernandes Ab- bade, sidnei Lasttoria. management of particuts with leg ulcer. *An . Bras . Dermatol, Vol 81, No .6. Rio de Janeiro Nov / Dec 2006.*
- (54) Rosie Pudner . Alginate and hydrofibre dressing in wound manage- ment . *JCN, May 2001.Vol 15 issue 05, pp 1-5.*
- (55) Vanessa Jones, Joseph E Grey and Keith G Harding . Wound dressing . *BMJ 2006; 332;777-780.*
- (56)Morgan DA, wound management products the drug tariff . *The pharma- ceutical Journal, Vol 263 . No 3 7072, p 820-825.Nov 20,1999.*
- (57) Cho ch, Lo J . Dressing the part .In : Mcgillis ST, editor . *Dermatologic clinics . Philadelphia : W.B. Saunders company ;1998.p25-47.*
- (58) Cuzzell J, Krasner D . Curativos . In : Gogia PP, editor . *Feridas : trata- mento e cicatrizcao . Rio de Janerio : Revinter L + da ;2003.p.103-14.*
- (59) Joseph J . Naoum ; Glen C Hunter . pathogenesis of varicose veins and implications for clinical management. *vascular .2007 Sept - Oct ;15(5):242-9.*
- (60) Lengyel I, Acsady G . Histo morphological and patho biochemi- cal changes varicose veins . A pos- sible explanation of the development of varicosities . *Acta Morphob Hung 1990;38:259-67.*
- (61) Poirier P, GILES td, Bray GA, etal . Obesity and (rdiovascular disease : pathophysiology, evaluation, and ef- fect of weight loss. *Arterioscler Thromb vasc Biol 2006 ;26:968-76.*
- (62) Sugerma HJ, Sugerma EL, Wolfe L, etal . risks and benefitsof gas- tric by boss in morbidly obese patients with severe venous stasis disease . *Ann Surg 2001; 234:41-6.*

Assessment of factors and conditions that influence HIV Positive Women's Rights to family resources in Abia, State of Nigeria

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ABSTRACT

Introduction:

Every country has different practices that influence rights to family resources. In developing countries, including Nigeria, cultural practices favour males for economic ventures more than females. There is evidence that encouraging HIV positive women's rights to family resources will lessen risks they take to overcome negative economic consequences of HIV and AIDS. This will help to achieve the much needed reduction in HIV prevalence in Nigeria.

Materials and method:

Total sample of 98 HIV positive women in a network of people living with HIV and AIDS and also 5 traditional rulers in charge of the communities studied, were involved.

Data collection instruments were questionnaire, focus group discussion and key informant interview. Using key informant interview with traditional rulers helped to authenticate responses of the women. Data were analyzed qualitatively and quantitatively with percentages.

Result:

Findings showed that factors like widowhood inheritance, subordinate roles of women, breadwinner roles of men, terming women as visitors, and seeing women as responsible for

the death of loved ones, negatively influenced rights to family resources of HIV positive women. The majority of the women were subjected to horrifying experiences like beating, chastisement, rejection and others which resulted in interpersonal conflicts, and violence for venturing to acquire material resources for the family. A good number of the traditional rulers interviewed did not support economic empowerment of women whether HIV positive or not. The premise is that women are subordinate to men and should not be allowed to take over family resources to the disadvantage of men. They argue that women would be obstinate if they are allowed much material goods.

As high as 85 (86.7%) of the women studied were denied rights to family resources. To survive in the communities, the women took two types of risks, acting as hired labourers and having sex without condoms. A total of 54 (55%) of them had sex without a condom. Common reason proffered for taking this risk was sex partners' dislike for condom use.

Conclusion:

It is therefore plausible to recommend that regular seminars and/or workshops should be organized to educate the traditional rulers and others on the need to accord HIV positive women access to family resources so as to enable them to cope with their health, economic and social needs.

Key words: inheritance rights, HIV/AIDS, unprotected sex, policies, Nigeria

Introduction

One of Nigeria's greatest challenges is to discourage cultural practices that negatively affect the economic existence of HIV positive women. With Nigeria's high prevalence of HIV and AIDS, 5% of women attending antenatal care services (Federal Ministry of Health 2002 HIV and AIDS surveillance

study), economic empowerment of HIV positive women should be given high priority in the society. There is growing evidence that eliminating practices that discourage women's inheritance rights to family resources, especially those HIV positive, will mitigate negative economic consequences of HIV and AIDS, and reduce poverty [1,2]. This is necessary because women's lack of economic empowerment is the key factor in the spread of HIV and AIDS [3].

The problem is that traditionally, customs forbid women to own resources like land, and houses that would bring them at par with men economically [4], yet women's rights to inherit housing and land are enshrined under the international human rights laws to which many countries including Nigeria are signatories. The Nigerian legal system is a combination of Nigerian legislation, English law, customary law and judicial precedents [5]. Each of the legal systems determines the right of the woman. Under statutory marriage, women and children could have property rights but under customary law which is commonly practiced in Nigeria, women do not have the right to inherit family resources [6,7]. Usually, if a husband dies, the widow, her children and husband's property are inherited by brothers and/or other male relations of the deceased husband. Traditionally, a widow only escapes being inherited if she is too old and/or frail to be inherited [8-10].

In Abia State, property ownership is a source of security for means of livelihood and also a quick capital by which additional economic resources are acquired. It is this premise that encouraged men to exclude women, including widows and those HIV positive from access to and/or control of family resources [11-13]. Nowadays, the devastating effects of HIV/AIDS due to economic hardship requires that HIV positive women should be encouraged to own resources to enable them cope with their economic demands [14, 15]. This is necessary because the economic burdens of HIV and AIDS have reduced household income by 80%, food consumption by 15-30% and primary school enrolment by 20-40% [16,17]. Denying

HIV positive women access to family resources because of stringent cultural practices could worsen their health conditions and chances of survival [18-20]. A good understanding of the health, economic and social needs of HIV positive women would encourage individuals to support economic empowerment of HIV positive women.

The custom of regarding married women as strangers who could leave when the marriage is estranged, contributes to the practice of denying them rights to family resources. The premise is that if women including those HIV positive are allowed access to family resources, that when their marriages are estranged, that they would probably take with them the acquired items (21-25).

The indigenous practice where the estate of a man who dies Intestate, would be inherited by his male children and/or relations rather than his female children has been described by [26-28] as a disadvantage to women's rights to resources. These authors favour the supreme court judgment (Mojekwu's judgment) that females should be encouraged to inherit family resources to minimize the economic hardship that most women are exposed to at the death of their breadwinners.

