CLASSICAL KAPOSI SARCOMA: *A CASE REPORT*

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ABSTRACT

An 82 years old woman was seen in July 2001,with a firm rubbery purple angiomatous plaques on the palmer aspect of the right hand and the dorsum of the right foot. The patient was complaining of persistent moderate pain and mild edema of the hand and foot. She also had hypertension, ischemic heart disease, and chronic renal failure. Skin biopsy was done and histopathological examination revealed classical kaposi sarcoma. Because of her bad general condition, the treatment was limited to analgesics and sedatives. This case was reported because of the rarity of Kaposi sarcoma in the city of Basrah.

INTRODUCTION

The most frequently encountered neoplasni in HIV-positive patients is an aggressive variant of Kaposi's sarcoma (KS)[1-4]. This tumour was first described by the Hungarian dermatologist Moritz Kaposi in 1872[1,4]. There are four recognized clinical subsets of this tumor. The sporadic or classical form is rare and affects elderly patients of predominant Mediterranean or Jewish origin. The lesions usually start with a reddish nodule on the lower extremity associated with lymph oedema. The disease is generally indolent, visceral involvemement occurs late, and the median survival ranges from 10 to 15 years. In 1950 an African variant was described; in equatorial Africa this form is endemic and relatively frequent (3-9 per cent of all neoplasms). This variant is usually more aggressive than the classical Kaposi's sarcoma with multiple localizations, and the median survival is 5 to 8 years. Kaposi's sarcoma in HIV-positive patients, which has been defined as epidemic KS, develops predominantly in homosexual men and to a much less extent in other risk groups such as intravenous drug users, hemophiliacs, polytransfused heterosexuals, and children of infected mothers. The incidence of this neoplasm in patients with AIDS ranges from 3 to 46 per cent for the various risk groups. Kaposi's sarcoma has also been reported in organ transplant recipients treated with immunosuppressive agents, and in patients treated with alkylating agents for multiple myeloma[4]. An infectious etiology for Kaposi sarcoma has been suspected since many years[12,13]. Recently a strong correlation has been found between one herpes virus and the development of all forms of Kaposi sarcoma. This virus was termed Kaposi sarcoma associated herpes virus and now is known as human herpes virus 8 (HHV-8)[14-28]. This virus has also been incriminated for the development of primary effusion lymphoma and multicentric castleman's disease[26]. HIV infection, corticosteroids. and renal transplantation are thought to provide altered immunity states (hat favor the replication of an already existing HHV-8 infection[29-3]. The histopathological findings, treatment, and prognosis of KS depend upon the stage and the clinical type of the tumour.

CASE REPORT

An 82-years old woman had a pigmented skin lesion on her right palm for more than ten years, which was diagnosed as melanoma and excised completely at that time. That lesion had recurred again and grew slowly and progressively. She then (developed hypertension, ischemic heart disease, and chronic renal failure. She was almost bed-bound and dependent on other family members for her daily activities. At presentation in July 2001 she had another skin lesion on the dorsum of her right foot of six months duration. She was complaining of pain in the hand and foot with poor appetite and general ill health. Physical examination revealed 4x3 cm purplish plaque consisting of multiple nodules on the right palm, and a similar lesion on dorsum of the right foot. The lesions were tender, rubbery, smooth surfaced plaques (Fig-1). New lesions appeared in (the face and neck consisting of 2x2cm, tender subcutaneous nodules. Serologic tests for HIV were negative. Skin biopsy was done under local anesthesia from the right hand, which revealed a predominance of spindle cells with slits and spaces consistent with classical kaposi sarcoma (Fig-2). The patient has refused admission to hospital and treatment at home was mainly supportive and aimed at pain relief.

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Then she deteriorated rapidly and died at home due to KS progression.

