Discussion

Numerous cutaneous manifestations have been reported in association with myelomatosis and other immunoproliferative disorders. Pruritus, which is the outstanding feature of many skin diseases, is also well-recognised in systemic disorders³ and is commonly seen in association with lymphoid neoplasms, myeloproliferative disorders, and other haematological diseases. Pruritus often antedates the correct diagnosis by some time. The present cases illustrate that myelomatosis may present with generalised pruritus, an association that has apparently not been reported. Zelicovici *et al*⁴ reported three cases of generalised pruritus with a paraproteinaemia, but these patients were eventually classified as benign gammopathies. Rajka⁵ suspected myelomatosis in one patient with pruritus but did not substantiate the diagnosis. Pruritus has been reported in myelomatosis but only as a feature of renal disease and not underlying malignancy.¹ Renal hypofunction was not present in either of our patients. The pattern of response after treatment suggests a direct cause and effect relationship. Thus myelomatosis should be considered in the differential diagnosis of generalised pruritus due to internal disease.

Requests for reprints to Dr J Gordon Erskine, Department of Haematology, Victoria Infirmary, Glasgow G42 9TY.

¹ Bluefarb, S M, *Archives of Dermatology*, 1955, **72**, 505.

² Beever, D G, *British Medical Journal*, 1972, **4**, 275.

³ Rook, A, Wilkinson, D S, and Eblins, F J G, *Textbook of Dermatology*. Oxford, Blackwell Scientific Publications, 1972.

⁴ Zelicovici, *et al*, *Israeli Journal of Medical Sciences*, 1969, **5**, 1079.

⁵ Rajka, G, *Acta Dermato-Venerologica*, 1966, **46**, 190.

(Accepted 10 December 1976)

Departments of Haematology and Dermatology, Glasgow Royal Infirmary

J G ERSKINE, MB, MRCP, senior registrar in haematology
R M ROWAN, MB, MRCP, consultant haematologist
J O'D ALEXANDER, MB, FRCP, consultant dermatologist
G A SEKONI, MB, CHB, registrar in medicine

Acute splenic abscess due to *Salmonella chester*

Isolated splenic abscess presenting as an abdominal emergency is an uncommon occurrence. Moreover, in the case we report it was due to a salmonella of unusual serotype.

Case report

A 20-year-old man was admitted to hospital with a three-day history of increasing pain in the left hypochondrium and loin, worse on inspiration and associated with vomiting, constipation, and frequency of micturition. Two days earlier urinary infection had been diagnosed as his urine had been found to contain protein and blood; he had been treated with co-trimoxazole. He had no history of abdominal trauma or travel to the tropics.

He was ill and dehydrated, with a temperature of 37.7°C, pulse 112/min, and blood pressure 125/75 mm Hg. His abdomen was distended and a tender mass was palpable in the left upper quadrant; the bowel sounds were infrequent. A clinical diagnosis of intra-abdominal abscess was made. The results of investigations showed: haemoglobin 13.2 g/dl; leucocytes $12.5 \times 10^9/l$ (12 500/mm³; 90% neutrophils); ESR 105 mm in first hour; blood urea concentration 9.9 mmol/l (70 mg/100 ml); serum amylase concentration 50 Somogyi units; blood glucose concentration 6.8 mmol/l (120 mg/100 ml); blood culture sterile; x-ray films of the abdomen after intravenous urography confirmed the presence of a mass in the left hypochondrium displacing the left kidney inferiorly. Laparotomy disclosed a large splenic abscess, its wall being necrotic adjacent to the hilum, which contained over two litres of turbid fluid, which was aspirated to facilitate splenectomy. The splenic bed was drained. Postoperatively he was given ampicillin, 500 mg six-hourly for 10 days, and gentamicin, 80 mg eight-hourly for five days.

The spleen weighed 1430 g, and contained a large cyst, whose wall was composed of fibrous tissue with evidence of acute and chronic inflammation. The inflammation had destroyed the lining in many places, but the appear-

ances were those of an infected simple cyst. Culture of the fluid yielded *Salmonella chester*, sensitive to ampicillin, chloramphenicol, and gentamicin. The patient had been shown to have achlorhydria and to be passing *S chester* in his stools. A person involved in the preparation of the patient's food has been found to be a carrier of *S chester*.

Discussion

S chester is an unusual serotype which has been recognised as causing sporadic cases of salmonellosis. *S typhimurium*¹ and *S agona*² have been reported as causing splenic abscess, illustrating the need to consider *Salmonella* spp as a cause of splenic abscess.