Family resources for this study refers to material goods in the family such as land, house, and other items that could generate funds.

The study aimed at noting the extent to which HIV positive women are allowed rights to family resources to enable them to generate finance to support themselves and avoid depending on others for assistance.

Study objectives are:

- to note the extent to which traditional rulers support economic empowerment of HIV positive women
- to identify types of relationships HIV positive women encounter with family members
- to document factors that influence HIV positive women's rights to family resources

Material and methods

Total sample of 98 HIV positive women in a network of people living with HIV/AIDS in Abia State was studied. Women in the network of people living with HIV/AIDS were used because of the difficulty in identifying HIV positive individuals in the society. Most individuals are reluctant for some reasons, to disclose their sero status. It was considered safer to study those who have openly declared their sero-status. Also 5 traditional rulers from the five communities where the PLWHA came from were interviewed. The traditional rulers were included in the study so as to authenticate the responses of the HIV positive women. Moreover, it was assumed that Traditional rulers as custodians of culture are veritable instruments capable of effecting changes that would positively influence HIV positive women's rights to family resources.

Study Area

The study area is Abia State of Nigeria. Abia State is located in the South-eastern part of Nigeria and comprises 17 local government areas with Igbo as the common language. The population is over 3 million [2006 draft census report] with HIV prevalence rate of 3.6% [ABIA State HIV/AIDS status 2006]. In the indigenous customary set up, males are entitled to marrying many wives, have extra-marital sexual relationships, maintain family name, and also inherit family resources unlike females.

Two types of marriages, statutory and customary are practiced. Customary marriage is most popular and enforces women married under this law to practice exogamy (leaving their descendents to live with that of their husbands). There are two types of inheritance, patrilineal and matrilineal but the most common is patrilineal. Under both types of inheritance, custom upholds males as the sole owner of any property in the family, and that sons and not daughters should be the surviving heirs. However, under matrilineal systems, unlike patrilineal, daughters might have the right to inherit family resources only if they choose not to be married but rather remain in the family to procreate male issues that would bear the family name particularly, where such families do not have male issues. Where such daughters fail to have male issues, then, the family resources from the mother's side would be given to the nearest male kin relation.

Also, if a woman is wealthy as to buy landed property, it will be deemed that she bought the land for either the son or the husband and not for herself. Culturally, men's social statuses are assessed by the number of wives and children they have. The higher the number of wives and children, the higher men are socially rated. Traditionally, inheritance is based on the principle of primogeniture (the right of the first born to succession). Where a man marries more than one wife, the first son of each wife would be entitled to rights to inherit family resources. The extent of share each son gets depends on the age. The eldest gets a larger share than others. For occupation, main occupation of people in the communities is subsistence farming and staple food is 'garri', a product of cassava.

Data Collection

Data were collected with questionnaire, focus group discussion and interview guide. The questionnaire which contained open-ended and closed-ended questions was administered for those who cannot read and write, and self-administered for those who can read and write. Interview guide contained structured questions which were used to uncover some personal information that respondents were unwilling to release at group levels. This method was termed necessary because of the sensitive nature of the study.

Twelve focus group discussions comprising 7-9 HIV positive women were organized with the help of three trained Research Assistants. The questions in the focus group discussion contained structured questions and women were grouped according to their ages, marital status, types of marriage, and locale.

Key informant interview was used for the traditional rulers. The questions were made up of open-ended questions which explored the views of the traditional rulers on economic empowerment of women and others. For uniformity in the analysis, the key informant guide was administered because of disparity in the education of the traditional rulers.

Results of focus group discussions which were grouped according to themes were translated and transcribed by the three trained Research Assistants. Data were analyzed both qualitatively and quantitatively using Tables and percentages.

Ethical Considerations

University Ethical Committee vetted and approved the study before its commencement. Following this approval, permission to conduct the study was obtained from the President of the network of people living positively with HIV/AIDS (PLWHA) as well as from the female members of the network in the State. Their respective approvals enabled the researcher to collect information from them uninhibited. The researchers also gave statements of confidentiality as well as briefs on the objectives of the study.

A request for permission to tape-record the session was made to participants and this was granted. In addition, a written permission to conduct the study was requested and obtained from the traditional rulers in the five communities studied.

Results

Findings from the traditional rulers

Five traditional rulers, all males, between the ages of 60 years and above were interviewed.

Traditional rulers' views on economic empowerment of HIV positive women None of the five traditional rulers interviewed supported economic empowerment of women whether HIV positive or not. They held a common view that women would be obstinate if they are allowed to acquire more assets than men. The Traditional rulers emphasized that culturally, women are subordinates to men and that they should not acquire family resources in preference to men. Moreover, they stressed that women are visitors in the family and that they could abscond from the family at will and therefore should not be entrusted with family resources.

The majority of the traditional rulers felt that it would be outlawed for anyone to allow females to inherit family resources when there are males in the family to do so. Their belief was that men as breadwinners should be in possession of family resources so as to enable them to plan disbursement for the benefit of all family members.

In the words of these traditional rulers: it would be difficult for anyone to convince elders as decision makers, to allow women irrespective of their health or marital status, to inherit family resources, especially immovable ones like land, housing and others when there are men to do so.

Traditional ruler's knowledge of the health needs of HIV positive women: Findings showed that the traditional rulers' overall knowledge of the health and nutritional needs of HIV positive women remained poor. Though the traditional rulers had a slight knowledge of modes of HIV infection, they were not aware that HIV positive women require special attention. For instance, the traditional rulers lacked the knowledge that HIV positive women need to eat balanced food, procure anti-retroviral drugs in order to cope with impacts of HIV and AIDS as well as seek treatment for opportunistic infections. The popular view of these rulers was that lazy women always fake sickness in order to avoid farming so as to depend on others for assistance. The rulers emphasized that women should engage in elaborate farming to produce enough food for their families' consumption.

The most intriguing findings in this section is that traditional rulers' lacked knowledge of those who are HIV positive in their respective communities. This finding suggests that most HIV positive women studied did not disclose their sero-status in the communities where they live. This lack of disclosure could be responsible for the traditional rulers' poor knowledge of basic needs of HIV positive women.

Traditional rulers' responses on factors that influence HIV positive women's rights to family resources: Traditional rulers worried that individuals in the communities stress on practices that disallow women rights to resources more than other factors. Using the words of some of the traditional rulers: 'We feel bad that culture regards women as visitors in their matrimonial homes and therefore, are neither involved in family decisions nor entitled to family resources. Women are usually accused of being responsible for any death that occurs in the family, that is why a family 'will' is scarcely made in their favour and even when made, it is usually not implemented'.

