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Serum angiotensin converting enzyme: a possible marker in Lennert's lymphoma?

Data on raised activity of serum angiotensin converting enzyme (SACE) in patients with lymphoma are rare. High SACE activities were reported in Lennert's lymphoma,^{1,2} and we report on two cases of Lennert's lymphoma in which SACE was monitored during the course of the disease and in which SACE reflected the activity of the tumour.

A 24 year old man was admitted with sustained fever, malleolaroedema, and generalised weakness. Physical examination showed subicteric sclerae, hepatosplenomegaly, and enlarged peripheral lymph nodes; pancytopenia was noted. A computed tomogram of the abdomen showed enlarged lymph nodes around the aorta, the vena cava, and the arteriae iliacae. Bone and lymph node biopsy specimens confirmed the diagnosis of Lennert's lymphoma.

The activity of SACE was 863 U/l (normal values 115–491 U/l).³ Conventional chemotherapy was started; the patient went to partial remission after one course; SACE dropped to 287 U/l. Because of long standing leucopenia, treatment was changed to a less aggressive chemotherapeutic combination. Increased hepatosplenomegaly, fever without evidence of infection, and pancytopenia recurred. The SACE activity rose to 689 U/l, and the patient remained resistant to further chemotherapy.

A 21 year old man presented initially with renal colic, which an intravenous pyelogram showed to be due to an excluded right kidney. Because of coexisting splenomegaly, a computed tomography scan of the abdomen was performed and an extensive tumoral mass was found. The histological appearances of an abdominal lymph node and the spleen were first thought to be diagnostic of Hodgkin's disease. The patient developed a mild hypercalcaemia.

Combination chemotherapy with two courses of MOPP (mustine-ovincin-prednisone and procarbazine) and three of ABVD (adriamycin-bleomycin-vincristine-dacarbazine) was begun. After an interruption of three months an inguinal lymph node relapsed and an additional course of MOPP and ABVD was administered.

Because of anorexia, vomiting, fever, icterus, hypercalcaemia and anaemia, the patient was referred to our hospital one and a half months later. Progression of abdominal lymphoma was noted on computed tomography and ultrasonography. Liver and bone biopsy specimens showed reactional histiocytosis and a haemophagocytic syndrome in association with malignant lymphoma, mostly closely resembling a T cell type. Lennert's lymphoma was diagnosed. SACE activity was 1004 U/l. The patient received one further course of chemotherapy. After some complications, the patient died of cardiac arrest. The SACE activity was 423 U/l.

Lennert's lymphoma is a haematological malignancy characterised by the presence of a high content of epitheloid histocytes, T cells, and rare Reed-Sternberg-like cells. In a study of sarcoidosis, Gaucher's disease, and other granulomatous disorders Lieberman *et al* found that three out of 11 patients with Lennert's lymphoma had raised SACE activities.

Reported cases of histiocytic medullary reticulosis have been associated with increased SACE activity. Grönhagen-Riska *et al* assumed that the increase in SACE activity reflected the monocytic line having reached the macrophage or histiocytic stage.⁴ Dereme *et al* reported two patients with non-Hodgkin's lymphoma who showed increased SACE activities.⁵ These authors suggested a direct role for SACE in the metabolism of vitamin D.

Lennert's lymphoma is possibly associated with an increase in SACE activity due to epitheloid proliferation. This can be helpful in differentiating Lennert's lymphoma from other malignancies. Tumour activity can also be evaluated by this marker during and after treatment.

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Infiltrative myeloid metaplasia: an unusual cause of gastric outlet obstruction

Myeloid metaplasia, histologically characterised by the simultaneous presence of all three bone marrow elements, is frequently found in myeloproliferative disorders. It is usually asymptomatic but may, on rare occasions, behave as a space occupying lesion or an aggressive infiltrate. In a population of 110 patients with myelofibrosis, aggressive infiltrates of myeloid metaplasia were found in six patients.¹ Infiltrative myeloid metaplasia has been reported in kidneys, ureters, breasts, small bowel, lungs, liver and spleen. We describe the first reported case (as far as we are aware) of gastric outlet obstruction caused by infiltrative myeloid metaplasia affecting the pyloric region of the stomach.

In 1978 a 58 year old man was found to have essential thrombocythaemia (platelet count $790 \times 10^9/l$). His platelet count was successfully controlled by treatment. By 1980 he had become anaemic (haemoglobin concentration 10.9 g/dl) with a normal platelet and white cell count. His peripheral blood film showed features suggestive of myelofibrosis.

By 1982 acute myeloblastic leukaemia had supervened (peripheral white cell count $24.6 \times 10^9/l$ with 27% blasts). With chemotherapy there was some improvement in his blood picture but he now developed intractable vomiting unrelated to chemotherapy and associated with abdominal distension. A barium meal showed no gastric