Splenic abscess is commonly a metastatic infection, organisms multiplying in a post-traumatic subcapsular haematoma or simple cyst following bacteraemia, perhaps otherwise insignificant. In this case the achlorhydria was probably responsible for his developing bacteraemia. Less frequently, an abscess may arise as a direct extension of a contiguous infection, or as a primary feature in tuberculosis, typhoid, leprosy, melioidosis, and relapsing fever.³

The presentation with signs of infection and an intra-abdominal mass is typical,⁴ but it is difficult to differentiate splenic abscess from subdiaphragmatic, perinephric, and hepatic abscess, owing to the rarity, and the unfamiliar nature of splenic abscess. Careful plain and simple contrast abdominal radiography are the most useful investigations, but, where available, selective coeliac angiography may be more accurate.⁴

The treatment of choice is abdominal splenectomy and drainage, though in very sick patients, in the presence of dense adhesions and massive abscesses, splenotomy and drainage with interval splenectomy have been successful.^{1 4 5}

¹ Vita, A, *et al*, *Microbiologica, Parazitologia, Epidemiologia*, 1969, **14**, 53.

² Sharr, M, *British Medical Journal*, 1972, **1**, 546.

³ McSherry, C K, and Dineen, P, *American Journal of Surgery*, 1962, **103**, 618.

⁴ Davidson, S G, Doig, J A, and Everard, G J H, *Journal of the Royal College of Surgeons of Edinburgh*, 1960, **6**, 44.

⁵ Gadacz, T, Way, L W, and Dunphy, J E, *American Journal of Surgery*, 1974, **128**, 182.

(Accepted 3 December 1976)

Military Wing, Musgrave Park Hospital, Belfast

I H K SCOTT, MA, FRCS, Major, RAMC surgical specialist (present address: BMH Munster, BFPO 17)
H W THOMAS, MB, FRCS, Major, RAMC consultant surgeon
R O WALTERS, MRCP, DCH, Major, RAMC senior medical specialist (present address: BMH Rinteln, BFPO 29)

Adrenergic crisis due to phaeochromocytoma

A sudden crisis is a recognised complication of poorly controlled hyperthyroidism in patients exposed to infection or surgery.¹ We report a similar situation arising from a phaeochromocytoma.

Case report

A man aged 38 presented with vomiting and pain in the left shoulder and a history of polydipsia and polyuria for one month with weight loss of 6.4 kg. He was very ill. Although not feverish, he was sweating excessively and had a peculiar blue, mottled skin. His pulse rate was 160/min, blood pressure was 190/130 mm Hg, and chest x-ray examination showed consolidation in the right lower lobe and a mass in the left hypochondrium thought to be the spleen. The ECG showed a supraventricular tachycardia. The urine contained glucose 2% and acetone. The plasma glucose was 31.5 mmol/l (568 mg/100 ml) and the actual bicarbonate 18.9 mmol (mEq)/l. He was rapidly rehydrated with physiological saline and given 20 units of insulin intramuscularly immediately, followed by 10 units intramuscularly hourly.

Four hours later his pulse rate had risen to 180/min, the blood pressure was 160/130 mm Hg, the plasma glucose was 33.9 mmol/l (611 mg/100 ml),

and the actual bicarbonate 16.6 mmol/l. To control the arrhythmia practolol 20 mg was given intravenously over 30 minutes. He was also given 30 units of insulin intravenously and 30 units intramuscularly. Later he became feverish and ampicillin was started. His condition rapidly improved with an immediate fall in the blood pressure to 140/100 mm Hg and the pulse rate to 130/min. The plasma glucose then fell to 10.6 mmol/l (191 mg/100 ml) with an actual bicarbonate concentration of 23 mmol/l.

The patient remained normotensive (blood pressure 130/80 mm Hg) and his diabetes was controlled by Actrapid MC (28 units at 8 am, 24 units at 6 pm). The left hypochondrial mass was shown by intravenous pyelography and abdominal aortography to be a large, left suprarenal tumour. This proved to be a pheochromocytoma (homovanillylmandelate 238 nmol/24 h). At laparotomy after combined alpha- and beta-blockade,² the diabetes being controlled by continuous glucose-insulin infusion, a suprarenal tumour weighing 500 g was removed. Chromaffin staining showed that it was adrenaline secreting. After the operation the patient required no further insulin and the homovanillylmandelate output returned to normal.

Comment

The patient evidently had a chest infection, and this may have triggered off an acute storm, or crisis, due to adrenaline release. The evidence to support an adrenergic crisis is considerable. (1) The blood pressure fell from 190/130 to 130/100 mm Hg after intravenous practolol. (2) A moderate diabetic ketoacidosis was resistant to insulin treatment until after beta-blockade. (3) The inappropriate sweating was not due to fever. (4) The supraventricular tachycardia responded to practolol. (5) The patient's skin was cold, with vasoconstriction and a blue, mottled appearance.

