

# An Obscure Cause of Leg Edema, Non-Hodgkin's Lymphoma

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## ABSTRACT

A 25-year-old female having asymptomatic edema of unilateral leg was provisionally diagnosed as a case of psoas abscess. Later, she turned out to be a case of non-Hodgkin's lymphoma. This presentation is contrary to usual age and mode of presentation of non-Hodgkin's lymphoma.

**Key words:** Unilateral leg edema. Psoas abscess. Non-Hodgkin's lymphoma.

## INTRODUCTION

Lymphomas are 6<sup>th</sup> most common cause of cancer in United States, with an incidence of approximately 60,000 cases per year. About 85% of patients with malignant lymphoma have non-Hodgkin's lymphoma, low grade or high grade. The incidence of Non-Hodgkin's Lymphoma (NHL) is increasing, particularly, among elderly and patients with AIDS.<sup>1</sup> In recent years, incidence of NHL is also found to be increasing in Pakistan.<sup>2</sup> Diffuse B-cell lymphoma is the most common non-Hodgkin's lymphoma.<sup>3</sup>

Mean age of presentation is 60 years with slight male predominance. Diffuse large cell lymphoma usually presents with rapidly enlarging symptomatic mass at a single nodal or extra-nodal site, B-symptoms, involvement of GIT, skin, bone or brain.<sup>4</sup>

Here, we report a case of 25 years old female, who had NHL. She presented with exclusively gross and progressively increasing edema of unilateral lower limb without generalized lymphadenopathy, B-symptoms, skin rash or other relatively pertinent symptoms. Diagnosis of NHL in this case was incidental when enlarged lymph nodes were found during surgical decompression of initially suspected psoas abscess, which, on histopathological examination, revealed malignant NHL (diffuse large cell type).

Age of presentation and mode of presentation (mimicking psoas abscess), in the absence of lymphadenopathy, are both rare for NHL.<sup>5,6</sup>

## CASE REPORT

A 25-year-old female, resident of Okara, was admitted in the Military Hospital, Rawalpindi, with 2 months history

of progressive swelling of left lower limb. She had diffuse ache in left lower limb, noticed swelling of left foot, which gradually progressed, proximally involving calf, thigh and groin. The color of the limb was shining white. It appeared grossly swollen as compared to right lower limb. There was no history of shortness of breath, palpitation, cough headache, seizures, weight loss, fever, night sweats, skin rash, intermittent claudication, Raynaud's phenomenon and trauma. She was married 4 years back and had a girl of one year. She had no menstrual irregularity.

Physical examination revealed a young lady of thin built, fair looking, slightly in pain, having grossly swollen tender white edematous left leg, overlying skin was shiny simulating as 'phlegmasia alba dolens'. Left leg had pitting edema (Figure 1). No lymph nodes were palpable in the neck, axilla and right groin. Left flank and loin appeared full with a doubtful ill-defined mass. There were no visible veins. Femoral, popliteal and dorsalis pedis pulses were not palpable because of gross edema.

Abdomen was soft with marked tenderness in left iliac fossa but there was no rebound tenderness and visceromegaly. Chest was clinically clear.

On blood analysis, hemoglobin was 10.7 g/dl. TLC (total leucocyte count was  $10.9 \times 10^3/\text{mm}^3$ , neutrophils 80%, lymphocytes 14%, eosinophils 1%, monocytes 1%). Serum albumin and electrolytes were within reference range. Liver function tests, renal function tests and urine analysis revealed normal values. HCV and HBV status was negative.

On ultrasonography, an oblong mass with thick hyper-echoic shadows was seen along left anterolateral abdominal wall. Straw coloured pleural effusion was aspirated from left side under ultrasound guidance. There was no visceromegaly, detectable para-aortic or mesenteric lymph nodes and ascites. Doppler ultrasound ruled out venous thrombosis. Pleural aspirate was yellowish in colour without visible blood, specific gravity 1.030, proteins 4 g/dl, cell count  $5000/\text{mm}^3$ , predominantly lymphocytes with many abnormal

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cells having hyperchromatic nuclei and high nuclear to cytoplasmic ratio, suggesting lymphocytic exudates.

CT scan showed bilateral pleural effusion, ascites, fluid collection along left psoas muscle extending down to femoral sheath and effusion of left hip joint. Grossly compressed left iliac and femoral vessels were seen. The external pressure appeared to be due to some necrotic mass but no clear lymph node enlargement was seen.