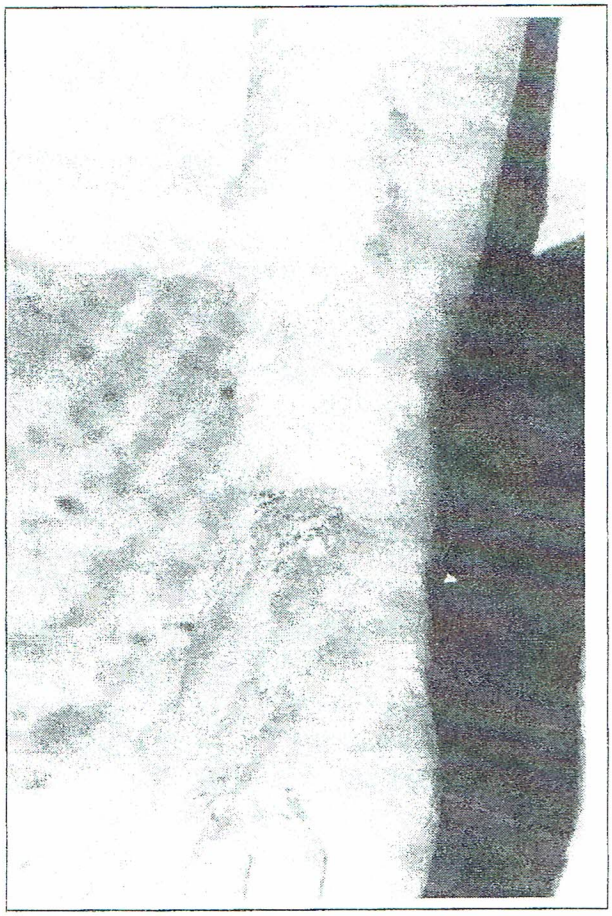

Fig 1. KS in the right hand.

Fig 2. KS in the right foot.

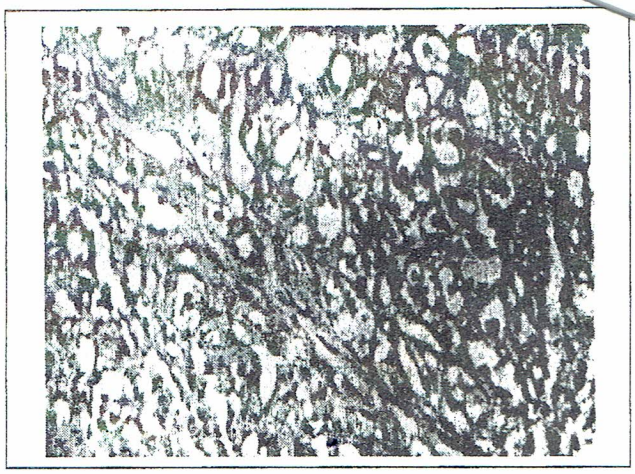

Fig 3. Histological features of skin biopsy from right hand.

DISCUSSION

Kaposi sarcoma is a disease of the reticuloendothelial system arising from both vascular and lymphatic endothelium[1,3]. The disease seems to be rare among dermatological cases in our community, as the last reported case in our city was a woman presented in the scientific conference of Basrah province in 1993[8]. The sporadic type is uncommon slowly progressive multifocal tumor, mainly found in elderly males of Eastern-European, Jewish, and Mediterranean origin, usually appearing on the feet or lower legs[2-6]. Although the disease is more common in elderly males, our present patient (and the last reported case mentioned above) is an elderly female. Further cases tracing is required to predict female sex predominance. In the epidemic form young males are commonly affected, but young females are also susceptible, and the disease is more aggressive in them[14]. Our patient was negative for HIV antibodies. A low level of sex hormones has been found in HIV-positive and HIV-negative women, but the susceptibility to KS cannot be linked to the sex hormones levels[14]. Although classical KS usually starts in the lower limbs, it has been started in the hand in our patient, and then it involved the lower limb more than ten years later. But this lady has typically demonstrated the indolent and slowly progressive course of classical KS, as compared to the aggressive epidemic type. This case also gives a lesson; not to confuse KS with melanoma. We think that this case was misdiagnosed in its first presentation because of the rarity of KS in our community, and because at that time (before

worldwide. Kaposi's sarcoma has been reported in renal transplant recipients, but not in renal failure patients. It develops in these patients after immunosuppressive therapy, and may improve when this therapy is interrupted[4]. The development of renal failure in this lady, being an immunosuppressing state, may have played a role in the recurrence and progression of KS. There are reports indicating a high incidence of HHV-8 infection in patients with renal failure that increase with increasing age[33,34], but we had no facility to test for HHV-8 in this old lady.

