Toxoplasmosis Presenting as Hyper Viscosity Syndrome due to Polyclonal Gammopathy

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Received: 6 February 2012 / Accepted: 25 August 2012 / Published online: 18 September 2012
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Abstract We are presenting a rare case of toxoplasma lymphadenopathy with hyper viscosity syndrome due to polyclonal gammopathy. A 30 year old female presented with generalized lymphadenopathy. Lymph node biopsy findings suggestive of toxoplasmosis were confirmed on serology. Bone marrow aspiration showed 50 % plasma cells. On serum electrophoresis broad, diffuse band noted, indicative of polyclonal gammopathy. M band was absent. The patient was immunocompetent and presented with hyper viscosity syndrome masking the symptoms of underlying toxoplasmosis.

Keywords Toxoplasmosis · Gammopathy · Polyclonal · Hyper viscosity

Introduction

Toxoplasma gondi is an obligate intracellular protozoan that infects up to one-third of the world's population [1]. Toxoplasmosis can present with varied signs and symptoms, of which asymptomatic lymphadenopathy is the most common. Our patient 30 year old female presented with generalized lymphadenopathy since 1 month followed by altered sensorium and mental instability. Extensive clinical

work up and review of lymph node biopsy confirmed the diagnosis as toxoplasmosis with hyper viscosity syndrome due to polyclonal gammopathy. In immunocompetent patient, toxoplasmosis is self limiting. In contrast in immunocompromised patients it is life threatening infection [2]. Polyclonal gammopathy is seen in inflammatory and infectious diseases. Hyper viscosity syndrome due to polyclonal gammopathy is rare.

Clinical Summary

A 30 years old female presented with complaints of generalized lymphadenopathy and weakness, decreased appetite and weight loss since 1 month.

Clinical examination revealed small, multiple, firm, nontender, mobile lymph nodes in cervical, preauricular, occipital and axillary region. She was being treated by a private doctor. Fine needle aspiration cytology and lymph node biopsy performed outside was reported as reactive lymphadenitis? Hodgkin's lymphoma. Patient was referred to our hospital.

Following investigations were done.

Table 1 shows the values of Investigation done.

USG abdomen showed hepatomegaly, bilateral raised renal cortical echogenicity and minimal free fluid in abdomen.

Urine examination—albumin +++, sugar ++, pus cell 2–3 cells/hpf. Urine protein 2 g/day. Random blood sugar—98 mg%. HIV negative.

Provisional diagnosis was reactive lymphadenitis with anemia due to chronic inflammation. Patient received iron and vitamin supplementation.

One week after admission patient's general condition worsened. Peripheral blood smear (PBS) revealed

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Table 1 Laboratory values of investigation done by the patient

Investigation	Report	Investigation	Report
Creatinine	2.3 mg/dl	Hemoglobin	3.9 g/dl
Na	142 mEq/l	TLC	8,800/cmm
K	3.9 mEq/l	Platelets	323,000/cmm
Uric acid	3.7 mg/dl	MCV	67 fl
Bilirubin	1 mg/dl	Ferritin	180 ng/ml ($n=30-150$ ng/ml)
SGOT	34 IU/l	HIV tri dot	Non reactive
SGPT	32 IU/l	HbsAg	Negative
Alkaline phosphatase	448 IU/l	ESR	76 mm at the end of 1 h
Sr protein	7.8 g/l	Sputum AFB	Negative
Albumin	3 g/l	PBS	Microcytic hypochromic with anisopoikilocytosis
Globulin	4.8 g/l	Serum viscosity	4.9 U

Fig. 1 Photomicrograph of bone marrow aspiration shows increased plasma cells. (Leishman's stain— $\times 100$)

microcytic hypochromic anemia with rouleaux formation. Bone marrow showed 50 % plasma cells which was the predominant series. Binucleate, trinucleate and occasional plasma blast were noted (Fig. 1).

Considering bone marrow finding Serum electrophoresis was carried out which showed diffuse broad band in gamma region, M band was absent. Serum calcium—9.8 mg/dl. Urine B-J protein—negative. No lytic lesion seen on X-ray skull.

During hospital stay patient had 3 episodes of convulsion with sudden onset of altered sensorium, thought to be due to hyper viscosity syndrome.

