

Letters to the Editor

Intrathecal Vincristine: Long-term Survivor of Potentially Fatal Chemotherapeutic Error

To the Editor: Vincristine (VCR), a vinca alkaloid, has been extensively used in cancer chemotherapy. Because of its anti-tumor activity and the relative lack of myelosuppression in conventional dosages, it is often used in combination with other chemotherapeutic agents for the treatment of various pediatric and adult malignancies.¹ The principal side effect that has limited the use of this agent is neurotoxicity, and is manifested by symmetrical peripheral mixed sensory-motor neuropathy.² The central nervous system is seldom affected because VCR is unable to penetrate a barrier.³ Other side effects include seizures, orthostatic hypotension, inappropriate anti-diuretic hormone secretion and worsening of pre-existing neurological disease.¹

Schochet et al. described the first case of accidental intrathecal VCR, causing a fatal ascending myeloencephalopathy in humans in 1968.⁴ A search of the literature revealed that such accidents are mostly fatal,⁴⁻¹⁰ except in a few cases in which rapid central nervous system (CNS) washout is carried out.¹¹⁻¹³ We describe the occurrence of another case and the treatment strategy that appears to have avoided the fatal consequence, and with less severe neurological complications.

Case History

A seven-year-old girl was diagnosed with central nervous negative acute lymphoblastic leukemia in June 1994 and was treated by the Children's Cancer Group (CCG) Protocol 1881. She was in remission and receiving maintenance chemotherapy with oral 6-mercaptopurine, methotrexate, and prednisone, as well as intravenous vincristine, and intrathecal methotrexate every third month. One year after the diagnosis, she inadvertently received her usual vincristine dose through an intrathecal route. The error was noted halfway through the injection when the patient had received approximately 0.5 vincristine. Immediate neurosurgical consultation was obtained and within 15 minutes, 75 mL of cerebrospinal fluid (CSF) was exchanged with Ringer's lactate.

Following this, the patient was immediately taken to the operating room in a sitting position where an intraventricular drain was inserted within an hour of the original event. The patient was then transferred to intensive care unit in a responsive state and the subarachnoid space continuously flushed with lactated Ringer's solution and fresh frozen plasma for 24 hours. At the same time, the patient was given 10 g of glutamic acid intravenously over a

24-hour period, followed by an oral dose of 500 mg, three times daily until the neurologic dysfunction stabilized.

Five days following the original event, the patient started to experience pain and weakness in the lower extremities, which progressed to complete paraplegia. She subsequently developed urinary retention and required intermittent catheterization. Urodynamic evaluation revealed an areflexic, hypotonic bladder, with minimal sensations noted at 200 mL. The patient was discharged one month after the mishap on intermittent catheterization and prophylactic Bactrim.

After consulting other colleagues, it was decided to resume the rest of the maintenance chemotherapy, though modified from CCG 1881 to St. Jude Study X. This protocol involves alternating chemotherapeutic agents without vincristine therapy. CNS prophylaxis was resumed with intrathecal methotrexate every three months. Presently, she is in remission and almost seven years off therapy. She did not regain motor function in the lower extremities and continues to have neurogenic bladder.

This case highlights once again the tragic consequences of procedural errors when handling chemotherapeutic agents. For the purpose of safety and optimizing patient care, specific institutional protocols must be developed and strictly adhered to for the administration of all chemotherapy. Physicians must therefore be aware of the danger, and comply with the preventive guidelines suggested by Shephard et al.,⁵ Williams et al.,⁷ and summarized by Fernandez et al.¹⁴ The only treatment measure which has shown some success in this kind of accident is immediate aggressive central nervous wash before vincristine binds to the brain tissue.^{11-13,15}

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References

1. Keplan RS, Wiernik PH. Neurotoxicity of anti-neoplastic drugs. *Semin Oncol* 1982;9:103-30.
2. Rosenthal S, Kaufman S. Vincristine neurotoxicity. *Ann Intern Med* 1974;80:733-7.
3. Jackson DV, Sethi VS, Spurr CL, McWhorter JM. Accidental vincristine administration. *J Neurol* 1982;288:209-13.
4. Schochet SS, Lampert PW, Earle KM. Neuronal changes induced by intrathecal vincristine sulphate. *J Neuropathol Exp Neurol* 1968;27: 645-58.