Findings from HIV positive women:

Background information:

The average age of women studied is 22 years. About two-thirds of them, 64 (65.3%) are widowed, 20 (20.4%) are married while 14 (14.3%) are single. Out of those married, 8 (8.2%) of them are cohabiting with their husbands, while 12 (12.2%) of others are divorced and/or separated. From the number that

are widowed, 48 (49%) of them were forced back to their natal homes, while 16 (16.3%) others were inherited by relations of their deceased husbands. For those that are single, 6 (6.1%) of them were about getting married while the rest were betrothed and had strong plans of getting married soon.

Out of the population of HIV positive women studied, only 16 (16.3%) had tertiary and secondary school education and are employed in some establishments while out of 82 (83.7%) who had primary school and non-formal education, 34 (34.7%) are self employed while the rest are mainly subsistent farmers. About 61 (62.2%) PLWHA complained of lack of financial assistance from relations. In all, 38 (38.8%) and 60 (61.2%) of the HIV positive women studied live in urban and rural areas respectively.

HIV positive women's responses on factors that affect rights to family resources:

The majority of the HIV positive women especially widows said they were deprived family resources because they were accused of playing key roles in the death of their husbands and/or other family members. As a result, they were subjected to some inhuman treatments such as stripping them, shaving of hairs including pubic hair, starving them, forcing them to cross coffins of those they are suspected to have killed as well as sleeping in the same room with such corpses. These treatments they said, were meted out to them to substantiate the allegations against them. Implications of these harsh treatments is that if during this period none of them died and/or fell sick, they would be vindicated, otherwise, they would be held responsible. In all, a total of 85 (86.7%) HIV positive women including widows said they were tortured and denied access to family resources.

Using the words of six of these women:

Our husbands' relations collected all our husbands' belongings on hearing of their death. They accused us of killing our husbands and as a result, brutalized us. Because of these accusations, they denied us support with our husbands' resources. Now some of our children have dropped out of school due to inability to pay their school fees.

When the women were asked whether those living in the urban areas also had similar experiences from their husbands' in the communities, a good number of the women responded in the affirmative.

relations like those in the communities, a good number of the women responded in the affirmative. The women worried because they felt that the elders sanctioned whatever dehumanizing actions their husbands' relations meted to them. Using the words of five women: The elders are not kindly disposed towards us. They support our brothers-in-law to maltreat us, If we ask for financial assistance, they would boo at us, insult and accuse us of also planning for their untimely death like we did in the case of our husbands. We are excluded from family decisions because we are regarded as visitors in our matrimonial homes. Moreover, when a family 'will' is made in our favour, the elders would discourage its implementation stressing that as visitors, we are not supposed to be heirs in the family.

The women complained that their greatest problem was how to raise money to procure anti-retroviral drugs, feed their children, and pay for their children's education.

Types of relationships HIV positive women enjoyed with family members: Findings showed that a good number of HIV positive women especially the widowed had horrifying experiences like rejection, discrimination, beating, chastisement, lack of care and support with some family members which translated into interpersonal conflicts.

Four (4) of the women who were inherited narrated their experiences as thus:

'We are not happy because our brothers-in-law who inherited us insult, brutalize, stigmatize, and deny us financial assistance. Men are generally wicked. They purposely would not provide our needs. If we complain they will label us as bad and threaten to drive us out of the family or kill us.'

Further reports from three (3) widows state that:

'We have six children, yet our brothers-in-law took all our husbands' resources because we refused to be inherited barely one month after the death of our husbands. When we complained, we were chased out of our matrimonial homes.

Result from the quantitative data also show horrifying experiences HIV positive women had with family relations see Table 1.

Types of experiences	Frequency
Chastisement	9 (9.2%)
Scolding	12 (12.2%)
Beating	14 (14.3%)
Rejection	18 (18.4%)
Telling off	12 (12.2%)
Intolerance	20 (20.4%)
Refusal to provide health care services	13 (13.3%)
Starvation	16 (16.3%)
Flogging	8 (8.2%)

Table 1: HIV positive women and types of experiences with family members

From this Table, the highest experience HIV positive women 20 (20.4%) encountered from family members was intolerance.

Responses the women gave on reasons for family members' actions against them are contained in Table 2.

Reasons	Frequency
Demanding husbands possessions	38 (38%)
Refusing to be inherited	22 (22.4%)
Knowing one's HIV status	29 (29.6%)
Asking for financial assistance	36 (36.7%)
Not having male children	19 (19.4%)
Demanding treatment when sick	11(11.2%)
Refusing to have sex without condom	20 (20.4%)
Being accused of killing husband	15 (15.3%)

Table 2: Reasons for Family Members' actions

From Table 2, the commonest reason 38 (38.8%) HIV positive women gave for their family members' action against them was demand for husbands' possessions. Further, the marital status of the women commonly chastised was explored. From the finding, respondents from all marital statuses were chastisements but the most commonly chastised were the widowed 48 (49%). Table 3 contains this.

Chastised	Married	Single	Widowed	Total
Yes	7 (7.1%)	4 (4.1%)	48 (49%)	59 (60%)
No	13 (13.3%)	10 (10.2%)	16 (16.3%)	39 (40%)
Total	20 (20.4%)	14 (14.3%)	64 (65.3%)	98 (100%)

Table 3: Marital Status of females chastised

From the Table, as high as 59 (60.2%) of the HIV positive women were chastised in all marital statuses.

Another important finding was that HIV positive women made a living by taking two types of risks. Firstly, 25 (25.5%) of them earned their livelihood by acting as hired labourers in the farm while as high as 54 (55.1%) at various periods had sex without a condom. These risks were more among women in the rural areas than those in urban areas. Table 4 contains details of their sexual practice.

