OPPORTUNISTIC INFECTIONS IN AIDS (AUTOPSY OF TWO CASES)

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Introduction

Since the first case of Acquired Immuno Deficiency Syndrome (AIDS) was reported in India in 1985 [1,2], there has been a rapid upsurge in the incidence of AIDS cases. Till December 1994, 885 cases of AIDS have been reported [2]. To begin with clinicians were unaware of the complexity of the AIDS. Autopsy findings at different centres all over the world have thrown light on opportunistic infections and other pathological lesions associated with AIDS [3].

The first two fatalities due to AIDS in the Indian Armed Forces occurred in May and June 1992. Both these patients were first diagnosed to be suffering from tuberculosis. Initial response to antitubercular regime was good but within 6-8 months both these patients died of disseminated cryptococcal infection. Autopsy findings in these two fatal cases of AIDS are presented.

CASE 1

A 49-year-old male, while being treated for fissure-in-ano, also complained of chronic cough, fever, loss of appetite and weight loss for one month. He denied any past history of tuberculosis and any extra-martial sexual exposure. He was initially treated with broad spectrum antibiotics. Four weeks after hospitalisation he developed herpes zoster. He was also found to have infiltrative lesions with break down in the upper and middle zones of lung fields. During the course of illness his sputum was also found positive for *M. tuberculosis*. He was put on antitubercular treatment (ATT). He showed good response but six weeks later he became acutely ill with high grade fever, bilateral pulmonary infiltrates, and right

sided pleural effusion and also behavioural changes. Investigation revealed HIV positive by ELISA and Western Blot. His T4 : T8 ratio was very low (0.9). He was diagnosed a case of AIDS. His condition kept on deteriorating and he died 8 months after initial hospitalisation.

The autopsy examination revealed severe icterus. Both pleurae were densely adherent to the chest wall with loculated turbid fluid in right pleural cavity. The lungs were markedly destroyed and petrified. The cut surfaces showed patchy areas of consolidation with tiny cavities all over. The hilar nodes were enlarged. Other organs such as liver, spleen, kidneys and adrenals were moderately enlarged. The brain was oedematous with congested leptomeninges.

The histopathological examination showed significant depletion of lymphocytes from lymphnodes (Fig 1) and the spleen showed cystic areas containing refractile round bodies. These cystic areas and refractile bodies could also be seen in kidneys (Fig 2), liver, adrenals, leptomeninges and myocardium. Special stains, mucicarmine and methylene blue, confirmed these to be cryptococci. No granulomas or acid-fast bacilli could be demonstrated in any organ or tissue.

CASE 2

A 30-year-old male presented with fever and weight loss of 3 months duration. On examination, he had hepato-splenomegaly and anaemia. The bone marrow aspirate showed *Leishmania donovani* bodies. Hence he was diagnosed and treated as a case of kala-azar. He was also transfused 7 units of blood during a stormy course of illness. Four weeks after hospitalisation he was found to have cervical lymphadenopathy and non-homogeneous opacities in the upper zone of the left lung. The lymphnode biopsy exhibited multiple granulomas with minimal caseation. No acid fast bacilli were seen on special staining. He also had pericardial effusion. He was treated for disseminated tuberculosis with ATT. He responded well during next 6 months. He had sudden deterioration with high grade fever with chills and rigor. He was also found to be positive for HIV by ELISA and

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Fig. 1 : Section of lymph node showing lymphocyte depletion with *C. neoformans* (arrow) (H&E x 150).

Fig. 2 : Section of kidney showing cystic changes in glomerulus with *C. neoformans* (PAS x 500).

Western blot. The T4 : T8 ratio was also low (1.2). He deteriorated rapidly and died due to multisystem failure.

The deceased was subjected to necropsy which revealed icterus, polyserositis, generalised lymphadenopathy and hepatosplenomegaly. The chest showed bilateral pleural adhesions with pleural effusion. The lungs were congested with areas of consolidation. The brain was oedematous. Histopathological examination in this case also showed marked depletion of lymphocytes from lymphnodes and spleen. Cryptococci were demonstrated in all organs and tissues with minimal cellular reaction. A hilar lymphnode revealed small caseating granuloma. Acid fast bacilli were conspicuous by their absence from all organs and tissues.

Discussion

Opportunistic infections in AIDS characteristically involve many organs and tissues. The infections endemic to a particular geographical region are commonly seen as fulminant opportunistic infections in immunocompromised hosts. Hence tuberculosis becomes the most common infection associated with HIV/AIDS in developing countries like ours. Many studies have proved that tuberculosis precedes manifestations of AIDS by months as *M. tuberculosis* is more virulent than other HIV-associated opportunistic pathogens [4]. Tuberculosis also shows an aggressive tendency to dissemination and is generally responsive to appropriate therapy [5].

Both our cases presented with definitive diagnosis of tuberculosis and showed a good response to ATT. They had features of full-blown AIDS 6-8 months later and on autopsy

M. tuberculosis could not be demonstrated from any organ or tissue, thus establishing the fact that the pathogens responded to therapy. Therefore a treating physician should have a high index of suspicion of tuberculosis in all cases of HIV/AIDS in our country. Cryptococcosis generally begins with primary pulmonary invasion and may spread haematogenously in severely immunocompromised hosts with a very low CD 4 count. In disseminated acute lesions, meninges, kidneys, GIT and other tissues may show presence of yeast with no cellular reaction, with or without fibrinous exudate [5]. These yeasts were very well demonstrated in both the cases as round refractile bodies, variable in size and without any hyphae. The cellular reaction was very minimal. *Cryptococcus neoformans* may also infect an apparently healthy immunocompetent host but the infection is more fulminant and frequent in patients with AIDS or other conditions associated with immunosuppression. Approximately 7% patients with AIDS present with cryptococcosis as complicating infection and it is the fourth most common cause of life threatening infection in AIDS [6].

It is estimated that by 2000 AD, approximately 400,000 HIV infections will have occurred in our country and HIV-associated tuberculosis may assume epidemic proportions. There has been a suggestion that chemoprophylaxis against tuberculosis in all HIV infected individuals may help prevent this opportunistic infection [7]. Association

of 2 or more pathogens may lead on to an uncontrollable dissemination and grave complications as seen in our cases.

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