Journal of Advances in Medicine

Citation: JAM: 5(2): 43-45, December 2016 DOI: 10.5958/2319-4324.2016.00010.9

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CASE REPORT

Abdominal Lymphadenopathy - Tuberculosis mimicking classy clinicoradiological features of Hodgkin's Disease

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Keywords: Lymphadenopathy, Hodgkin's, periportal

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ABSTRACT

bdominal lymphadenopathy is regularly encountered clinical entity and planning a management on likely diagnosis may prove disastrous. In the present case, clinical features with CT findings of massive diffuse paraaortic, mesenteric and splenic group of lymph nodes, mass at the head of the pancreas, encasement of portal vein, gross splenomegaly and ascites added with suggestion from bone marrow biopsy almost clinched the diagnosis in favour of Hodgkin's disease. Biopsy, however turned the table topsy turvy by confirming these lymph nodes to be of tuberculous origin. Histopathology, thus remains a must tool of information to find a correct diagnosis as observed in this case.

Though the pulmonary involvement is maximally observed tuberculous infection in this country, abdomen is the most commonly affected anatomic area in patients with extrapulmonary tuberculosis (1,2,3). Abdominal tuberculosis can be asymptomatic or cause non-specific symptoms such as weight loss, abdominal pain, fever, abdominal distension, vomiting, diarrhoea, and anorexia (4) Lymph nodes are affected more frequently than other anatomical sites, such as solid viscera and gastrointestinal tract. Periportal, peripancreatic,

mesenteric, omental and upper para-aortic nodes are frequently affected⁽⁵⁾. Abdominal tuberculosis can be asymptomatic or cause non-specific symptoms such as weight loss, abdominal pain, fever, abdominal distension, vomiting, diarrhoea, and anorexia⁽⁶⁾. Abdominal tuberculosis can mimic a variety of other abdominal conditions/diseases and only a high degree of suspicion can help in the diagnosis

otherwise it is likely to be missed or delayed resulting in high morbidity and mortality. (7,8.9.10)

Case Report

A 14-year-old child presented in our emergency department with complaints of fever mild to moderate with generalized swelling of body. Lymph node enlargement was observed in left side of the neck, bilaterally in inguinal area and axillae. There was no history of any systemic illness in the past.

On arrival, his oral temperature was 38.6°C, pulse rate 110/min, respiratory rate 28/min, and blood pressure 126/70 mm Hg in right arm supine position.

General examination revealed mild-to-moderate anemia, bilateral pedal edema and generalized lymphadenopathy which included bilateral submandibular, submental and left posterior cervical lymph nodes along with bilateral inguinal

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region and axillae. Lymph nodes were non tender, firm on consistency, 1 to 2 cm in size and non mobile. On Abdominal examination, mild hepatomegaly, moderate splenomegaly and tense ascites were present. Rest of the systems were within normal limits.

Laboratory values revealed Hb 6.7 g/dL, white blood cell count of 5.6 × 109/L with neutrophils 46%, lymphocytes 40% platelet 66 × 109/L, total serum bilirubin 0.13 mg/dL, serum glutamic oxaloacetate transminase 20.4 IU/L, serum glutamic pyruvate transminase 8.7 IU/L, random blood glucose 82 mg/dL, albumin 1.07g/dL, protein 3.0g/d, prothrombin time 14 sec with INR 1.4. Ultrasound abdomen showed mild hepatomegaly with moderate splenomegaly with bulky pancreatic head with moderate peritoneal collection. Multiple retroperitoneal and mesenteric group of lymph nodes were present. Portal vein diameter was normal.

Ascitic fluid examination showed total cell count of 124 cells with neutrophils 22%, lymphocytes 70% with 8% mesothelial cells, protein 2.2, SAAG- 0.8, ADA- 36 IU/L. Ascitic fluid culture was sterile and negative for malignant cells.