Ever used condom during sex	Respondents by residence		
Response category	Urban	Rural	Total
Yes	11 (11.2%)	9 (9.2%)	20 (20.4%)
No	17 (17.3%)	37 (37.8%)	54 (55.1%)
Do not have sex	10 (10.2%)	14 (14.3%)	24 (24.5%)
Total	38 (38.8%)	60 (61.2%)	98 (100%)

Table 4: HIV positive Women and their Sexual Practice by Residence

Women's reasons for unprotected sex	N=54 Frequency of response
Male partners dislike condom use	47 (87%)
Ashamed to negotiate condom use	19 (35.2%)
Do not have condom	13 (24%)
Wants to have babies	12 (15.4%)
Needs financial assistance	39 (72.2%)
Fear of being beaten	17 (31.5%)
Would not like to be termed promiscuous	21 (38.9%)
Do not want people to know my sero-status	27 (50%)
No reason	9 (16.7%)

Table 5: HIV positive Women and their Sexual Practice by Residence

From the table, the highest reason 47 (87%) the women had for this awful practice was their male partners' dislike for condom use

Discussion

There were striking similarities between information given by the traditional rulers and the responses of the HIV positive women on factors that discourage HIV positive women's rights to family resources. The HIV positive women enumerated factors such as perceiving women as visitors in their families, seeing women as responsible for all deaths in the family, cultural rights for men to inherit property of the deceased including the widow, breadwinner role of men and others. On the other hand, the traditional rulers deliberately and carefully enumerated actions like cultural rights for men to inherit possessions of the deceased, perceiving women as visitors, subordinate role of women, seeing women as the cause of all deaths in the family, and others.

Also, a good number of the HIV positive women had horrifying experiences like beating, rejection, discrimination, chastisement and others from family members which resulted in denying them access to family resources. Based on this finding, it is safe to assume that HIV positive women studied arguably experienced domestic violence.

A good number of the HIV positive women spoke at length about the difficulties and frustrations they had faced in the past or they anticipated having to overcome in future. One of the most frequently mentioned experiences was their lack of access to health care services. They worried that during episodes of sickness that neither the hospital authorities nor their family members assist them financially to receive prompt treatment rather, that they would be scolded for being sickly. The inability of family members and hospital authorities to financially assist HIV positive women to receive adequate treatment during health problems shows that HIV positive women were not provided with their health needs. This finding suggests lack of care and support for the HIV positive women. Findings on lack of care and support for HIV positive women agrees with that of (3, 6, 7) and is at variance with that of [12,14] which documented increased care and support for people living with HIV/AIDS. This finding presupposes that the family members of HIV positive women are not aware of their basic needs. It is not to be over emphasized that HIV positive women need finance to meet the demands of purchasing anti-retroviral

drugs, eating balanced food, treating opportunistic infections, paying children's school fees and other family needs. Therefore HIV positive women should be allowed rights to family resources to enable them meet their health, financial and social needs. This will reduce the tendency of depending on others for assistance. Denying HIV positive women rights to family resources is at variance with the recommendations of (21, 22, 23) that supreme court ruling of Mojekwu to allow female's rights to inherit family resources be implemented.

In the present study, HIV positive women took two types of risks in order to earn their livelihood: acting as hired labourers in the farm, and having sex without condoms. The risks of acting as hired labourers could easily wear them down, reduce their immunity and further expose them to several infections especially opportunistic infections. Also, HIV positive women having sex without condoms increases actions that encourage HIV infection. The finding that HIV positive women engaged in unprotected sex agrees with that of [19, 20].

Although the traditional rulers had good knowledge of modes of HIV infection, their overall knowledge of health, financial and social needs of HIV positive women remained poor. They exhibited little or no concern for the welfare of HIV positive women. This is shown by the traditional rulers' attitude of linking constant illness and other life experiences of the HIV positive women with laziness and/or lack of zeal for the women to engage in elaborate farming to raise enough food for sustenance like others. This is also shown by their negative attitude of discouraging economic empowerment of women.

Conclusions

The findings of this research provide an introduction to problems HIV positive women encounter in their attempt to survive in the communities, as well as issues and concerns of stakeholders towards the wellbeing of HIV positive women and how these women cope with these problems. The most crucial need is the one the women identified themselves which is to create a society which will support their rights to access family resources to reduce their dependence on family members in their attempt to mitigate the demands of HIV infection.

Therefore, encouraging policies that would promote inheritance rights of HIV positive women could reduce the risks they take to survive in the communities. If HIV positive women are continuously denied rights to possess family resources, government's efforts to reduce the impact of HIV and AIDS on women would yield no significant result unless the government with the support of traditional rulers enact policies that would discourage some cultural practices that negatively influence women's existence by denying them access to family resources.

It is therefore plausible to recommend that government and traditional rulers should discourage factors that dehumanize women, pauperize them and deny them access to family resources. This recommendation is necessary because only the government with the help of traditional rulers can make policies capable of changing social norms, customs, and other practices that negatively influence rights to possess material. It is felt that since the traditional rulers decide what obtains in each community, that there is need to organize seminars and/or workshops to enlighten them on the benefits of empowering HIV positive women and allowing them access to family resources.

Many questions are generated by this study. Although this particular study was limited by the number of respondents, and the lack of generalization of result, there are clear indications that the concerns raised are as difficult as they are real. Marginalized groups of HIV infected women both in the rural and urban areas are perceived to have few resources and are at great risk of managing complex health problems including poverty. In addition, exploration of the experiences of HIV positive women in the communities would yield important information for HIV prevention.

References

1. Mphale, M.M. Emmanuel G.R. And Mokhantso G.M. HIV/AIDS and its impact on land tenure and livelihoods in Lesotho "Background paper for FAO/SARPN workshop on HIV/AIDS and land tenure, Pretoria South Africa 2002; 24- 25 June
2. Whiteside, A. poverty and HIV/AIDS in Africa. Third World Quarterly, 2002;23 (2) : 313 - 332.

