CYTOLOGICAL DIAGNOSIS OF PRIMARY LYMPH NODE INFARCTION IN A HIV INFECTED INDIVIDUAL: A RARE FINDING

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ABSTRACT

Lymph node infarction refers to a syndrome of spontaneous coagulative necrosis. One case of lymph node infarction will be encountered in every 450 lymph node biopsied lesions. It is a rare phenomenon associated with various neoplastic and nonneoplastic conditions. The non-neoplastic conditions include polyarteritis nodosa, viral infections (Parvovirus B19, Infectious mononucleosis), thrombosis, gold injections, fine needle aspiration cytology and finally may be idiopathic. The neoplastic lesions most commonly associated with infarction are malignant lymphoma and metastatic melanoma.

Case report: A 40 year HIV infected male presented with fever, cough with expectoration and generalized weakness since 10 days and a solitary right axillary swelling measuring 2x1 cm, tender and mobile on palpation. Fine needle aspiration cytology of the lymph node was performed which revealed poorly preserved morphology of polymorphous population of lymphocytes with amorphous debris and focal areas of necrosis. Secondary causes for lymph node infarction like tuberculosis and fungal infections were ruled out by specific and systemic investigations.

Conclusion: Fine needle aspiration cytology remains a very important tool for diagnosing the non-neoplastic and neoplastic lesions of lymph nodes.

INTRODUCTION

Lymph node infarction refers to a syndrome of spontaneous coagulative necrosis. It can involve the whole lymph node or partially involve it. It involves most of the nodular parenchyma sparing only a narrow capsular rim. It is a rare phenomenon reported in literature in association with various neoplastic and nonneoplastic conditions. The non-neoplastic conditions associated with infarction are namely polyarteritis nodosa, viral infections (Parvovirus B19, Infectious mononucleosis), cholesterol atheromatous embolism, thrombosis, gold injections, intestinal volvulus, postmediastinoscopy, mononeuritis multiplex, fine needle aspiration cytology and finally may be idiopathic (Rosai 2011; Johnston et al., 2003; Kojima et al., 2002; Shah and 1978; Roberts et al., 2001; Mahy and Davies 1984; Miller and Nelems 1989; Tsuibo et al., 1999; Davies and Webb 1982; Tsang and Chan 1992; Watts et al., 1980; Ren et al., 1990). Hemorrhagic infarction of hilar lymph nodes has also been reported recently in association with heart lung transplantation (Rosai 2011; Johnston et al., 2003). One case of lymph node infarction will be encountered in every 450 lymph node biopsied lesions thus making it a rare entity. The neoplastic lesions most commonly associated with infarction are malignant lymphoma and metastatic melanoma (Rosai 2011; Bhargava et al., 1989; Toriiumi et al., 1988; Maurer et al., 1986). The incidence of lymphoma in patients with infarcted lymph nodes ranges from 40-90% (Maurer et al., 1986; Cleary et al., 1982). The condition was first described in man by Daves and Stansfeld in 1972. The differential diagnosis pertaining to the present case could also be necrotizing lymphadenitis (Kikuchi's lymphadenitis), mucocutaneous lymph node syndrome, and necrotizing granulomatous inflammation. In general, the subtotal type of necrosis occurs in superficial lymph nodes (axillary and inguinal), whereas extensive necrosis more often occurs in deep lymph nodes (mesenteric) (Maurer et al., 1986; Cleary et al., 1982; Davies and Stansfeld 1972). There is no evidenced based literature about lymph node infarction due to primary HIV infection. Most of the cases are identified by histopathological examination. Cytological studies are done rarely and the findings of infarct are oftenly missed.

Case report

A 40 year HIV infected male presented with fever, cough with expectoration and generalized weakness since 10 days and a solitary right axillary swelling measuring 2x1 cm, tender and mobile on palpation. There was no generalized
lymphadenopathy. The patient was treated with antifebrile drugs and antibiotics for his lung infection. He was on regular anti-retroviral treatment and was not taking any Anti-Tubercular treatment. Hematological investigations revealed mild leukocytopenia and normocytic normochromic anemia. ESR was significantly raised. Sputum culture and ZN stain was negative for any bacilli and AFB respectively. Chest X-ray findings were normal with no foci of particular lesions.

