limb ischaemia in a vascular laboratory if the clinical diagnosis of claudication is in doubt.

References

- 1 Foley WT. Treatment of gangrene of the feet and legs by walking. *Circulation* 1957;15:689-700.
- 2 Skinner JS, Strandness DE. Exercise and intermittent claudication. II. Effect of physical training. *Circulation* 1967;36:23-9.
- 3 Larssen OA, Lassen NA. The effect of daily muscle exercise in patients with intermittent claudication. *Lancet* 1966;ii:1093-6.
- 4 Dahllöf AG, Holm J, Schersten T, Sivertsen R. Peripheral arterial insufficiency; effect of physical training on walking tolerance calf blood flow and blood flow resistance. *Scand J Rehabil Med* 1976;8:19-26.
- 5 Ekroth R, Dahllöf A, Gundeval B, Holm J. Physical training of patients with intermittent claudication: indications, methods and results. *Surgery* 1978;84:640-3.
- 6 Jonason T, Jonzon B, Rinqvist I, Oman-Rydberg A. Effect of physical training on different categories of patients with intermittent claudication. *Acta Med Scand* 1979;206:253-8.
- 7 Leading article. Intermittent claudication. *Br Med J* 1976;ii:1165-6.
- 8 Gollnick PD, Armstrong RB, Saubert CW, et al. Enzyme activity and fiber composition in skeletal muscle of untrained and trained men. *J Appl Physiol* 1972;33:312-9.
- 9 Merzbacher CF. A diet and exercise regimen: its effect upon mental activity and personality; a pilot study. *Percept Mot Skills* 1979;48:367-71.
- 10 McAllister FF. The fate of patients with intermittent claudication managed non-operatively. *Am J Surg* 1976;132:593-5.

(Accepted 21 March 1980)

SHORT REPORTS

Primary hyperparathyroidism presenting with visual failure and palatal swelling

We report the case of a patient with primary hyperparathyroidism in which the presenting symptoms included unilateral visual failure. This recovered completely after removal of a parathyroid adenoma.

Case report

A 47-year-old housewife presented with a midline palatal swelling for 10 weeks. She also complained that vision in her right eye had been diminishing over four weeks. She had had sciatica two years previously and still had diffuse back pain. She was edentulous, wearing full upper and lower dentures. There was a smooth, fairly soft swelling 3 cm × 1.5 cm in the midline at the posterior aspect of the hard palate, displacing the upper denture, and two small fusiform swellings on the lateral aspects of the maxillary alveolus in the incisor regions. Slight localised bossing of the left frontal bone was apparent. Visual acuity in the right eye was very poor and deteriorated to perception of light over 10 days. Corneal calcification was noted bilaterally at the limbal equator. The retinal arteries in the right eye appeared slightly attenuated and the pupil was sluggish, but there was no lens opacity, myopia, proptosis, impairment of ocular movement, or papilloedema and no ocular lesion sufficient to account for the loss of vision. Visual acuity in the left eye was normal. X-ray examination showed ill-defined radiolucent areas in the maxillary alveolus coinciding with the fusiform swellings. The right optic foramen appeared on tomograms as an ill-defined region of radiolucency with no clear margin. The left optic foramen appeared normal. The phalanges showed subperiosteal erosions. There were cystic changes in the pelvic girdle, ribs, and frontal bone.

Serum concentrations measured were as follows: calcium 3.85 mmol/l (15.4 mg/100 ml) (normal 2.1-2.6 mmol/l (8.4-10.4 mg/100 ml)); phosphate 0.72 mmol/l (2.3 mg/100 ml) (normal 0.8-1.4 mmol/l (2.5-4.5 mg/100 ml)); albumin 45 g/l (normal 34-50 g/l); alkaline phosphatase 303 IU/l (normal 20-90 IU/l); parathyroid hormone 6.65 µg/l (normal <1.0 µg/l) by radioimmunoassay using a human parathyroid hormone standard; creatinine 91 µmol/l (1.03 mg/100 ml) (normal 50-120 µmol/l (0.56-1.35 mg/100 ml)). An excision biopsy specimen of the palatal swelling showed a vascular, fleshy tumour completely eroding the bone of the hard palate, macroscopically typical of a giant-cell lesion. Histologically it was a giant-cell lesion containing a large amount of osteoid, some showing early ossification.