3. World Bank "HIV/AIDS and gender equity." gender and development briefing Notes. Washington, D C: World Bank 03
4. Customary law manual , Enugu: Government printer 1977
5. Ogbu, O.N., Human rights law and practice in Nigeria: An introduction 1999 constitution Enugu: Cidjap publishers.
6. Macmillan, J. HIV/AIDS, the law challenges for women "paper presented at the FAO/SARPN workshop on HIV/AIDS and land tenure Pretoria South Africa: 2002; 24-25 June
7. Afigbo , A.E. Widowhood practices in Imo State of Nigeria and Africa. A Proceeding Of the Better Life programme for rural women's workshop Owerri, Imo State 1989.
8. Umeasiegbu, R.N. The way we lived: Igbo customs and stores London: Publishing Company. 1977
9. Cathi, A. "Using rights and the law to reduce women's vulnerability to HIV "HIV/AIDS Policy Law Rev. 2000; 5(4) 72.
10. Park, A.E.W., The source of Nigerian law, London: Sweet and Maxwell 1963.
11. Eze, O.C. Study on the right to adequate housing in Nigeria, Lagos: Shelter Rights Initiative 1996.
12. Deere, C. D. Leon, M. Empowering women: land and property rights in Latin America, Pittsburgh: University Press 2001.
13. Chukwuemerie, A.I. the inheritance rights of women under the Nigerian customary law: New developments and unresolved questions. Abia State University law journal 2003; . 8: 96-132,
14. Food and Agriculture Organization , gender and access to land, FAO land tenure Studies 4: Rome , Food and Agriculture Organization 2002.
15. Ngwira , N. Asiyatu, C. Ngeyi, K. and Edrinnie, K. Upholding Women's Property rights and inheritance rights in Malawi. Changes required to meet the challenges" paper presented to the 8th women world congress Kampala Uganda 2002;21-26 July
16. Anyanwu , F.C. Udo, C.O. , Okpala, P.N. socio-cultural practices as Correlates of Psychological disposition of widows in Imo State. Nigerian School Health Journal 1999; (1&2) . 11: 66-71.
17. Ubesie ,T. Odinala ndi Igbo Ibadan: Oxford University Press 1978.
18. Odusanya, O. O. and Alakija, W. HIV: knowledge and sexual practice amongst Students of a school of community health in Lagos, Nigeria. African Journal of Medicine and Medical Sciences 2004; .33 .(1) : 45-49
19. Drimie, S. "The impact of HIV/AIDS on land: Case studies from Kenya, Lesotho and South Africa" Synthesis report for the FAO Southern African Regional Office. Pretoria: Human Science Research Council 2002.
20. Muchunguzi, J.K. HIV/AIDS and women land ownership rights in Kagera Region Northwestern Tanzania "Background paper for Africa. FAO/ SARPN workshop on HIV/AIDS and land tenure Pretoria, South Africa: 2002; 24-25 June.
21. Oyajobi, O.U. Gender discrimination and fundamental rights of women in Nigeria, Journal of human rights law and practice. 1991;. 1, (1) 75-96.
22. Ezielo, J . women's right in Nigeria: problems and prospects in the new Millennium. A paper presented b at the workshop organized by National Human RIGHTS Commission on 'women and Law in Umuahia 2000:26th January,.
23. Customary law manual . Enugu: Government Printer , 1977.
24. Uche U. Ewelukwa post-colonialism. Gender customary injustice: widows in African Societies Human Rights Quarterly 2002; 24 , 424-486.
25. Eniola, A. the principles of African customary law Eniola publishers, Ogbomoso, Nigeria 1997.
26. Nwogugu, E.I. family law in Nigeria. Lagos: Heinemann Educational Books.1990.
27. Oyajobi, A.U. Gender discrimination and fundamental rights of women in Nigeria. Journal of human rights law and practice . Civil liberty organization lagos : 1991;. 1 (1).
28. Ipaye, O.A. some aspects of women and the law: the Nigerian experience. Journal of human rights law and practice. Civil Liberties Organization, Lagos: 1995 ; .5.(1).

Case report: Endorphins and diabetes mellitus

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CASE REPORT

Mr. X, is a 51 year old man known to be diabetic for 7 years. He is not hypertensive and has no history of cardiovascular diseases. He is using Gliclazide 80mg BID and metformin 500mg OD. His blood glucose is well controlled (HbA1c 6.5%), his blood pressure is also well controlled (132/75mmHg). All his lab investigations were within normal limits except his serum total cholesterol and total triglyceride (6mmol/L, 5mmol/L respectively).

His physical activity was limited and he is not keen about his diet control. He is working as an administrative staff member in one of the big companies in the region.

Mr X is a heavy smoker. He started smoking at the age of 20 years. He smoked 20 cigarettes per day.

On his last visit, his doctor advised him to quit smoking. Mr. X read about a new device which arrived at the local anti-smoking clinic called Silver Spike Point (SSP) and he asked his doctor about this device and if it may affect his blood glucose if he decided to use it.

Discussion

The previous scenario is a real scenario of one of our patients who plans to quit smoking after a long journey with cigarette smoking (31 years). In the following paragraphs I will explain the principles that stand behind the use of silver spike point in smoking cessation and its effect on glucose homeostasis.



The Silver Spike Point device (SSP)

The prevalence of smoking among diabetics

Cigarette smoking is the leading avoidable cause of mortality in the USA, accounting for 400,000 deaths each year. (1) The Resistance Atherosclerosis Study (IRAS) was a prospective study of the associations of insulin sensitivity and cardiovascular risk factors. In this study a test for association between smoking and diabetes was checked among 906 participants free of diabetes at baseline and followed for 5 years for diabetes incidence. From current smokers 25% developed diabetes at 5 years compared with 14% never smoking (OR 2.66, P=0.001). (2)

E S ford et al in their paper published in the Journal of Preventive Medicine in 2004 analyzed data from the behavioral risk factor surveillance system for 1990 - 2001. They found that the prevalence among adults with diabetes was 23.6% (Men 25.4%, Women 22.2%) in 1990 and 25.2 (men 24.8%, women 21.9%) in 2001. (3)

Wannamethee et al studied 7,735 men in the British Regional Heart Study and they found that cigarette smoking was associated with increased risk of diabetes after adjustment for confounders. (4)

It is known as Needle Free Acupuncture, developed in Japan in 1976 following a joint academic/industrial study between Osaka Medical College (Department of Anesthesiology) and Nihon Medix Company Limited (Figure1) . (5)

The device had many advantages in comparison with traditional acupuncture (Table 1).

The device is used by many antismoking clinics to help smokers to overcome the withdrawal symptoms resulting from smoking cessation.

The physiological action of the SSP

Clinical trials have shown that SSP low frequency electrical stimulation facilitates the discharge of endorphins (morphine like substances) as does traditional acupuncture. (6)

In 1999, clinical researchers reported that inserting acupuncture needles into specific body points triggers the production of endorphins. (7)

In another study, a high level of endorphins was noted to form in cerebrospinal fluid after patients underwent acupuncture. (8)

Also in another study, investigators showed a significant rise of plasma endorphin levels after electroconvulsive therapy (ECT) for treating depression. (9) (10)

The role of endorphins

The term endorphin consists of two parts; Endo (endogenous) and Orphis (Morphines) intended to mean "morphine - like substance" originating from within the body. (11)

Pain free
 Needle free
 Non invasive
 No danger of Infection or contagion
 Simple and straight forward technique
 Free from complications
 Suitable for many people Including hypersensitive patients, children and the elderly
 Opportunity of applying additional therapy techniques
 Wide Range of Points Can Be stimulated
 Eliminates the time consuming practice of needling

Table 1: Advantages of the SSP device over traditional acupuncture

Strenuous exercise
 Excitement
 Pain
 Death
 Orgasm
 Over exposure to sun
 Spicy food

Table 2: Stimuli for endorphins secretion

Endorphins are endogenous opioid polypeptide compounds. They are produced by the pituitary gland and the hypothalamus in vertebrates during certain circumstances and act via opioid receptors in the body (Table 2). (12)(13)(14)

Opioid neuropeptides were first discovered in 1975 by two independent groups of investigators.