Antiepileptic drugs, injection dexamethasone and intravenous fluids were given and the patient remained stable. MRI brain and CSF study were done in view of above symptom. MRI showed inflammatory etiology. CSF showed lymphocytes, proteins 30 g% and sugar 35 g%.

Lymph node biopsy was reviewed; biopsy was fragmented and showed lymphoid follicles with prominent

Fig. 2 Photomicrograph of lymph node biopsy shows epithelioid cell clusters, plasma cells and monocytoid cells. (H and E— $\times 10$) **Fig. 3** Photomicrograph of lymph node biopsy shows epithelioid cell clusters, plasma cells and toxoplasma cyst (*inset*). (H and E— $\times 40$)

germinal centers. Sub capsular sinus and para follicular area showed epithelioid cell clusters, monocytoid B cell hyperplasia, plasma cell infiltration and occasional?

Toxoplasma cyst. Findings were suggestive of toxoplasma lymphadenitis (Figs. 2, 3).

For confirmation serum toxoplasma antibody was done which showed toxoplasma IgG positive in high titer, toxoplasma Ig M negative.

Patient was put on T. Cotrimoxazole for 14 days, intra venous fluids and anti epileptic drugs. Iron and vitamin supplements were given to the patient. After 14 days of antitoxoplasma treatment bone marrow aspiration was repeated that showed decreased plasma cell percentage to 15 %. After treatment patient's condition improved and renal parameters reversed to normal.

Discussion

Infection of human with *T. gondii* is common worldwide, with the prevalence varying according to environment, eating habits and age [3]. Clinical presentation of *T. gondii* infection depends on the age and immune status of patient. In the majority of immunocompetent patients it a self limiting illness, manifest most typically by isolated cervical or occipital lymphadenopathy lasting for less than four to 6 weeks. The lymph nodes are usually discreet, non-tender, and do not suppurate. Patient may present with malaise, fever, fatigue, rash, confusion and signs and symptoms of meningoencephalitis. Very infrequently immunocompetent hosts might also suffer from myocarditis, polymyositis, pneumonitis, hepatitis or encephalitis. After the acute phase, almost all patients will remain chronically infected with tissue cyst that are dormant and patient may be asymptomatic [2].

In contrast, toxoplasmosis in immunocompromised patients can be a life threatening infection. In this population, toxoplasmosis almost always occurs as a result of reactivation of chronic disease and 50 % with central nervous system involvement [2].

Our patient was immunocompetent and presented with hyper viscosity syndrome due to polyclonal gammopathy. Normal serum viscosity ranges from 1.4 to 1.8 U; our patient's serum viscosity was 4.9 units. Hyper viscosity syndrome is most commonly associated with plasma cell dyscrasias and is due to large size of immunoglobulin M [4, 5]. Hyper viscosity syndrome due to polyclonal gammopathy is rare, it is documented in SLE [6]. Our patient presented with altered sensorium attributed to hyper

viscosity. Hyper viscosity was secondary to reactive plasmacytosis leading to polyclonal gammopathy.

Polyclonal gammopathy is characterized by hyper gamma globulinemia seen as broad based peak in the beta and/or gamma region and is associated with chronic infections (tuberculosis, syphilis, chronic fungal infection, toxoplasmosis) and chronic inflammatory diseases affecting liver and collagen (e.g. primary biliary cirrhosis, chronic active hepatitis, systemic lupus erythematosus) [6]. The excess of immunoglobulin may be resolved through the adequate treatment of underlying disease responsible for polyclonal gammopathy. The patient improved after antitoxoplasma treatment. Repeat bone marrow showed reduced plasma cell percentage and decreased gamma globulins.

The present case highlights careful morphological evaluation of lymph node biopsy and correlation of bone marrow aspiration findings. Plasma cell response in lymph node with monocytoid B cell hyperplasia can be mistaken for Hodgkin's lymphoma in fine needle aspirate by inexperienced pathologist. Abnormal renal parameters may be due to acute tubulointerstitial injury (? Hyper viscosity related). Clinical features of toxoplasma meningoencephalitis may overlap with hyper viscosity syndrome.

Our case is presented on account of its unusual presentation as hyper viscosity due to polyclonal gammopathy masking the symptoms of toxoplasmosis. This case represents first case presenting with hyper viscosity mimicking plasma cell dyscrasia with overlapping features of toxoplasma meningoencephalitis.

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