A large single tumour of the left inferior parathyroid gland weighing six grams was removed. Histologically it was typical of a parathyroid chief-cell adenoma. Postoperatively all the biochemical abnormalities resolved. She was given 0.5-2 µg 1-α-hydroxycholecalciferol daily for 12 weeks, by when the alkaline phosphatase concentration had returned to normal. Radiological healing of the skeletal lesions was subsequently confirmed. In particular, the right optic foramen became sharply defined, normal in size, and with a slightly sclerotic border. Vision returned in the right eye within two weeks of the parathyroidectomy. Acuity was 6/9 after six weeks and 6/5 after five months.

Comment

This patient had skeletal changes affecting many bones because of primary hyperparathyroidism. The palatal giant-cell tumour was

typical of lesions described in this condition and well known to affect the jaw bones.¹ Corneal calcification is associated with hypercalcaemia² and papilloedema has been reported,³ but visual failure due to hyperparathyroidism has not been recorded. It may have resulted from distortion of the neurovascular bundle in the optic canal by another giant-cell tumour, and this hypothesis is supported by the return of vision and restoration of the optic foramen (radiologically) that followed removal of the parathyroid adenoma.

We thank Drs A R Manners, E Dillon, and A K Lamballe for radiological advice, Drs R Lawler and H E Simpson for histological reports, and the Supraregional Assay Service (Middlesex Hospital Laboratory, London) for measurements of parathyroid hormone.

¹ Black BK, Ackerman LV. Tumours of the parathyroid. *Cancer* 1950; 3:415-44.

² Cogan DG, Albright F, Bartter FC. Hypercalcemia and band keratopathy. *Arch Ophthalmol* 1948;40:624-38.

³ Murphy KJ. Papilloedema due to hyperparathyroidism. *Br J Ophthalmol* 1974;58:694-7.

(Accepted 4 March 1980)

North Tees General Hospital, Stockton on Tees, Cleveland TS19 8PE
D CARR, MB, MRCP, consultant physician
B J P HALL-PARKER, LMSSA, DO, consultant ophthalmologist
B SUMMERSGILL, BCHD, FDSRCS, consultant oral surgeon
R W THOMSON, FRCS, consultant surgeon

Chronic thrombocytopenic purpura associated with toxoplasmosis

Many aetiological factors play a part in the pathogenesis of idiopathic thrombocytopenic purpura. Among these are viral infections,¹ drugs, and other autoimmune diseases.² Many cases are truly idiopathic. We report the case of a child with chronic thrombocytopenic purpura associated with toxoplasmosis. The parasite *Toxoplasma gondii* has not been reported as a possible cause of thrombocytopenic purpura.

Case report

A 2-year-old girl presented with purpura. One month earlier she had had a fever, but the cause was not known. Physical examination showed purpura, enlarged lymph nodes, and mild hepatosplenomegaly. The platelet count was 10-20 × 10⁹/l (10 000-20 000/mm³). The result of bone marrow examination was unhelpful. After a short course of steroid treatment the purpura and thrombocytopenia disappeared. A Sabin-Feldman dye test, performed because of an enlarged spleen and liver and lymphadenopathy, was positive at a titre of 1/4096. Three months later the titre was 1/1024, and one year later 1/256.

Three months after the first episode of purpura she presented again with diffuse purpura. The platelet count was $6 \times 10^9/l$ ($6000/mm^3$), and the results of examination of the bone marrow was consistent with the diagnosis of idiopathic thrombocytopenic purpura. She had clearly had a relapse. The results of tests for LE cells, antinuclear factor, and red-cell antibodies were negative. Cellular and humoral immunity function tests were normal. The thrombocytopenia and purpura persisted despite treatment with steroids and azathioprine, so a splenectomy was performed when she was 6 years old. After this she had a complete clinical and haematological recovery.