EFERENCES

1. John H, Pestalozzi DM, Hauri D. Kaposi sarcoma of the glans penis with meatal obstruction. Case report and literature review.Swiss-Surg. 1996; (3): 134-136.
2. Lecker S, Melbye M, Ugesky Laeger. Kaposi sarcoma: An epidemiological perspective. Ugeskr-Laeger.1995; (38): 5232-5236.
3. Schwartz R. Kaposi sarcoma. Advances and perspectives. J-Am-Acad -Dermatol. 1996; 34:804-814.
4. Monfardini S. Epidemic Kaposi's sarcoma; in Pcckiuan-M; Pinedo-H; Vcronesi-U. Oxford textbook of oncology. Oxford university press, Oxford. 1995; 2:1896-1901.
5. Friedman-Kein AE, Saltzman BR. Clinical manifestations of classical, endemic African, and epidemic AIDS-associated Kaposi's sarcoma. J-Am-Acad-Dermatol. 1990; 22:1237.
6. Tappero JW, Conant MA, Wolfe SF, et al. Kaposi's sarcoma: Epidemiology, pathogenesis, histology, clinical spectrum, staging criteria and therapy. J-Am-Acad-Dermatol. 1993; 28:371.
7. Wolf K. The enigma of Kaposi's sarcoma: An answer at last. Clin-Exp-Dermatol. 1992; 17:146.
8. Al-Rubiay K, Al-Timimi A. Kaposi sarcoma in south of Iraq. The scientific conference of the province of Basrah; 24th-25th, 1993.
9. Karasek MA. Origin of spindle-shaped cells in kaposi sarcoma. Lymphology. 1994; 27(1): 41-44.
10. Ryan TJ. Grip and stick and the lymphatics. Lymphology. 1990; 23 (2): 81-84.
11. Trattner A, Hodak E, David M; et al. The appearance of kaposi sarcoma during corticosteroid therapy. Cancer. 1993; 72 (5): 1779-1783.
12. Bendsoe N, Dictor M, Blomberg J, et al. Increased incidence of kaposi sarcoma in Sweden before the AIDS epidemic.Eur-J-Cancer.1990; 26 (6): 699-702.
13. Sinkovics JG. Kaposi's sarcoma: its oncogenes and growth factors. Crit-Rev-OncolHematol. 1991; 11(2): 87-107.
14. Schofer H, Roder C. Kaposi sarcoma in Caucasian women. Clinical, chemical, laboratory and endocrinologic studies in 8 women with HIV-associated or classical Kaposi sarcoma. Hautarzt. 1996; 46 (9): 632-637.
15. Whitby D, Howard MR, Tenant Flowers M, et al. Detection of kaposi sarcoma associated herpesvirus in peripheral blood of HIV-infected individuals and progression to kaposi's sarcoma. Lancet.1995; 23, 346(8978): 799-802.
16. Said JW, Chien K, Tasaka T, et al. Ultrastructural characterization of human herpesvirus 8 (Kaposi's sarcoma-associated herpesvirus) in kaposi sarcoma lesions: electron microscopy permits distinction from cytomegalovirus (CMV). J-Pathol. 1997; 182(3): 273-281.
17. Brooks LA, Wilson AJ, Crook T. Kaposi's sarcoma-associated herpesvirus (KSHV) / human herpesvirus 8(HHV8) ? a new human tumour virus. J-Pathol. 1997; 182(3):262-265.
18. Dictor-M. Human herpesvirus 8 and kaposi's sarcoma. Semin-Cutan-Med-Surg. 1997; 16(3): 181-187.
19. Gillison ML, Ambinder RF. Human herpesvirus 8. Curr-Opin-Oncol. 1997; 9(5):440-449.
20. Engelbrecht S, Treurnicht FK, Schncider JW, et al. Detection of human herpes virus 8 DNA and sequence polymorphism in classical, epidemic, and iatrogenic Kaposi's sarcoma in South Africa. J-Med-Virol.1997; 52(2): 168-172.
21. Kemeny L, Gyulani R, Kiss M, et al. Kaposi sarcoma-associated herpes virus; human herpesvirus 8. Orv-Hettil. 1997; 20, 138(16): 979-983.
22. Blackbourn DJ, Ambroziak J, Lennette E, et al. Infectious human herpesvirus 8 in a healthy North American blood donor. Lancet. 1997; 349 (9052): 609-611.
23. Kennedy MM, Lucas SB, Russel-Jones R, et al. HHV 8 and female Kaposi's sarcoma. J-Pathol. 1997; 183 (4): 447-452.
24. Humphrey RW, Davis DA, Newcomb FM, et al. Human herpesvirus 8 (HHV 8) in the pathogenesis of Kaposi's sarcoma and other diseases. Leu-Lymphoma. 1999; 28 (3-4): 255-264.
25. Offermann MK. Consideration of host-viral interaction in the pathogenesis of Kaposi's sarcoma. J-Acquir-Immune-Defic-Synd. 1999; 21, 1: 558-565.
26. Neipel F, Fleckenstein B. The role of HHV-8 in Kaposi's sarcoma. Semin-Cancer-Biol.1999; 9(3): 151-164.
27. Cesarman E, Knowles DM. Kaposi's sarcoma-associated herpes virus: a lymphotropic human herpesvirus associated with Kaposi's sarcomas primary effusion lymphoma, and multicentric Castleman's disease. Semin-Diagn-Pathol. 1997; 14 (1): 54-66.
28. Galetskii SA, Kadyrova EL, Kozyrev-Iu L, et al. PCR diagnosis of sequences of a novel human herpes virus type 8 in patients with Kaposi sarcoma in Russia. Vopr-virusol. 2000; 45(4): 13-17.
29. Yamada Y, Funasaka Y, Nishioka E, et al. A case of classic Kaposi sarcoma in a Japanese man: detection of human herpes virus 8(HHV-8) infection by means of polymerase chain reaction and immunofluor-escence assay. J-Dermatol. 2000; 27(6): 391-396.
30. Brenner B, Rakowsky E, Katz A, et al. Tailoring treatment for classical Kaposi's sarcoma: Comprehensive clinical guidelines. Int-J-Oncol. 1999; 14(6): 1097-1102.
31. Touloumi G, Hatzakis A, Potouridou I, et al. The role of immuno-suppression and immune-activation in classic Kaposi's sarcoma. Int-J-Cancer. 1999; 9, 82(6): 817-821.
32. Mitsuyasu, ?R ?T. Update on the pathogenesis and treatment of Kaposi sarcoma. CurrOpin-Oncol. 2000; 12(2): 174-180.
33. Almuneef M, Nimjee S, Khoshnood K, et al. Prevalence of antibodies to human herpesvirus 8(HHV 8) in Saudi Arabian patients with and without renal failure. Transplantation. 2001; 27,7 1(8): 1120-1124.
34. Herr H, Kim JU, Kang G-H, et al. Kaposi's sarcoma occurring during short-term dialysis: report of two cases. J-Korean-Med-Sci. 2001; 16(1): 130-134.

