Dear Editor,

A 63-year-old male patient with chronic renal failure and hypertension was referred to the hematology department after hypergammaglobulinemia was detected as part of the work up. Rheumatoid arthritis (RA) was part of the patient’s past medical history. Past surgical history included aortic aneurysm. Physical examination revealed small lymphadenopathies in the left inguinal region, the largest of which was 1 cm in size. Hepatosplenomegaly was not observed. Livedo reticularis was present in both legs which can occur with RA. It is caused by spasms or lack of blood flow in the blood vessels supplying skin. The rest of the physical exam was normal. In complete blood count, C-reactive protein (CRP) was 24.98 mg/L (reference range 0-5 mg/L), erythrocyte sedimentation rate as 110 mm/hr (reference range 0-15 mm/hr), hgb was 6.8 g/dL. Sedimentation and CRP are non-specific tests that indicate inflammation associated with disease severity in patients with RA but are used in diagnosis of the disease and monitoring its activity. The rest of the blood tests were normal. Computed tomography showed more lymphadenopathy less than 1 cm in size in the neck, paratracheal area, aortopulmonary area, subcarinal area and bilateral hilar levels 1, 2 and 5. Superficial ultrasonography showed lymph nodes in the left inguinal region, the largest of which was 42x15 mm in size, with a visible fatty hilus and increased cortical thickness. Bone marrow biopsy that was done six months prior to presentation revealed increased myeloid lineage cells, and plasma cells stained polyclonally with lambda and kappa.

Excisional biopsy of lymphadenopathy in the inguinal region was performed. On microscopic examination, the lymph node structure was preserved. There was no effacement in the lymph node structure as seen in lymphoma. No Reed-sternberg like large cells were seen. There were areas of reactive follicular hyperplasia (Figure 1). Increased plasma cells were observed in the medulla and interfollicular area. The lambda and kappa immunohistochemical studies showed polyclonal staining of plasma cells (Figure 2). The findings observed in the patient who was followed up due to RA were primarily compatible with rheumatoid lymphadenopathy. Since the findings were compatible with rheumatoid lymphadenopathy and CRP remained elevated, it was decided to start the patient on a drug with interleukin inhibitor effect.

RA is an autoimmune disease that may affect all systems, especially the joints.[1] Enlarged lymph nodes are not uncommon in RA. In a study involving one hundred patients, lymphadenopathy was observed in 82% of patients of which was 42x15 mm in size, with a visible fatty hilus and increased cortical thickness. Bone marrow biopsy that was done six months prior to presentation revealed increased myeloid lineage cells, and plasma cells stained polyclonally with lambda and kappa.

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and was most seen in the axillary region.\(^2\) However, lymphadenopathy can be seen anywhere in the body.\(^3-5\) Lymphadenopathy may be localized or systemic. Clinically, it may lead to suspicion of lymphoma.\(^6\) Lymph nodes are mobile and not tender. Systemic findings including fever, anemia and weight loss may be associated with rheumatoid lymphadenopathy. Splenomegaly, polyclonal hypergammaglobulinemia and cryoglobulinemia may be observed.\(^6\)

The risk of lymphoma is twice as high in patients with RA compared to the normal population.\(^7,8\) Hodgkin lymphoma is more common than non-Hodgkin lymphoma.\(^9\) Hodgkin lymphoma cases are of classical type. Most of the non-Hodgkin lymphomas occurring in RA patients are of the diffuse large B-cell lymphoma type.\(^10,11\) The use of methotrexate (MTX) in RA treatment may also lead to severe bone marrow suppression and development of lymphoproliferative diseases.\(^12-14\)

When lymphadenopathy develops in a patient with RA, lymphomas and lymphadenopathy secondary to the disease may be considered in the differential diagnosis. Multicentric Castleman disease should also be included in the differential diagnosis due to increased plasma cells. Immunohistochemical positivity with human herpes virus 8 allows an easy differentiation from rheumatoid lymphadenopathy.

**Conclusion**

In conclusion, although the risk of lymphoma increases in RA patients with or without treatment, it should not be forgotten that RA also causes lymphadenopathy.

**Ethics**

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**References**


