campylobacter-like organism ("Campylobacter pyloridis")—clinical correlations and distribution in the normal population. *J Med Microbiol* 1986;**22**:57–62.
4 Morris JA, Gardner MJ. Calculating confidence intervals for relative risks (odds ratios) and standardised ratios and rates. *Br Med J* 1988;**296**:1313–6.
5 Smithius RHM, Vos CG. Heterotopic gastric mucosa in the duodenal bulb: relationship to peptic ulcer. *Am J Roentgenol* 1989;**152**: 59–61.
6 De Cothi GA, Newbold KM, O'Connor HJ. Campylobacter-like organisms and heterotopic gastric mucosa in Meckel's diverticula. *J Clin Pathol* 1989;**42**:132–3.

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

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] Deremee *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.

P ZACHÉE
M GEBOERS*
H NEELS†
W BEELAERTS†
S SCHARPÉ
*Departments of Internal Medicine, *Pathology, and †Clinical Chemistry, Stuivenberg Hospital, 2000 Antwerp, and the Department of Medical Biochemistry, University of Antwerp, 2610 Wilrijk, Belgium*

References
1 Lieberman J, Nosal A, Schlessner L, Sastre-Foken A. Serum angiotensin-converting enzyme for diagnosis and therapeutic evaluation of sarcoidosis. *Am Rev Resp Dis* 1979;**120**:329–35.
2 Tamura A, Hashimoto K, Tanoue M, *et al*. A case of so-called Lennert's lymphoma, suspected of sarcoidosis due to uveitis occurring first and elevation of angiotensin-converting enzyme. *Jpn J Thorac Dis* 1984; **22**:1033–9.
3 Neels H, van Sande M, Scharpé S. Sensitive colorimetric assay for angiotensin-converting enzyme in serum. *Clin Chem* 1983; **29**:1399–403.
4 Grönhagen-Riska C, Klockars M, Selroos O. Elevated angiotensin 1-converting enzyme in histiocytic medullary reticulosis. *N Engl J Med* 1983;**308**:283–4.
5 DeRemee RA, Banks PM. Non-Hodgkin's lymphoma associated with hypercalcemia and increased activity of serum angiotensin-converting enzyme. *Mayo Clin Proc* 1986; **61**:714–8.

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 prepyloric region of the stomach.

In 1978 a 58 year old man was found to have essential thrombocythaemia (platelet count 790 × 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 × 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

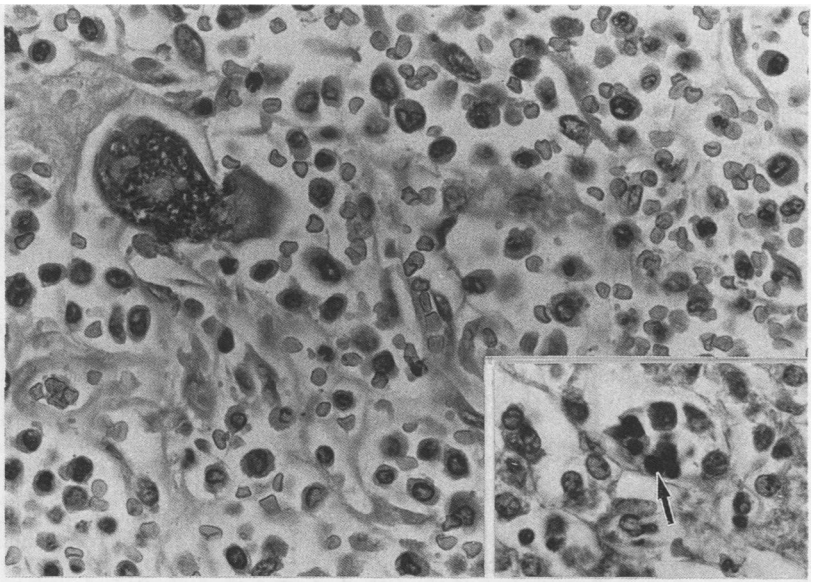

Figure *Pre-pyloric area of 58 year old man with acute myeloblastic leukaemia showing presence of bizarre megakaryocytes and erythroid nests (inset).*

abnormality. He died after a brief septicaemic illness.

Post mortem examination showed hepatosplenomegaly and generalised lymphadenopathy. The entire pre-pyloric region of the stomach was diffusely thickened, indurated, and yellow, but the pyloric lumen was not grossly narrowed.