5. Shephard DA, Steuber CP, Starling KA, et al. Accidental intrathecal administration of vincristine. *Med Pediatr Oncol* 1978;5:85-8.
6. Styler H, Liwnicz B, Herrik MK, et al. Fatal myeloencephalopathy caused by intrathecal vincristine. *Neurology* 1980;30:867-71.
7. Williams ME, Walker AN, Bracikowski JP, et al. Ascending myeloencephalopathy due to intrathecal vincristine sulphate: a fatal chemotherapeutic error. *Cancer* 1983;204:11-7.
8. Gaides WG, Dikerman JD, Walter CL, et al. Intrathecal vincristine: report of fatal case despite CNS washout. *Cancer* 1983;52:799-81.
9. Bain PG, Lantos PL, Djurovic V, et al. Intrathecal vincristine: a fatal chemotherapeutic error with devastating central nervous effects. *J Neurol* 1991;238:230-4.
10. Al-Fawas IM. Fatal myeloencephalopathy due to intrathecal vincristine injection. *Ann Trop Pediatr* 1992;12:339-42.
11. Dyke RW. Treatment of inadvertent intrathecal injection of vincristine. *N Engl J Med* 1989;321:1270-1.
12. Zaragoza MR, Ritchey ML, Walter A. Neurologic consequences of accidental intrathecal vincristine: a case report. *Med Pediatr Oncol* 1995;24:61-2.
13. Michelagnolo MP, Bailey CC, Wilson I, Livingston J, Kinley SE. Potential salvage therapy for inadvertent intrathecal administration of vincristine. *Br J Hematol* 1997;99:364-7.
14. Fernandez CV, Esau R, Hamilton D, Fitzsimmons B, Pritchard S. Intrathecal vincristine: an analysis of reasons for recurrent fatal chemotherapeutic error with recommendations for prevention. *J Pediatr Hematol Oncol* 1998;20:587-90.
15. Bleck TP, Jacobsen J. Prolonged survival following the inadvertent intrathecal administration of vincristine: clinical and electrophysiologic analyses. *Clin Neuropharmacol* 1991;14:457-62.

Immediate Pulmonary Edema Following Carotid Endarterectomy

To the Editor: We read with interest the recent article entitled by El-Dawlatly on pulmonary edema following carotid endarterectomy.¹ He mentioned in his article the use of positive end-expiratory pressure (PEEP) of 10 mm Hg as a mode of treatment in a ventilated patient. The pressure in this situation is usually expressed in cm H₂O: 1 mm Hg = 1 Torr and 100mm H₂O = 7.35 Torr, so 10 mm Hg = 13.60cm H₂O. If it was not a misprint, then we think the accurate pressure used should have been 10 cm H₂O.

We also accept the use of PEEP in a ventilated patient but we could not understand why the patient needed to be ventilated in the first place. Was he in respiratory failure or was he ventilated for prophylaxes? The other question was why frusemide was used in this case since negative pressure pulmonary edema does not mean overhydration, and is simply dealt with by changing the dynamics of pressure equilibrium between the oncotic pressure in pulmonary capillary vessels and intra-alveolar pressure generated by inspiration against force of obstructed larynx.

This type of edema, as El Dawlatly rightly said in his paper, has been reported in children suffering upper airway

obstruction,^{2,3} and in adults.⁴ He mentioned that only a few cases have been reported, and that most of these cases were after laryngeal spasm with unilateral pulmonary edema, while the laryngeal obstruction happen while the patient is in lateral position, and that the spectrum of management is wide and depends on the clinical sequelae of the initial edema. We use only high airway pressure⁵ in the form of CPAP of 5 cm H₂O to revert negative pressure pulmonary edema, and recently had a case of a Saudi male who presented for paraumbilical hernia repair with no previous history of respiratory and cardiac diseases. He had three previous anesthetics without any problem. He received

FIGURE 1. Portable chest x-ray immediately after admission to recovery room and soon after settling the episode of severe laryngeal spasm.