A provisional diagnosis of left psoas abscess with external pressure on iliac and femoral vessels was made. Patient was counseled regarding underlying pathology. Initially anti-coagulants were started which were stopped after CT scan was done.

As the swelling of the leg was increasing with agonizing pain, impending ischemia of leg was feared so emergency decompression of vessels and drainage of psoas abscess was planned. An oblique left inguinal incision was made. Peritoneal cavity was opened and 700 ml of serosanguineous fluid was drained. All the tissue planes were grossly edematous, oozing straw coloured fluid. Large juicy, pulpy lymph glands were seen along iliac vessels, which were removed (Figure 2). A second incision was made below left inguinal ligament. Enlarged lymph nodes along femoral vessels were removed for biopsy and decompression.

On histopathology, the large abdominal mass of lymph nodes turned out to be due to malignant NHL (large cell type). The inguinal lymph node showed complete effacement of nodal architecture by malignant tumour, comprising of diffused sheets of neoplastic cells having large pleomorphic vesicular nuclei with prominent nucleoli, suggesting the diagnosis of diffuse large cell non-Hodgkin's lymphoma.

Postoperative progress was fair with reduction of pain and swelling. Case was referred to oncology department and chemotherapy was started. On 4 months follow-up, she showed smooth recovery on therapy.



Figure 1: Gross swelling of unilateral leg.

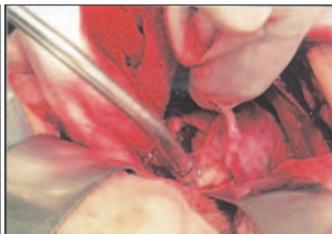


Figure 2: Lymphadenopathy along left internal iliac vessels.

## DISCUSSION

The etiology of leg edema is multifactorial, revolving around the intricate balance of capillary blood and oncotic pressures, tissue pressures, capillary permeability, and lymphatic flow. Changes in any of these factors can offset the extravascular fluid balance and result in edema formation.

The development of lymphedema is a slow, insidious process beginning in the most distal portion of the extremity. Ninety-five percent of unilateral lymphedema involves obstruction of lymphatics due to infection, post-operative lymphatic disruption and malignancy. Carcinoma of prostate in males and lymphomas and pelvic tumours in females are commonly responsible. Presentation of lymphoma, as leg edema, is uncommon, especially in the absence of generalized lymphadenopathy.

Diffuse large cell lymphomas are most common types of NHL and account for 60-70% aggressive lymphoid neoplasm and 85% of these originate in B-cells. The mean age of presentation is 65-70 years of age and patients present with rapid symptomatic lymphadenopathy. B-symptoms (fever, night sweats, weight loss > 10% of normal body weight), skin rash and symptoms pertinent to extra-nodal involvement.<sup>6</sup>

Excisional biopsy is the standard diagnostic procedure. Prognosis and approaches to treatment are influenced by histological subtypes and grade of the tumour. Laboratory studies should include complete blood count and serum chemistry. Radiological examination including screening chest radiograph, CT scan of chest, abdomen and pelvis is usually included in the work-up.<sup>7</sup> In patients with CNS involvement, spinal fluid examination is indicated and bone marrow biopsy is standard procedure. Cell surface markers also help in this regard.

The most successful treatment has been based upon combination therapy including chemotherapy, irradiation and biological therapy.<sup>8-10</sup> Localized aggressive lymphomas are usually treated with 4 cycles of CHOP (cyclophosphamide, doxorubicin, oncovin, and prednisolone) combination chemotherapy followed by involved field radiation therapy.

Response depends upon tumour bulk, dose of chemotherapy and physiological reserves of patient. This chemo-irradiation is associated with high risk of permanent sterility, left ventricular dysfunction and hematological injury leading to myelodysplastic syndrome and tumour lysis syndrome.

Conventional chemotherapy regimens cure fewer in more than 40% of patients with aggressive NHL and do not cure patients with indolent lymphoma. Patients with persistent or recurrent NHL of any histological subtypes can not be cured with standard therapies, although, 15-50% of such patients can achieve long-term disease free survival when treated with high dose chemotherapy and autologous stem cell transplantation. The newer modalities being evaluated for NHL seem promising and include monoclonal antibodies therapy. NHLs are ideal for these because they are not only sensitive to many of the monoclonal antibodies but are also radiosensitive e.g. Rituximab.<sup>10</sup> Biological or immuno therapy can be used to boost, direct or restore effectiveness of immune system.

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