The first group was led by John Hughes and Hans Kosterlitz who succeeded in isolating opioid neurotransmitters from the brain of a pig and call it enkephalins. (15)

The second group was led by Simantov R and Solomon H Snyder and they succeed in isolating these opioid neurotransmitters from the brain of calves. (16)

Until now, there are four types of endorphins created in the human body. They are named alpha, beta, gamma and sigma endorphins. These endorphins are differing in number and types of amino acids; they have between 16-31 amino acids in each molecule. (17)

Beta endorphins are the most powerful endorphins in the body. They are usually found in the hypothalamus and pituitary gland.

The actions of endorphins

All endorphins bind to the opioid receptors in the brain. They cleared very rapidly from the blood. Acupuncture is thought to result in the release of more endorphins. (18) Also some suggest that endorphins have a role in the development of obesity, diabetes and psychiatric diseases. (17)

Beta endorphins are released into the blood and into the spinal cord and brain from hypothalamic neurons. The beta endorphins that are released into blood cannot enter the brain in large quantities because of the blood brain barrier. Also, beta endorphin has the highest affinity for the U1-opioid receptor (Table 3). (19) (20)

Classically U receptors are presynaptic and inhibit neurotransmitter release, through this mechanism they inhibit the release of GABA and disinhibit

the dopamine pathways causing more dopamine to be released (19).

Opioid receptors have many other important roles in the brain and periphery (Table 4). (21)

Smoking and dependency

Cigarette smoking is a cycle of craving, smoking, calming and craving. Within seconds, smoking sends nicotine to the brain. Nicotine starts a series of biochemical reactions causing the release of dopamine and other substances giving the feeling of pleasure and calm. (22)

Evidence indicates that people smoke primarily to experience the psychological properties of nicotine and that the majority of smokers become dependent upon nicotine. (22) In humans, nicotine produces positive reinforcing effects including mild euphoria (23), increased energy, heightened arousal, reduced stress and anxiety and appetite suppression. (24)(25) Although nicotine produces its effects through nicotine acetylcholine receptors, other

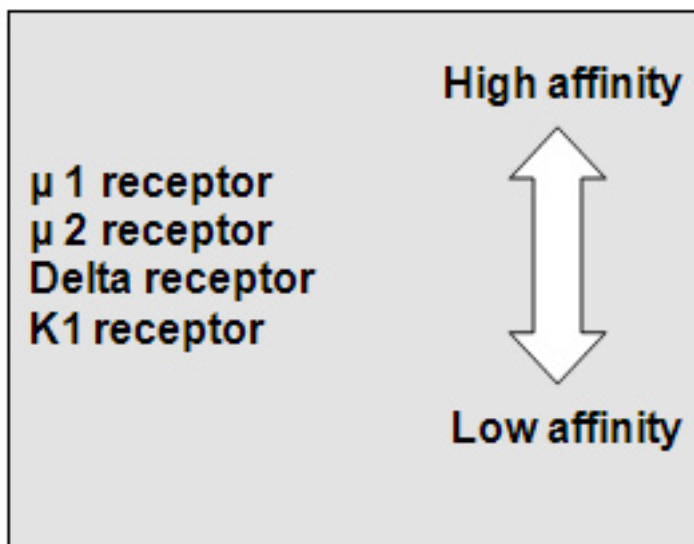


Table 3: Affinity of Opioid receptors to beta endorphin

neurological systems involved in nicotine reinforcement interact with the midbrain dopamine system. These systems include the opioid system.

Stimulation of the dopamine system appears to be of critical importance for acute positive reinforcing properties of nicotine.

Nicotine also affects the release of endogenous opioid peptides. (26) The endorphin system has been hypothesized to be involved in mood regulation, psychomotor stimulation, analgesia reproduction and temperature regulation. (27)

How can endorphins help in smoking cessation?

Endorphins compete with nicotine on the receptors responsible from positive reinforcement feelings. While the smokers begin to withdraw from the smoking habit, the endorphins produced by electrotherapy (SSP) continue to give the same feelings. After time the body restarts to secrete endorphins endogenously without the need of nicotine. In such way the smoker can quit smoothly without passing through the vicious cycle of craving, smoking, calming and craving.

The relation between endorphins and glucose homeostasis

In an animal model study, investigators examined the administration of beta endorphins introduced centrally on glucose homeostasis on a conscious dog. (28) Intracerebroventricular administration of beta endorphin (0.2mg/h) caused a 70% increase in plasma glucose.

The mechanism of hyperglycemia was thought through:

- Early increase of glucose production
- Lack of inhibition of glucose clearance

The changes explains the marked increases in plasma epinephrine (30 fold) and norepinephrin (6 fold) that occurred during infusion. Interestingly intravenous administration of beta-endorphin did not alter glucose homeostasis. The investigators in this study concluded that beta endorphins act centrally to cause hyperglycemia by stimulating sympathetic out flow and pituitary - adrenal axis. (28)

In another study (29), Paolisso G et al evaluated the effect of human beta endorphins on pancreatic hormone levels and on glucose metabolism in normal subjects. The study showed that infusion of 143 nmol/h beta endorphins in 7 subjects caused a significant rise in plasma glucose concentrations (+1.7 +0.3 mmol/L) which was preceded by a

significant increase in peripheral plasma glucagons levels (+44 +13ng). From this study the investigators concluded that naturally occurring opioid peptide beta endorphin produced hyperglycemic effects in man which appears to be mediated by Glucagon. The opioid seems to have no direct effect on glucose metabolism.

Back to animal model studies, an interesting study (30) aimed to determine whether supraphysiological levels of beta endorphin inhibit the ACTH and CRH response to insulin induced hypoglycemia in human subjects. The researchers in this study noted that IV infusion of beta endorphin increases glucose and delays the onset of hypoglycemia following insulin. (30)

Conclusion

Back to our patient, Mr X's doctor explained to him the previous information and encouraged him to try the SSP device but also advised him to monitor his blood glucose closely during the period of using the device. Also he was advised to contact his doctor if he noticed unexplained rising in his blood glucose.