A large rise in circulating adrenaline could explain the hypertension, sweating, tachycardia, and vasoconstriction.³ This would also decrease circulating insulin and initiate hepatic glucose release.⁴ Beta-blockade reduces the metabolic rate, lowers the blood pressure, and decreases the pulse rate in hypermetabolic patients with high urinary catecholamine output.⁵ In our patient beta-blockade was undoubtedly life saving. We feel justified in calling this a case of "adrenergic crisis."

We thank Drs J M Fowler and R P L Waldram for their help in preparing this case report.

¹ Werner, S C, and Ingbar, M D, *The Thyroid*, 3rd edn, p 660. New York, Harper & Row, 1966.

² Ross, E J, et al, *British Medical Journal*, 1961, 1, 191.

³ Goldenberg, M, et al, *Archives of Internal Medicine*, 1950, 86, 823.

⁴ Porte, D, et al, *Journal of Clinical Investigation*, 1966, 45, 28.

⁵ Wilmore, D, et al, *Annals of Surgery*, 1974, 180, 653.

(Accepted 10 December 1976)

Departments of Medicine and Chemical Pathology, Basingstoke District Hospital, Basingstoke

D J B THOMAS, MB, MRCP, medical registrar

C N MACDOUGALL, MB, senior house officer

P R BROUGH, MB, house officer

H S PLATT, BSC, MD, consultant chemical pathologist

Raised amniotic fluid concentrations of alpha-fetoprotein in a twin pregnancy

Amniotic fluid levels of alpha-fetoprotein (AFP) have been used since 1972 in the diagnosis of neural tube defects of the fetus.¹ At present the normal range for AFP in amniotic fluid is based on tests in single pregnancies because amniocentesis is rarely performed on patients with a multiple pregnancy. We report here a twin pregnancy, initially undiagnosed, in which amniotic fluid AFP levels were raised.

Case report

A 27-year-old primigravida was first examined at 13 weeks' gestation and the uterus noted to be large for dates. Ultrasonic scan showed an apparently

single pregnancy at 18 weeks, no abnormalities being detected. At this time plasma AFP was 225 µg/l and a second sample was 560 µg/l at 20 weeks' gestation, both considerably raised concentrations.² Clear amniotic fluid was obtained by amniocentesis at 21 weeks and the AFP level was 55 mg/l (upper limit of normal in single pregnancy at 21 weeks 24 mg/l).³ The cells in the amniotic fluid were karyotyped as normal male.

The patient was admitted to hospital for consideration of termination of pregnancy. A further ultrasonic scan was performed because the uterine size was greater than the period of gestation and showed a twin pregnancy. One infant had a biparietal diameter of 61.5 mm and the other was 31.8 mm, but only one fetal heart movement was observed (see figure). Abdominal x-ray examination gave further confirmation. A diagnosis was made of twin pregnancy with death of one fetus. The pregnancy continued to term and a normal baby boy weighing 2.9 kg was delivered as well as a fetus papyraceous.

Ultrasonic scan showing two fetal heads with grossly different biparietal diameters.

Discussion

Multiple pregnancy is usually associated with raised concentrations of AFP in maternal plasma. In general obstetric practice, once a multiple pregnancy has been diagnosed, the raised plasma AFP concentration is ignored and amniocentesis is not performed to exclude fetal neural tube defect. Nevertheless, amniotic fluid AFP concentrations may be raised in twin pregnancy in the absence of a fetal abnormality.^{4 5} In our patient the raised amniotic fluid AFP concentration may have been due to the multiple pregnancy or to death of one of the fetuses. The amniotic fluid appeared clear and normal, which virtually excludes the possibility that the amniotic sac with the dead fetus was sampled.

To avoid termination of a normal twin pregnancy on the basis of raised maternal plasma concentrations of AFP, leading to amniocentesis, the following criteria should be used. Firstly, a normal range of AFP concentrations in multiple pregnancy should be obtained, but it will take many years before this is available. Secondly, if there is a discrepancy between the gestational age assessed both clinically and by ultrasound, the ultrasound examination should be repeated to exclude a multiple pregnancy.

¹ Brock, D J H, and Sutcliffe, R G, *Lancet*, 1972, 2, 197.

² Leighton, P C, et al, *Lancet*, 1975, 2, 1012.

³ Gordon, Y B, et al, *British Journal of Obstetrics and Gynaecology*, 1976, 83, 771.

⁴ Brock, D J H, Scrimgeour, J B, and Nelson, M M, *Clinical Genetics*, 1975, 7, 163.

⁵ Pinker, G D, personal communication, 1976.

(Accepted 10 December 1976)

Departments of Human Reproduction, University of Southampton

A T LETCHWORTH, MD, MRCOG, senior lecturer

Departments of Obstetrics and Gynaecology, and Reproductive Physiology, St Bartholomew's Hospital Medical College and the London Hospital Medical College, London

Y B GORDON, MRCOG, FCOG(SA), lecturer

P C LEIGHTON, FRCS, MRCOG, lecturer

M J KITAU, AIMLT, technician