RISK MARKERS OF PRE-ECLAMPSIA

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ABSTRACT

The aim of the present study is to identify risk factors for the development of pre-eclampsia in our locality. To achieve the above aim, a total of 354 pregnant women were enrolled in a case-control study design to compare 177 pre-eclampsia patients with 177 controls selected from the two major maternity hospitals in Mosul. For risk factors, the first analysis treats each risk factor alone through computation of the odds ratio, 95% CI and P-values. Adjustment of the results with backward logistic regression model reveals several significant predictors. Risk factors found by the present study should be taken into consideration during antenatal care.

INTRODUCTION

World Health Organization (WHO) and International Society for the Study of Hypertension in Pregnancy (ISSHP) currently define pre-eclampsia (PE) as the occurrence of hypertension in combination with proteinuria developing after 20 weeks of gestation in previously normotensive non-proteinuric patient[1,2,3]. The incidence of PE worldwide is reported to be 5% although remarkable variations are reported depending on the exact definition used and the population studied[4,5]. Hypertensive disorders of pregnancy (HDP) are one of the principal causes of maternal deaths. A two community based studies carried out in Mosul concluded that HDP were at the top of the list of high risk pregnancies with an estimated proportions of 28.3% and 29.3%, respectively[6,7]. Al-Jawadi[8] in her work reinforced the above findings by reporting similar results with PE being the 2nd most frequent complication during pregnancy after anaemia with an incidence of 115 per 1000. The aim of the present study is to determine and delineate the local risk factors responsible for this disorder, and to indicate the strength of association of each of the established factor with the development of PE.

MATERIALS AND METHODS

In Mosul City, the centre of Ninevah Governorate, there are two main maternity hospitals that drain patients from urban and rural areas. Each of these hospitals acts as a regional referral hospital. To achieve the aim of the present study, a hospital based case-control design was adopted[9]. Cases were those pregnant women who fulfill the case definition of PE and have been collected from the two hospitals by non-randomized consequetive technique. Cases were interviewed at the time of labour or shortly before operation for those who delivered by caesarean section (CS). For cases of eclampsia, information was obtained from their close relatives or from patients themselves after recovery from the urgent stage. The sample of controls was selected according to the method of paired sampling with individual matching. So, for each case of PE, one control was selected according to the following criteria: No history of hypertension before and throughout the current pregnancy, non–proteinuric (by general urine examination), admitted next to the case and to the same hospital, had normal delivery and interviewed at time of labour. Data collection was conducted during a six months period (1st of July 2001–31st of December 2001).

Statistical analysis was carried out using SPSS packages Version 10. Odds ratio (OR) and its 95% confidence interval (CI) was computed. Chi-squared (χ^2) test for contingency table was used to find the statistical association[10]. Backward logistic regression analysis was done aiming to identify risk factors (independent variables) that may predict the occurrence of PE (dependent variable) in a pregnant lady[11].

RESULTS

During six months period spent for data collection, a total sample of 354 pregnant women were collected (177 women for each sample of PE cases and controls).

Background of the study population is shown in (Table-1). The majority of women were housewives with no significant risk difference

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