CECT Scan Abdomen showed periportal lymph nodes causing encasement of portal vein along with enlarged necrotic head of pancreas forming a mass. Mesentric, upper paraaortic and splenic lymph nodes were enlarged. Lymph nodes showed necrosis with no enhancement. Mild hepatomegaly with moderate splenomegaly present. Moderate peritoneal collection was present. Findings were suggestive of either infective (most likely tuberculosis) or of neoplastic etiology. (Fig. 1)

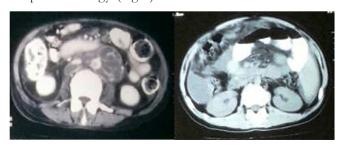


Fig. 1: Showing Mesentric, upper paraaortic and splenic lymph nodes were enlarged. Lymph nodes showed necrosis with no enhancement.

Bone Marrow Aspiration revealed non-specific lymphoid proliferation and was reported to be inconclusive. Bone marrow Biopsy revealed atypical lymphoid cells of undetermined significance. Findings were reported to be suggestive of Hodgkin's Disease.

Histopathological examination of biopsy specimen from left posterior cervical lymph node revealed hypertrophied stratified squamous epithelium with subepithelial zone consisting dense chronic inflammatory infiltrate and epithelioid cells in fibrocollagenous stroma. Ziehl–Nielsen staining was positive for acid-fast bacilli characterizing it as granulomatous lesion.

In the mean time report for immunohistochemistry for CD 15 and 45 came out to be negative indicating Hodgkin's disease unlikely.

Patient was kept on Antitubercular therapy and was followed with an interval of 15 to 20 days. Patient became afebrile, have shown improvement in general well-being and weight gain.

DISCUSSIONS

Abdominal lymphadenopathy often poses clinical challenge in recognizing the diagnosis. Lymph nodes could be localized or generalized. Distribution of lymph nodes in abdominal cavity may sometimes point towards a possibility but usually the diagnosis could only be established with use of invasive procedures including FNAC and biopsy.

Presence of diffusely distributed lymph nodes in most area of abdomen is a similar situation which requires invasive approach to establish the diagnosis. Suggestion of a likely diagnosis by other investigations may lead to wrong management as could have happened in the case in consideration.

This case has presented with long term fever, weight loss, hepatosplenomegaly and ascitis.

CT findings here have revealed periportal lymph nodes causing encasement of portal vein along with enlarged necrotic head of pancreas forming a mass. Mesentric, upper paraortic and splenic lymph nodes are enlarged. Lymph nodes showed necrosis with no enhancement. Mild hepatomegaly with gross splenomegaly present. Moderate peritoneal collection was present.

This CT picture advocated more for Hodgkin's disease as is suggested by features like diffuse massive lymphadenopathy, encasement of intraabdominal structures, splenic lymph nodes and gross splenomegaly. In the mean time, Bone Marrow aspiration findings also revealed non-specific lymphoid hyperplasia presenting with atypical lymphocytes. Report concluded these findings highly suggestive of Hodgkin's disease. But to our surprise, concomitantly conducted immunochemistry markers for Hodgkin's

disease reported negative putting a question mark on almost established diagnosis suggested by clinical features, CT findings and bone marrow aspiration. Biopsy was performed from one significant enlarged lymph node from posterior cervical group of lymph nodes. Histopathology revealed etiology as tubercular with description of distinct granulomatous lesion. The patient was kept on ATT following the confirmation.

This case was interesting as it suprised with changes occurring with progress made towards achieving a diagnosis. Abdominal lymphadenopathy as is observed in scores of cases, leads to highly variegated patterns of abdominal lymph nodes. Intraabdominal sites, appearance, relation to abdominal viscera and involvement of intraabdominal structures can be quite overlapping and may mimic the patterns of different diseases simultaneously. Such presentations as in case in consideration could be regularly encountered and histopathological evaluation to confirm a diagnosis is must for consequent management.

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