References

- 1) American Diabetes Association. Clinical Practice recommendations. Diabetes Care; 27(1):S74-S75. 2004
- 2) Capri GF, Ronny AB, Deborah FF, David CG and Lynne EW. Smoking and incidence of diabetes among U.S Adults.

Receptor	Function
<p>Mu (μ) receptors (1, 2 and 3)</p> <p>μ 1</p> <p>μ 2</p>	<p>Supraspinal analgesia Physical dependence</p> <p>Respiratory depression Meiosis Euphoria Reduced GI motility Physical dependence</p>
Delta	<p>Analgesia Antidepressant effect Physical dependence</p>
Kappa receptors K1, 2 and 3	<p>Spinal analgesia Sedation Meiosis Inhibition of ADH release</p>

Table 4: Function of Opioid receptors

3) Earl S Ford, Ali H Makdad and Edward W Gregg. Trends in cigarette smoking among US adults with diabetes: findings from the Behavioral Risk Factor Surveillances System. Preventive medicine; 39:1238 - 1242. 2004

4) Wannamethee et al. Smoking as a modifiable risk factor for type 2 diabetes in middle aged men. Diabetes Care 24:1590-1595. 2001

5) <http://www.nihonmedix.co.jp/english/02about/advantage.html>

6) Cai-Lian. CuiLiu-Zhen and Wuand Fei Luo. Acupuncture for the Treatment of Drug Addiction. Neurochemical Research; 33(10): 2013-2022.2008

7) Napadow V,Ahn A,Longhurst J,Lao L,Stener-Victorin E, Harris R,Langevin HM. The Status and future of acupuncture clinical research. Journal of alternative and complementary medicine;14(7):861-9.2008

8) Clement - Jones V et al. Increased beta endorphin but not met-enkephalin levels in human cerebrospinal fluid after acupuncture for recurrent pain. Lancet 2(8201):946-9. 1980

9) Abenyakar S, Boneval F. Increased plasma [beta]-endorphin concentrations after acupuncture: comparison of electroacupuncture, traditional Chinese acupuncture, TENS and placebo TENS. Acupunct Med 1994;12(1): 21-3.

10) A. Weizman, I. Gil-Ad, D. Grupper, S. Tyano and Z. Laron. The effect of acute and repeated electroconvulsive treatment on plasma β -endorphin, growth hormone, prolactin and cortisol secretion in depressed patients. Psychopharmacology; Volume 93, Number 1: 122-126.1987

11) Dorland's illustrated medical dictionary 29th edition. Philadelphia: W.B.Saunders Co. 2000

12) RASMUSSEN Natalie Ann and FARR Lynne A. Beta-endorphin response to an acute pain stimulus. Journal of neuroscience methods; 2009, vol. 177, no2, pp. 285-288

- 13) D. V. Taylor, J. G. Boyajian, N. James, D. Woods, A. Chiciz-Demet, A. F. Wilson and C. A. Sandman. Acidosis stimulates beta-endorphin release during exercise. *J Appl Physiol* 77: 1913-1918, 1994.
- 14) Bancroft, J. (1984). Hormones and human sexual behavior. *Journal of Sex and Marital Therapy*, 10, 3-21
- 15) Hughes J, Smith T, Kasterlitz H, Fothergill L, Morgan B, Morris H. Identification of two related penta peptide from brain with potent opiate agonist activity. *Nature* 258(5536):577- 80.1975
- 16) Rabi Simantov and Solomon H Synder . Morphine like peptides in mammals with the opiate receptor. *Proc Natl Acad Sci USA* 73(7):2515-9.1976
- 17) Dalayeu JF, Nores JM, Bergal S. Physiology of beta endorphin. A close up view and review of the literature. *Biomedicine and pharmacotherapy*;47(8):311-20.1995
- 18) Ji-Sheng Han. Acupuncture and endorphins. *Neuroscience Letters*; 361(1-3): 258-261. 2004
- 19) Alistair D Corbett, Graeme Henderson, Alexander T McKnight and Stewart J Paterson. 75 years of opioid research: the exciting but vain quest for the Holy Grail. *Br J Pharmacol*. 2006 January; 147(S1): S153-S162.
- 20) Zhorov BS, Ananthanarayanan VS. Homology models of μ -opioid receptor with organic and inorganic cations at conserved aspartates in the second and third transmembrane domains. *Arch Biochem Biophys*. 37:31- 49, 2000.
- 21) MARTIN W.R. History and development of mixed opioid agonists, partial agonists and antagonists. *Br. J. Clin. Pharmacol*. 1979;7:273S-279S
- 22) Stalerman IP. Behavioral pharmacology of nicotine: multiple mechanisms. *British journal of addiction*;86:533-536.1991
- 23) Pomerleau CS and Pomerleau OF. Euphoriant effects of nicotine in smokers. *Psychopharmacology* 108:460-465.1992
- 24) Benowitz NL. Pharmacology of nicotine: addiction and therapeutics. *Annual review of pharmacology and toxicology* 56:597-613.1996
- 25) Stlerman IP and Javris MJ. The scientific case that nicotine is addictive. *Psychopharmacology* 117:2-10.1995
- 26) Pomerleau OF and Pomerleau CS. Neuroregulators and the reinforcement of smoking: towards a biobehavioral explanation. *Neuroscience and biobehavioral reviews* 8:503- 513.1984
- 27) Cesselin F. Opioid and anti opioid peptides . *Fundamental and clinical pharmacology* 9:409-433.1995
- 28) Radosevich PM, Lacy DB, Brown LL, William PE and Bumrad NN. Central effects of beta endorphins on glucose homeostasis in the conscious dog. *Am J Physiol*;256(2 Pt 1):E322-30.1989
- 29) Paolisso G et al. Primary role of glucagons release in the effect of beta endorphin on glucose homeostasis in normal man. *Acta Endocrinol (Copenh)*;115(2):161-9.1987.
- 30) WJ Inder, JH Liyese MJ. Ellis, M J. Evans and RA. Donald. The effect of beta endorphin on basal and insulin hypoglycemia stimulated levels of hypothalamic-pituitary adrenal axis hormones in normal human subjects.

Warfarin-Induced Skin Necrosis: A rare but serious complication

Case report

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ABSTRACT

Warfarin induced skin necrosis is a rare but serious complication of treatment with anticoagulants. Doctors should consider this reaction when suspicious skin lesions appear, regardless of the manner in which warfarin treatment was initiated; early detection and proper management are essential.

We present two cases of skin necrosis following treatment with warfarin.

Key Words: warfarin, skin necrosis, anticoagulants.