Comment

All but 5% of patients with thrombocytopenic purpura are considered to have the idiopathic form, in which no preceding infection can be shown. One-half of this group have the true idiopathic form and the other half the postviral idiopathic form. Possibly, therefore, a patient who is thought to have the true idiopathic form might move to the postviral group when an infectious illness can be shown to have preceded the onset of purpura. This may have been the case with our patient. She fulfils the criteria proposed by Feldman³ for the diagnosis of toxoplasmosis. A titre of 1/4096 in the dye test and a rising titre in two serum specimens are compatible with the diagnosis of a recently acquired infection.

Toxoplasma infection is much more common in people with compromised immunological states. This raises the possibility that both the idiopathic thrombocytopenic purpura and the toxoplasmosis in our patient resulted from an altered immune mechanism that was not detected by standard laboratory procedures. Cell-mediated immunity may play a part in the pathogenesis of idiopathic thrombocytopenic purpura. Recently, blast transformation of lymphocytes in the presence of autologous platelets was shown.⁴ Is the production of antiplatelet antibodies mediated through the inhibition of suppressor T cells? Or are there two parallel mechanisms: one of cell-mediated immunity and the other of humoral immunity? Is toxoplasma infection the result of an altered immune state, or did the parasite cause such a change in cell-mediated immunity that idiopathic thrombocytopenic purpura appeared? These and other questions await an answer.

Interestingly, we could not detect autoantibodies in the sera of our patient after splenectomy. In our laboratory we found a significant increase in the incidence of autoantibodies in patients who have had splenectomies. In view of our patient's condition, we suggest that the term "postviral idiopathic thrombocytopenic purpura" should be extended to "postinfectious idiopathic thrombocytopenic purpura" to include the possible infectious agents that, although they are not viruses, may play a part in causing thrombocytopenic purpura.

¹ Doan CA, Bouroncle BA, Weisman BK. Idiopathic and secondary thrombocytopenic purpura. *Ann Intern Med* 1960;53:861-9.

² McClure PD. Idiopathic thrombocytopenic purpura in children: diagnosis and management. *Pediatrics* 1974;55:68-74.

³ Feldman HA. Toxoplasmosis. *N Engl J Med* 1968;279:1370-5.

⁴ Wybran J, Fudenberg HH. Cellular immunity to platelets in idiopathic thrombocytopenic purpura. *Blood* 1972;40:856-61.

(Accepted 4 March 1980)

Department of Paediatrics "A," Rokach Hospital and Sackler School of Medicine, Tel Aviv University, Israel

S DIAMANT, MD, chief physician

Z SPIRER, MD, head, division of paediatrics

Primary thrombocythaemia in monozygotic twins

The aetiology of myeloproliferative disorders is unknown but there is some evidence that genetic factors may play a part. Although there are several reports^{1,2} of familial polycythaemia there are very few of primary thrombocythaemia. This report is of primary thrombocythaemia in monozygotic twins.

Case report

A 35-year-old man (A) had lost 6 kg in weight when he presented with a 10-week history of partial blindness in one eye. He had a right subhyloid haemorrhage and his spleen was enlarged 7 cm below the costal margin.

He was anaemic and had a platelet count of $954 \times 10^9/l$ ($954\,000/mm^3$). His asymptomatic twin brother (B) had a radiologically enlarged spleen and a platelet count of $1330 \times 10^9/l$ ($1\,330\,000/mm^3$) but he was not anaemic. The full blood counts are given in the table. B's blood film was morphologically normal but that of A showed poikilocytes, teardrop cells, and target cells. Their bone marrows yielded only cells of the normal male karyotype. The morphology of marrow smears and bone trephines from both men were similar. The smears contained no stainable iron, erythropoiesis was normoblastic, and there were no dyserythropoietic changes. There were sheets of platelets and numerous megakaryocytes. There was no increase of reticulin and no fibrosis in the bone trephines. All other investigations, including measurement of the leucocyte alkaline phosphatase concentration, were normal except that A had hyperuricaemia, with uric acid concentration 0.59 mmol/l (10 mg/100 ml) (normal 0.15-0.42 mmol/l (2.5-7.0 mg/100 ml)).