FIGURE 2. Portable x-ray in the ICU 6 hours after initiation of CPAP of 5 cm H₂O through facemask applied tightly with re-breathing bag.

premedication consisting of 2 mg lorazepam orally 2 hours before surgery. He was induced with 180 mg propofol, 100

mg fentanyl and 40 mg atracurium. The trachea was intubated with size 8.5-cuffed endotracheal tube (ETT) without difficulties. The tube position was checked and asserted to have bilateral lung ventilation. The anesthesia and surgery were uneventful. When the patient was recovering, he was positioned in the right lateral position, and when he awoke, he apparently tried to pull the tube but the anesthesiologist restrained him and after oropharyngeal suction, the ETT was removed. Saturation of the hemoglobin as shown on pulse oximetry monitor was 100%. Immediately after extubation, the patient went into laryngeal spasm, which hindered his ventilation and started to desaturate. He was given 100% oxygen through anesthetic circuit with face mask fixed tight. When his saturation reached a minimal of 67%, xylocaine 2% in a dose of 70 mg was given intravenously. This helped to ease

the laryngeal spasm and coordinated respiration was possible. The larynx was visualized to see if there was a foreign body or secretion. A clear thick secretion was seen and was cleared by suction. Hemoglobin oxygen saturation rose to 88%. The patient was moved to the recovery room where the diagnosis of negative pressure pulmonary edema due to aspiration of secretion causing laryngeal spasm was made, and was confirmed by chest x-ray, which showed right-sided faint infiltration (Figure 1). Blood gases done showed hypoxemia with normocarbia. Respiratory physiotherapy, racemic epinephrine and salbutamol nebulization was given. CPAP of 5 cm H₂O was applied through facemask, which helped to restore the saturation to 100% (Figure 2). Prophylactic antibiotics were started on the assumption that the laryngeal spasm may be induced by aspiration of secretions. The patient was moved to the intensive care unit to continue therapy. Within 6 hours, he showed rapid improvement and was discharged within 24 hours.

The right positioning of the tube, the laryngeal spasm, and the verification of the absence of one lung intubation during surgery helped us in reaching a quick diagnosis. The examination of the larynx which showed no gastric regurgitation helped to reduce the possibilities to only one of simple shocking with secretions. If such a possibility happened in a recovering patient, it could have produced exaggerated laryngeal spasm.

There is an ongoing debate in anesthesiology regarding extubation during deep or light anesthesia to prevent laryngeal spasm. It is recommended to treat the intubation and extubation of the trachea with due care and avoiding rough handling. Extra care should be taken in patients with recent history of upper airway infection. Their airways are hyperactive, and the slightest irritation would produce excessive response.

In our patient, we believe that oropharyngeal suction during recovery from anesthesia and the presence of small

amount of secretion around the cuff were the most likely cause. The patient recovered fully with supportive measures and CPAP.

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References

1. El-Dawlatly AA. Immediate pulmonary edema following carotid endarterectomy. *Ann Saudi Med* 2001;21:75-6

Reply

To the Editor: I would like to thank Takrouri et al. for expressing interest in my article and drawing the attention to the expression of PEEP in cm H₂O instead of mm Hg. For academic purposes, the PEEP has to be expressed in cm H₂O, however, in my case the difference between mm Hg and cm H₂O was only +3, which I think, has no clinical significance, especially if we know that most modern ventilators have a built-in PEEP of +2 to 3. As I indicated in my report, the spectrum of treating NPPE is wide, and depends on the severity of upper airway obstruction and the degree of desaturation.¹ Although the SPO₂ in my case dropped to 80%, with direct laryngoscopy there was moderate epiglottis edema, which caused glottic closure upon inspiration and hence NPPE. Moreover, following carotid endarterectomy (CEA) surgery, and due to edema of the tissues and compression elicited on the vagus and hypoglossal nerves in the neck and its branches, laryngospasm and difficulty in breathing are known immediate complications. Therefore, re-intubation of the trachea was undertaken. Similar cases have been reported where re-intubation and controlled ventilation with PEEP were used with successful outcome.^{2,3} The use of furosemide in my case was indicated. I think whether it is cardiogenic or non-cardiogenic pulmonary edema, furosemide if given, will speed the recovery of the patient and this has been supported in many published reports.^{4,5} Finally, NPPE usually clears rapidly with supportive care. Cardiogenic pulmonary edema and aspiration must be ruled out. Treatment is directed at correction of hypoxia with supplemental oxygen and use of diuretics (furosemide). Occasionally and depending on the severity of symptoms, patients may require intubation.