Histological examination of the pre-pyloric area showed a heavy and diffuse polymorphous infiltrate within the submucosa and muscularis externa. The infiltrate was composed predominantly of myeloid cells at all stages of maturation, the commonest single cell type being myelocytes. There were also occasional bizarre megakaryocytes (figure) and erythroid nests (inset). The appearances were therefore those of myeloid metaplasia. The affected part of the stomach contained no distinguishable submucosal or myenteric ganglion cells. As no gross pyloric narrowing was seen at barium meal or at necropsy it is presumed that infiltration by haemopoietic cells of the pre-pyloric and pyloric smooth muscle led to a functional gastric outlet obstruction. Similar foci of myeloid metaplasia were evident in the liver, spleen, renal parenchyma and lymph nodes. Histological examination of the bone marrow showed the typical changes of myelofibrosis, but with an excess of primitive cells (10–15%). Residual leukaemia was thus clearly present in the bone marrow, but the gastric infiltrate composed of haemopoietic cells showing trilineage differentiation by definition represented myeloid metaplasia rather than a leukaemic infiltrate. The aggressive behaviour of the pre-pyloric myeloid metaplasia seen in this patient is in keeping with current views of the neoplastic nature of myeloproliferative disorders[2,3]; its apparent rarity may well be related to lack of recognition. Infiltrative myeloid metaplasia should therefore be considered as a possible cause of unexplained symptoms in patients with myeloproliferative disorders, particularly as the lesion may be radiosensitive.[1,4]

S M ISMAIL,*
Department of Histopathology, University Hospital of Wales, Heath Park, Cardiff CF4 4XW,
K MYERS,
Department of Haematology, West Wales General Hospital, Carmarthen, Dyfed

References

1 Glew RH, Haese WH, McIntyre PA. Myeloid metaplasia with myelofibrosis: the clinical spectrum of extramedullary haematopoiesis and tumour formation. *Johns Hopkins Med J* 1973;**132**:253–65.
2 Jacobson RJ, Salo A, Fialkow PJ. Agnogenic myeloid metaplasia: a clonal proliferation of hemopoietic stem cells with secondary myelofibrosis. *Blood* 1978;**51**:189–94.
3 Fialkow PJ, Faguet GB, Jacobson RJ, Vaidya K, Murphy S. Evidence that essential thrombocythaemia is a clonal disorder with origin in a multipotent stem cell. *Blood* 1981;**58**:916–9.
4 Lieberman PH, Rosvolt RV, Ley AB. Extramedullary myeloid tumours in primary myelofibrosis. *Cancer* 1965;**18**:727–36.

API-20NE and Sensititre Autoidentification systems for identifying *Pseudomonas* spp

Pseudomonas species are the most common non-enteric Gram negative rods associated with infection, and of these, *Pseudomonas aeruginosa* is most often implicated.[1,2] Many clinical laboratories often undertake the identification of these organisms as a prelude to epidemiological studies, and in previous comparative studies the API 20NE system has yielded the highest rate of correct identifications and fewer incorrect identifications than other comparable systems.[3]

The Sensititre Autoidentification system (Sensititre Ltd) is based on fluorospectrophotometry and provides a fully automated method for the identification of Enterobacteriaceae, oxidase positive fermentative rods, and non-fermentative Gram negative rods. The identification of Enterobacteriaceae by the API 20E and Sensititre systems has recently been compared.[4]

The table gives the results of the identification of 140 isolates by the two systems. Species identification was obtained with 102 (73%) isolates with both systems. The API 20NE system yielded an identification of *Ps aeruginosa* for 95 isolates: the Sensititre system identified 43 isolates as *Ps aeruginosa* and 56 isolates as *Ps putida*.

The identification of *Ps aeruginosa* by the Sensititre system was confirmed by a similar identification for most isolates by the API 20NE system. Those isolates identified as *Ps putida* by the Sensititre system were predominantly identified as *Ps aeruginosa* or

Table *Identification of 140 isolates of Pseudomonas sp with API 20NE and Sensititre Autoidentification systems*

	Number (%) of isolates yielding acceptable identification	
Organism	*API-20NE*	*Sensititre*
Species identification		
Ps aeruginosa	95 (68)	43 (32)
Ps putida	0	56 (40)
Ps fluorescens	3 (2)	0
Ps stutzeri	2	0
Ps maltophilia	2	2
Ps alcaligenes	0	1
Genus identification only:		
Pseudomonas sp	38 (27)	38 (27)