Blood counts in monozygotic twins with primary thrombocythaemia

	Age when tested (years)	Haemoglobin (g/dl)	Mean cell vol (fl)	White cells ($10^9/l$)	Platelets ($10^9/l$)	Red cells ($10^{12}/l$)
A	35	11.8	87	12.4	954	3.84
B	35	13.5	88	14.9	1330	4.39
Father	67	11.4	93	6.7	328	3.59
Mother	65	15.7	90	8.5	250	4.92
Sister	37	12.3	88	6.3	235	3.93
Daughter of A	15	12.7	90	7.8	390	4.28
Son of A	11	12.3	84	8.6	390	4.48
Son of B	7	11.8	81	7.4	350	4.41

Conversion: SI to traditional units—Mean cell volume: 1 fl = $1 \mu m^3$.

The red cell genotype of the twins and their close relations together with tissue typing studies suggested that the twins were monozygotic. None of their relations had thrombocytosis. The twins were both treated for primary thrombocythaemia. After initial treatment with radioactive phosphorus the platelet count of both men fell below $400 \times 10^9/l$ ($400\,000/mm^3$) within four months. By then A had gained 7 kg and his haemoglobin concentration had risen to 13.6 g/dl. During the subsequent six years neither has developed polycythaemia but both have had several courses of busulphan when their respective platelet counts rose above $400 \times 10^9/l$.

Comment

Dedichen *et al*³ in a paper describing nine patients with haemorrhagic thrombocythaemia included a 40-year-old woman with five similarly affected siblings. The platelet count of four of the siblings was not available but the fifth, a man, had splenomegaly associated with thrombocythaemia. Beretta Anguissola and Prato⁴ described thrombocythaemia associated with gastrointestinal haemorrhage in three male members of three generations of an Italian family. Flickers and Speck⁵ described a 47-year-old man with primary thrombocythaemia. He had been taking thiotepea for two years when cytogenetic studies showed hypodiploidy in 8 of 70 karyotypes. His sister, who had hepatosplenomegaly and thrombocythaemia, refused to have a marrow aspirate, but probably her brother's chromosomal abnormalities were the result of cytotoxic therapy.

The present report is unique in that it describes primary thrombocythaemia in monozygotic twins. There is no evidence that any of their close relations has a similar disorder, but the presentation of the same myeloproliferative disease in the twins is evidence that a genetic factor may play some part in its pathogenesis.

I thank Professor S Peart for referring the patient, Dr S Lawler for the cytogenetic studies, and Dr R Sanger for red cell genotyping.

¹ Modan B. Polycythaemia. A review of epidemiological and clinical aspects. *J Chronic Dis* 1971;18:605-45.

² Levin WC, Houston EW, Ritzmann, SE. Polycythaemia vera with Ph' chromosomes in two brothers. *Blood* 1967;30:503-12.

³ Dedichen J, Refven O, Ytreus K. Essential thrombocythaemia. *Nord Med* 1959;62:1265-70.

⁴ Beretta Anguissola A, Prato V. Trombocitosi ereditaria, prima osservazione. *Minerva Med* 1961;52:4545-8.

⁵ Flickers M, Speck B. Thrombocythaemia. Familial occurrence and transition into blastic crisis. *Acta Haematol (Basel)* 1974;51:257-65.

(Accepted 4 March 1980)

Department of Haematology, St Mary's Hospital Medical School, London W2 1PG

HELEN DODSWORTH, MRCP, MRCPATH, senior lecturer