**CYTO-PATHOLOGY**

FNAC of the Right axillary mass was performed under aseptic conditions which yielded a straw coloured thick fluid. On microscopy, dry and wet smears (Pap/Giemsa/H&E) (Fig 1-5) showed poorly preserved morphology of polymorphous population of lymphocytes (coagulative necrosis) showing cell shadows, amorphous debris and focal areas of necrosis. Very few well preserved lymphocytes were seen. No evidence of granulomas or caseating material seen. Ziehl Neelson stain for AFB was negative. PAS stain for fungal filaments was also negative.

**DISCUSSION**

Despite the challenge implicit in Holman and Self's (1938) provocative statement that infarction of lymph nodes in man 'is
a rarity, if it ever occurs', we have been unable to trace any morphological description of changes that have been ascribed to infarction in previously normal lymph nodes. However, infarction of lymphomatous or metastatic neoplasms in lymph nodes is well known (Davies and Stansfeld 1972). The condition was first described in man by Davies and Stansfeld in 1972 as a rare occurrence (Davies and Stansfeld 1972). In this case the patient being HIV infected and on Anti-retroviral therapy for past 3 years made us to believe that due to immunocompromised status of the patient due to the viral infection, it itself or an unknown resolved secondary infection may be the cause of the coagulative necrosis of the axillary lymphnode, due to compromised blood supply. The other causes of lymph node infarction are due to Non-neoplastic and Neoplastic lesions. Among the non-neoplastic lesions first being Tuberculosis, the smears are characterized by completely amorphous, granular material without identifiable cellular material suggestive of caseous necrosis along with presence of granulomas. ZN stain for AFB is positive. These features were not present in our case. Hence Tuberculosis was ruled out as the cause of node infarct. Other granulomatous lesions were also ruled out due to absence of any granuloma and any other associated features. Absence of inflammatory cells such as neutrophils and nuclear debris also ruled out any supplicative lesion. Among the neoplastic lesions the most common lesions are malignant lymphoma and metastatic tumors that cause lymph node infarct. The incidence of lymphoma in patients with infarcted lymph nodes ranges from 40-90% (Maurer et al., 1986; Cleary et al., 1982), which is characterized by presence of monotonous population of cells along with the shadow cells and enlargement of other group of lymph nodes.

The presence of ill defined shadows of polymorphous population of lymphocytes and a solitary lymph node enlargement ruled out lymphoma as the cause of lymph node infarct. Absence of any other metastatic cells also ruled out any other malignant neoplasm as the cause. Any other non-neoplastic or neoplastic lesions were ruled out by specific and systemic investigations. The inflammatory events in the perinodal connective tissue are remarkably similar, both in morphology and time course, to those seen around human myocardial infarcts (Mallois White, and Salcedo-Salgar, 1939). The heavy polymorphonuclear leucocytic infiltrate at three days largely confined to the perinodal connective tissue, in which respect it differs fundamentally from the polymorphonuclear exudate of supplicative lymphadenitis. By the third week the polymorphonuclear leucocytes are replaced by mononuclear cells, and a zone of vascular and maturing granulation tissue appears in close apposition to the periphery of the necrosis. Finally, months after infarction in those nodes that are not irreversibly damaged, there is evidence of regeneration of lymphoid tissue. This regeneration, presumably effected by means of the lymphoid cell circulation, is not complete for the stigmata of granulation tissue, fibroed necrotic blood vessels, recanalization of occluded lymph and blood vessels, and the disturbed architectural arrangement of the lymphoid tissue remain. In general subtotal type of necrosis occurs in superficial lymph nodes and extensive necrosis in deep lymph nodes. The reason for the apparent rarity of lymph node infarction may lie in the abundance of the vascular supply and in the well developed anastomoses (Davies and Stansfeld 1972). Fine needle aspiration cytology remains a very important tool for diagnosing the non-neoplastic and neoplastic lesions of lymph nodes. It gives a presumptive assumption of pathological process in the lymph node and thus aids the clinicians/surgeons to take further actions. Idiopathic lymph node infarction is an uncommon finding. The lack of evidence based literature on Human Immunodeficiency Virus being the primary cause of lymph node infarction prompted us to present this case. The presence of preserved morphology of lymphocytes suggestive of coagulative necrosis, amorphous debris and other areas of necrosis seen in the smears suggested infarction of the lymph node which is a rare finding on cytology. Thus FNAC gives a good idea to search and rule out any idiopathic cause of lymph node infarction. Global infarction of the lymph node may mask the underlying pathology. Any ill defined lesion may coexist or follow lymph node infarction, therefore the patient needs constant surveillance.

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REFERENCES


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