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References

1. Lathan SR, Silverman ME, Thomas BL, Waters WC. Postoperative pulmonary edema. *South Med J* 1999;92:313-5.
2. Andersen C, Kancir CB, Nielsen KD. Laryngospasm-induced pulmonary edema. *Acta Anaesthesiol Scand* 1988;32:710-1.
3. Bourke AM. Unilateral pulmonary oedema following postextubation laryngospasm. *Anaesthesia* 1997;52:928.
4. Scarbrough FE, Wittenberg JM, Smith BR, Adcock DK. Pulmonary edema following postoperative laryngospasm: case reports and review of the literature. *Anesth Prog* 1997;44:110-6.
5. O'Connor D. Post obstructive pulmonary edema. *Anaesth Intensive Care* 2000;28:452-3.

Leech as a Rare Cause of Vaginal Bleeding in an Adolescent

To the Editor: Various conditions may induce vaginal bleeding during adolescence. Leech infestation is an extremely rare cause of this presentation. The aquatic leeches, usually species of *Limnatis*, are injurious to humans. The larger species suck the blood of bathers. The smaller ones, usually ingested in drinking water, infest the respiratory or digestive passages. At times, they invade the vagina, urethra and eyes of bathers. The painless and often unnoticed wound caused by the bite bleeds readily because of an anticoagulative secretion, hirudin, and heals slowly.¹ A *Medline* search dating back to 1960 found only three cases of vaginal bleeding due to leech infestation.

Case Report

A 12-year-old girl was referred to Niknafs Maternity Center, with mild-to-moderate vaginal bleeding of one-month duration. The medical history revealed that the bleeding started after travel to a rural area near Kerman City, Iran. Two weeks before admission, she had presented to another physician and had been treated with oral contraceptive pills (4 pills per day for 7 days) without investigation. However, the bleeding persisted and had become heavier within the last week before admission. Menarche had not occurred as yet and there was no history of sexual intercourse, blood dyscrasia and similar problem in the past.

On physical examination, the patient looked pale with a pulse rate of 85/min and blood pressure of 100/75 mm Hg.

There was no evidence of petechia, purpura or ecchymosis on her body. Secondary sexual characteristics were consistent with Tanner classification 3-4.² Investigation for platelet count, bleeding, clotting and prothrombin times and transabdominal pelvic ultrasound were all within normal limits. Pregnancy test was negative and complete blood count revealed a hemoglobin value of 8.5 g/dL. It was decided to perform vaginal examination under general anesthesia. External genitalia and hymenal ring were intact, but deep in the posterior fornix of the vagina, a 3 cm darkish mass was identified. After removal by a ring forceps, the mass was confirmed to be a leech. Bleeding stopped after 24 hours and the patient was discharged on the third day of admission.

Discussion

Although abnormal vaginal bleeding in adolescents frequently happens as a result of dysfunctional uterine bleeding, other causes must be carefully ruled out. Polyps, leiomyomata, vaginal adenosis, coagulation disorders, complications of pregnancy, pelvic inflammatory disease and malignancies of genital tract are alternative pathologies. In such situations, extensive investigations should be

carried out. The differential diagnosis may be assessed by pelvic examination (more likely to be inadequate in an adolescent than an older woman), laboratory tests and pelvic ultrasound.²

In our patient, paraclinical investigation did not reveal any pathologic findings. With respect to the ethnic considerations in our country (vaginal examination is not allowed in virgins), pelvic examination was avoided until it was absolutely necessary. Because the patient had not responded to hormonal medication and there was no remarkable finding in investigations, a decision on performing a vaginal examination under general anesthesia was made to exclude alternative pathologies. On this examination, leech was identified to be the cause of the bleeding.

The ectoparasitic leeches comprise the parasitic members of the phylum. The species of medical importance are either aquatic or terrestrial. They have variously sized, muscular, often pigmented oval bodies with a tough cuticle, suckers at both ends, hard jaws, and a muscular pharynx.¹ Many people who have experienced the delights of immersing their naked body in a muddy-bottomed river or pond are familiar with leeches. In Southeast Asia, leeches cause much blood loss as well as ulceration and inflammation of the bites, which continue to bleed even after the leeches are filled to repletion and drop off.³ There are only few reports on the settling of this parasite in the vagina, respiratory and urinary tracts.⁴⁻⁷ Leeches may be

removed after loosing their hold, by applying a local anesthetic, a strong salt solution, or a lighted match.¹

It can be concluded that leech infestation had occurred in our patient by the contaminated stream water. This rare cause of adolescent vaginal bleeding should be kept in mind especially in areas where leech infestation is prevalent.