Introduction

Warfarin-induced skin necrosis (WISN) is a rare, unusual, and unpredictable complication of anticoagulant therapy. It occurs in 0.01 to 0.1 percent of warfarin-treated patients.

Anticoagulants are used frequently in the management of wide variety of medical diseases, so awareness and early detection and management of this rare complication are essential.

Case 1:

A 23-year-old female, previously healthy, with family history of DVT (deep vein thrombosis), two weeks post normal vaginal delivery she developed right leg swelling and pain, diagnosed to have popliteal and femoral vein thrombosis proved by Doppler ultrasound, she was started on clexan (low molecular weight heparin) and warfarin.

One week later she developed skin eruptions and discoloration on the dorsum of the right foot. Here INR was

within the therapeutic range, arterial Doppler ultrasound was free, and coagulation profile studies showed protein C and protein S deficiency.

The clinical impression was warfarin induced skin necrosis. Warfarin was discontinued and she was started on low molecular weight heparin.

She was referred to the plastic surgery department for wound care, underwent frequent debridement and dressings, and her wound was covered with split thickness skin graft.

Case 2.

A 19-year old female, previously healthy, with family history of DVT, one week post normal vaginal delivery started to complain of right leg pain and swelling. Doppler ultrasound showed extensive deep vein thrombosis.

She was started on heparin; two days later warfarin was added, five days later she had right loin skin discoloration and necrosis, her INR was within therapeutic range, abdominal and pelvic ultrasound and CT was free.

Coagulation profile studies showed Activated protein C resistance (Factor V Leiden). Warfarin was discontinued and she was started on low molecular weight heparin; also vitamin K and fresh frozen plasma was given.

Skin necrosis continued to progress and involved necrosis of subcutaneous tissue; plastic surgeon was consulted, patient underwent excision of necrotic skin and subcutaneous tissue with frequent dressings. Here the wound was closed primarily with small raw areas healed by secondary intention.

Discussion

The mechanism of action of warfarin involves inhibition of vitamin K-dependent coagulation factors. Inhibition of protein C and Factor VII is stronger than inhibition of the other vitamin K-dependent coagulation factors II, IX and X. This results from the fact that protein C and Factor VII have shorter half lives. This difference in effect is proportional to the initial dose of vitamin K-antagonist [1, 2].

As a result of this imbalance in coagulation factors inhibition, paradoxical activation of coagulation occurs, resulting in a hypercoagulable state and thrombosis. This results in blood clots that interrupt the blood supply to the skin, causing necrosis. Protein C is an innate anticoagulant, and as warfarin further decreases protein C levels, it can lead to massive thrombosis with necrosis and gangrene of limbs [1, 2]. Development of the syndrome is associated with the use of large loading doses at the start of treatment [3].

The prothrombin time (or international normalized ratio, INR) is highly dependent on factor VII, which explains why patients can have a therapeutic INR (indicating good anticoagulant effect) but still be in a hypercoagulable state [1, 2].



derivatives [6].



In one third of cases, warfarin necrosis occurs in patients with an underlying, innate and previously unknown deficiency of protein C. There have also been cases in patients with other deficiencies, including protein S deficiency, activated protein C resistance (Factor V Leiden) and antithrombin III deficiency[3,4].

Although the above mentioned explanation is the most accepted theory of pathogenesis, others believe that it is a hypersensitivity reaction or a direct toxic effect [3].

This syndrome is more often in obese, middle aged woman. The median age is around 54 years with male to female ratio 1:3 [5]. The onset of the drug eruption usually occurs between the third and tenth days of therapy with warfarin

Initial presentation involves pain and redness in the affected area. As they progress, lesions develop a sharp border and become petechial, then hard and purpuric. They may then resolve or progress to form large, irregular, bloody bullae with eventual necrosis and slow-healing eschar formation [6, 7, 8]. This syndrome can involve any area in the skin but more often in: breasts, thighs, buttocks and penis. In rare cases it can involve the fascia and muscles [7].

The differential diagnosis includes many conditions such as pyoderma gangrenosum or necrotizing fasciitis [9].

Treatment includes: discontinuation of warfarin, Vitamin K as an antidote to warfarin action, heparin or low molecular weight heparin (LMWH) can be used

to prevent further clotting, fresh frozen plasma or pure activated protein C also has been used [9,10,11].

Heparin and LMWH act by a different mechanism than warfarin, so these drugs can also be used to prevent clotting during the first few days of warfarin therapy and thus prevent warfarin necrosis (this is called 'bridging') [9].

The necrotic skin areas need proper wound care with frequent proper dressings. Healing can occur spontaneously with or without scarring. In severe cases surgical debridement and skin grafting are required. The leading cause of death is related to underlying disorders for which anticoagulation is started, for example, recurrent pulmonary embolism. [12].

References

1. McKnight JT, Maxwell AJ, Anderson RL (1992). "Warfarin necrosis". *Arch FAM Med* 1 (1): 105-8.
2. Berkompas DC. Coumadin skin necrosis in a patient with a free Protein S deficiency: Case report and literature review. *India Med* 1991; 84(11):788-91.
3. Hiers CL. Case presentation of Coumadin-induced skin necrosis. *J Arkansas Med Soc* 1993; 89(9): 443-4.
4. Verhagen H. Local hemorrhage and necrosis of skin and underlying tissues during anticoagulant therapy with dicumarol or dicumacyl. *Acta Medica Scandinavica* 1954; 148:453.
5. Kipen CS. Gangrene of the breast: A complication of anticoagulant therapy. *N Engl J Med* 1961; 265:638-40.
6. Brooks LW, Blais FX. Coumadin-induced skin necrosis. *J Am Osteopath Assoc* 1991;91(6):601-5.
7. Eby CS. Warfarin-induced skin necrosis. *Hematol Oncol Clin North Am* 1993;7(6):1291-300.
8. Essex DW, Wynn SS, Jin DK. Late onset warfarin-induced skin necrosis: Case report and review of the literature. *Am J Hematol* 1998; 57:233-7.
9. Gelwix TJ, Beeson MS. Warfarin-induced skin necrosis. *Am J Emerg Med* 1998; 16(5):541-3.
10. Ad-EI D, Meirovitz A, Weinberg A, et al. Warfarin skin necrosis: Local and systemic factors. *Br J Plast Surg* 2000;53:624-6.
11. Chan YC, Valenti D, Mansfield AO, Stansby G. Warfarin induced skin necrosis. *Br J Surg* 2000; 87:266-72.
12. De Franzo AJ, Marasco P, Argenta LC. Warfarin-induced necrosis of the skin. *Ann Plast Surg* 1995; 34:203-8.

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