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References

1. Brown HW, Neva FA. The Nematheintes or roundworms. In: Basic Clinical Parasitology, 5th edition. London: Prentice-Hall International, 1993:99-103.
2. Berek JS, Adashi EY, Hillard PA. Novak's Gynecology, 12th edition. Baltimore: Williams & Wilkins, 1999:333-6, 772-4.
3. Chandler AC, Read CP. Introduction to Parasitology. Singapore: Toppan, 1981:240-3.
4. Hailmariam B. Postmenopausal vaginal bleeding due to vaginal wall leech infestation. Ethiopia Med J 1995;33:183-5.
5. Turner FM. Pharyngeal leeches. Lancet 1969;27:2:1400-1.
6. Keegan HL, Radke MG, Murphy DA. Nasal leech infestation in a man. Am J Trop Med Hyg 1970;19:1029-30.
7. Hamid MS, Mohd Nor GR. Severe urological complication of leech bite in the tropics. Br J Urol 1996;77:164-5.

Serum Lipid Concentrations and Indices of Obesity among Adult Subjects of the West Indies

To the Editor: Obesity constitutes a major public health problem both in developed and developing countries.¹ The prevalence of obesity or overweight is higher in women than in men, with rates as high as 36% in Nigerian women² and up to 50% in Bantu women in South Africa.³ Other studies in Latin America, the Caribbean and the Middle East have shown that about 30% of the population in these countries may be classified as being overweight.⁴ In many of these developing countries, both underweight and overweight people can be seen, although excess weight appears first among the affluent and then among the low-income classes.⁴ Several studies have shown that the main causes of overweight are a nutritional transition to lipid-rich diets and reduced physical activity, especially among urban dwellers.^{1,5,6}

Trinidad and Tobago is a rapidly developing country with stable economy. Previous reports have shown high prevalence rates of obesity and increased blood lipid concentrations among diabetic⁷ and apparently healthy subjects.⁸ Given the economic costs of management of obesity-related diseases, it is believed that surveillance and prevention programs are necessary to prevent or reduce obesity/overweight and dyslipidemia in developing

countries. Thus, indices of obesity and dyslipidemia were assessed in apparently healthy workers and students in a multidisciplinary institution in Trinidad, West Indies.

Subjects and Methods

One hundred and fifty-six adults aged between 17-40 years were recruited through poster advertisements and announcements in Eric William's Medical Sciences Complex in Trinidad, West Indies. All subjects gave informed voluntary consent to participate in the study, and our Institutional Ethics Committee approved the protocol. The subjects provided answers to various questions posed in a questionnaire on: age, ethnic group, occupation, previous record of body mass index (BMI) and family history of diabetes. Recordings on waist size (cm) at the level of the umbilicus with the patient standing and breathing normally, and hip circumferences (cm) at the largest projection of the buttocks, were obtained by tape measurement. Weight (kg) and height (m) were measured in light clothing without shoes. Of the initial 156 participants, 78 subjects volunteered for lipid screening, and subsequently a fasting venous blood sample was taken from each volunteer for plasma glucose and serum lipid measurements. Plasma glucose, serum total cholesterol (T-chol), triglycerides (TG) and high-density lipoprotein-cholesterol (HDL-chol) concentrations were measured using commercial kits (dry slides) in multi-channel auto-analyzers (Johnson & Johnson, Ortho-Clinical Diagnostics Inc.,

TABLE 1. Background characteristics, anthropometric indices and lipid levels of the subjects.

Parameters	All n=156 (%)	Male n=46 (%)	Female n=110 (%)
Employed/unemployed	69/87	17/28	52/59
Positive family history of diabetes	96 (61.5)	30 (66.7)	66 (59.5)
Previous record/knowledge of BMI	26 (16.7)	7 (15.6)	19 (17.1)
African origin	46 (29.5)	9 (20.0)	37 (33.3)
East Indian origin	80 (51.3)	30 (66.7)	50 (45.0)
Mixed ethnicity	30 (19.2)	6 (13.3)	24 (21.6)
Age (year)	26.7±0.6	25.2±1.1	27.3±0.8
Weight (kg)	62.8±1.2	70.7±2.2	59.6±1.3**
Height (m)	1.6±0.1	1.7±0.01	1.6±0.01**
Body mass index (kg/m ²)	23.1±0.3	23.6±0.7	22.9±0.4
Waist circumference (cm)	79.6±0.9	85.6±1.6	77.2±1.1**
Hip circumference (cm)	96.7±0.8	97.8±1.4	96.3±0.9
Waist/hip ratio	0.82±0.01	0.87±0.010	0.80±0.01*
Plasma glucose (mmol/L)	4.3±0.1	4.6±0.4	4.1±0.04
Total cholesterol (mmol/L)	4.7±0.1	4.6±0.3	4.7±0.1
Triglycerides (mmol/L)	1.0±0.05	1.2±0.2	0.9±0.04*
HDL-cholesterol (mmol/L)	1.5±0.03	1.3±0.1	1.6±0.03*

LDL cholesterol (mmol/L)	3.0±0.1	3.1±0.3	3.0±0.1
Obese (BMI ≥30 kg/m ²)	12 (7.7)	4 (8.9)	8 (7.3)
Underweight (BMI <20 kg/m ²)	42 (27.1)	9 (20.0)	33 (27.1)
Total cholesterol >5.2 mmol/L	22 (28.2)	5 (22.7)	17 (30.4)
HDL cholesterol <0.90 mmol/L	2 (2.6)	2 (9.1)	0

P*<0.05; *P*<0.01 for comparison between male and female subjects, results are expressed as mean±SE; BMI=body mass index; HDL=high density lipoprotein; LDL=low density lipoprotein.

Rochester, USA). Low-density lipoprotein-cholesterol (LDL-cholesterol) was calculated. Dyslipidemia was defined as T-cholesterol levels >5.2 mmol/L or LDL-cholesterol >3.37 mmol/L or TG >2.26 mmol/L or HDL-cholesterol <0.90 mmol/L.⁹ Obesity was defined as body mass index (BMI) >30 kg/m²; overweight as BMI >25 kg/m² (females) and BMI >27 kg/m² (males), and underweight as BMI <20 kg/m².¹⁰ The Statistical Package for the Social Sciences (SPSS) software was used in all analyses and a *P*-value of <0.05 was considered statistically significant. The results are shown in Table 1.

Results and Discussion

The results of the study on apparently healthy workers and students at the Medical Sciences Complex in Trinidad, West Indies, showed that 83% of the subjects had no knowledge or record of their BMI prior to the study. Subjects who were overweight (17.4%) and underweight (27%) were identified, 62% of the subjects had positive family history of diabetes, and 28.2% hypercholesterolemia was observed. These findings constitute predictors of obesity-related diseases such as diabetes, hypertension and cardiovascular diseases. The finding that a majority of the participants had no knowledge or record of their BMI prior to the study suggests that the problem of obesity or overweight has not yet been widely recognised.¹¹

Obesity is a serious metabolic disorder, with annual estimated cost of US\$3 billion in Britain¹² and US\$69 billion in the USA.¹¹ It has been suggested that BMI should be routinely assessed in primary care clinics for both adults and children, and that obese patients should be treated with drugs similar to any other chronic disease.¹² This suggestion has come at a time when surgical treatment of obesity is becoming increasingly popular in the United States as a means of weight reduction.¹³ It is rather interesting that both overweight and underweight subjects were observed among the participants, a feature previously recognized as common in populations with records of malnutrition and nutrition-related disorders.⁴ The observation of obesity and overweight may be related to nutritional transition to lipid-rich diets, reduced physical activity and less energy expenditure.^{1,5,6} However, the high prevalence of underweight (27%), especially among female participants may not be explained as malnutrition-related, given that the participants were either employed or medical

students from affluent backgrounds. It is plausible that the observed underweight may be more related to overzealous quest for weight control such as strict adherence to dietary prescriptions and vigorous exercise in gymnasia, although this attribute was not ascertained in the questionnaire.

Nonetheless, the consequences of such extreme weight reduction may be disastrous following the recent report associating extremes of BMI with reduced probability to achieving pregnancy in women receiving assisted reproduction treatment.¹⁴ Although that report of Wang et al. is yet to be confirmed in other populations, the present finding is worrisome, especially as a greater proportion of the underweight subjects are females.

Obesity or overweight is one of the important risk factors for metabolic syndrome X, which includes, among others, diabetes, hypertension, dyslipidemia and insulin resistance.^{15,16} Perhaps, obesity is the greatest risk factor for diabetes, because a BMI >35 kg/m² increases diabetes risk by 93-fold in women and by 42-fold in men.¹¹

The high prevalence of a positive family history of diabetes, particularly among females, has significant health implications for the black population and the developing countries. For instance, several previous studies in black populations have shown that the prevalence rates of obesity were higher in women than in men,¹⁻³ and that obese black women tend to have reduced rates of glucose disposal and greater degree of insulin resistance than obese white women.¹⁷ Again, the high prevalence rate of hypercholesterolemia, especially in females, is an additional cardiovascular disease risk burden.¹⁶ Granted that a high proportion of our subjects were underweight (27%), subanalysis of our data (not shown) showed that the observed hypercholesterolemia was primarily in obese and overweight participants. The implication is greater risk for diabetes and cardiovascular disease among the obese and overweight female subjects.

In conclusion, both obesity and underweight conditions were identified in our study, similar to what prevails in other developing populations. It is proposed that screening for indices of obesity and hypercholesterolemia should be incorporated into the routine clinical examinations in all vulnerable populations to facilitate early identification, evaluation and treatment of obesity-related disorders such as diabetes, hypertension and cardiovascular diseases.

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References

1. Gurney M, Gorstein J. The global prevalence of obesity: an initial overview of available data. *World Health Stat Q* 1988;41:251-4.
2. Johnson TO. Prevalence of overweight and obesity among adult subjects of an urban African population sample. *Br J Prev Soc Med* 1970;24:105-9.
3. Sloan C. Weight, height and skinfold thickness of Zulu adults in Durban. *S Afr Med J* 1960;34:505-9.
4. Delpeuch F, Maire B. Obesity and developing countries of the South. *Med Trop* 1997;57:380-8.
5. Hirsch J, Leibel RL. New light of obesity. *N Engl J Med* 1988;318: 509-10.
6. Ravussin E, Lilloja S, Knowler WC, Christain L, Freymond D, Abbott WGH, et al. Reduced rate of energy expenditure as a risk factor for body weight gain. *N Engl J Med* 1988;318:467-72.
7. Ezenwaka CE, Davis G. Increased cardiovascular risk factors in newly diagnosed type 2 diabetic patients in a primary health care center in Trinidad. *Diabetes Res Clin Pract* 2000;50:137-45.
8. Ezenwaka CE, Premanand N, Orrett FA. Studies on plasma lipids in industrial workers in Central Trinidad and Tobago. *J Natl Med Assoc* 2000;92:375-81.
9. The Expert Panel. Report of the National Cholesterol Education Programme. Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults. *Arch Intern Med* 1988;148:36-69.
10. World Health Organization Working Group. Use and interpretation of anthropometric indicators of nutritional status. *Bullet WHO* 1986; 64:929-41.
11. Jung RT. Obesity as a disease. *Br Med Bull* 1997;53:307-21.
12. Bower H. Guidelines to tackle the tidal wave of obesity. *BMI* 1996; 313:1225.
13. Baxter J. Obesity surgery: another unmet need. *BMJ* 2000;321:523-4.
14. Wang JX, Davis M, Norman RJ. Body mass and probability of pregnancy during assisted reproduction treatment: retrospective study. *BMJ* 2000;321:1320-1.
15. Reaven GM. Role of insulin in human disease. *Diabetes* 1988;37: 1595-607.
16. DeFronzo RA, Feranninni E. Insulin resistance: a multi-faceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidaemia and atherosclerotic vascular disease. *Diabetes Care* 1991;18:173-94.
17. Van der Merwe MT, Crowther NJ, Schlaphoff GP, Gray IP, Joffe BI, Lonroth PN. Evidence for insulin resistance in black women from South Africa. *Int J Obesity Relat Metab Disord* 2000;24:1340-6.