performed in this hospital with no untoward effects, and no side effects were recorded during this study.

The practical application of myocardial scanning in the patient with acute myocardial infarction would be enhanced by the addition of portable scanning facilities.

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SHORT REPORTS

Splenoma with Portal Hypertension

Splenomas or splenic hamartomas are non-capsulated, single or multiple nodules in the spleen and consist of native elements in gross disproportion.¹ The first case of splenoma was recorded in 1865 by Rokitsky,¹ and until August 1970 only 39 cases had been reported.² All but four of these were asymptomatic (excluding abdominal swelling) and discovered accidentally at exploratory laparotomy or necropsy. Of the four symptomatic cases two had pancytopenia,^{2,4} one had anaemia,^{4,5} and one had thrombocytopenia.³ We report here the first recorded case of symptomatic splenoma associated with haemodynamically proved portal hypertension but without any of the reported haematological abnormalities.

Case Report

A 35-year-old Kashmiri Moslem weaver presented with pain in the left abdomen of 11 months' duration. The liver was enlarged (3 cm) and non-tender, and there was a palpable mass in the left hypochondrium, which moved slightly with respiration. The routine haematological measurements showed nothing abnormal except occasional Howell-Jolly bodies in the peripheral smear. The results of urine and stool analysis and liver function tests were normal. Blood sugar, urea, and creatinine were normal. Intravenous pycnography, barium meal examination, and serum electrophoresis for proteins showed nothing abnormal. Immunological studies showed proportions of normal T and B cells in blood, normal T-cell function, poor T-cell response to phytohaemagglutinin, and poor B-cell population in the spleen.

A splenoportogram showed a 5-cm circular dye-opaque area drained by a tortuous aberrant large vein (1.5 cm), joining a small main splenic vein before the formation of the portal vein. The haemodynamic studies showed high intrasplenic pressure (300 mm H₂O; normal, 150 mm H₂O), slightly raised wedge hepatic venous pressure (7.56 mm Hg; normal 5.4, S.D. 1.8), raised free hepatic venous pressure (6.12 mm Hg; normal 3.1, S.D. 1.6 mm Hg); and raised estimated hepatic blood flow (1.27 l/min; normal 0.8-1.2 l/min). Splenic vein thrombosis was diagnosed preoperatively.

At exploratory laparotomy a vascular tumour of the spleen was found as well as interconnecting vascular channels among the greater omentum, the posterior wall of the stomach, the splenic flexure, and the splenic tumour. The liver seemed to be fatty. Splenectomy and devascularization of the stomach were performed, and a wedge biopsy specimen of the liver taken.

The spleen weighed 290 g (see figure) on the surgical desk. There were three distinct parts; the main spleen, an "isthmus," and the tumour mass. The isthmus connected the lower pole of the spleen to the tumour. Histological examination showed a transition from normal to abnormal splenic architecture. The latter consisted of malformed, ill-defined lymphoid follicles "invading" the red pulp. The red pulp was unremarkable. Splenic vasculature showed perivascular haemorrhages and subintimal fibrous and fatty plaques suggestive of portal hypertension. The liver showed portal fibrosis. Splenoma with predominating abnormal lymphoid components was diagnosed.

Comment

The high intrasplenic pressure, the drainage of portosystemic collaterals into the azygos system, and the histological character of

External surface of spleen showing tumour (T) arising from lower pole of spleen (S) with interconnecting "isthmus" (I).

the splenic vasculature were considered adequate evidence of portal hypertension.

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Postgraduate Institute of Medical Education and Research, Chandigarh, 160011, India

A. G. BHAGWAT, M.D., F.R.C.P., Assistant Professor of Pathology
D. V. DATTA, M.D., PH.D., Associate Professor of Hepatology
SUNANDA MITRA, M.S., Pool Scientist in Surgery
B. K. AIKAT, M.D., PH.D., Director and Professor of Pathology

Ocular Perforating Injury Caused by a Sparrow

Ocular conditions caused by birds are not as rare as might be supposed. Thus a sensitive person may develop acute allergic blepharoconjunctivitis through contact with feathers. The Newcastle virus, which causes fowl-pest, can cause acute follicular conjunctivitis. Psittacosis can cause iritis and subacute focal retinitis leading to macular oedema.¹

Bird seed husks may become adherent to the eye and set up a foreign body irritation. Kühl² showed that ocular injuries caused by birds tend to occur in the spring and be specific for the type of bird. Thus owls (10 cases) with curved beaks and claws tend to attack at dusk and cause limbal and scleral perforating injuries with severe intraocular damage and consequently a poor prognosis. Injuries from chicken and cocks (four cases) show a similar pattern. Birds with sharp straight beaks, however, like blackbirds (two cases),² storks, cranes, herons (five cases),³ and even bitterns (one case),⁴ cause clean central perforations, often with little associated intraocular damage, which heal spontaneously and have a good prognosis. This paper reports a perforating injury caused by a sparrow.

Case Report

A previously healthy 36-year-old woman was walking in a shopping precinct in March 1974 when a sparrow flew into her right eye. She was seen two hours later with a 5-mm central corneal perforating injury and a flat anterior chamber. Four hours after the injury the anterior chamber had reformed and showed an aqueous flare and cells (see fig.). The vision was reduced to counting fingers at six feet. The pupil dilated well with mydriatics and there was a faint paracentral disturbance of the lens capsule within the pupillary aperture where the tip of the sparrow's beak had presumably just touched the lens. She was treated with antibiotics, padding, and bandaging, and as the corneal oedema and scarring of the wound edges cleared the vision improved. Six months later it was 6/24 unaided and could be improved with a pin-hole to 6/6. On gonioscopy the anterior-chamber angle appeared to be recessed, but the intraocular pressure was not raised. The lens capsule disturbance had not progressed and there was no underlying lens opacity. The retina was normal, and at the time of writing all treatment had stopped. Her intraocular pressure will continue to be monitored as the angle recession could lead to a late rise in pressure. Her left vision was normal, and as a visual acuity of 6/24 in her right eye was acceptable to her a contact lens or a corneal graft was not considered.

Right eye (A) Six hours after central corneal perforating injury, and (B) Six months later.

Discussion

This case supports Kühl's² conclusions on the seasonal timing, nature, and prognosis of an ocular injury caused by a bird with a short straight beak. It also emphasizes the chances of success with conservative management in such cases. The lack of complications was presumably due to the speed and cleanness of the perforation, which caused

minimal damage to intraocular structures and allowed the anterior chamber to reform spontaneously.

Janiszewska-Zygier⁵ drew attention to the fact that most owl injuries affect teenage boys and occur in the breeding season. The same is true of storks, cranes, and herons.³ All these birds are prepared to attack the eyes of anybody endangering their young. It is not known why the eyes should be specifically attacked, but presumably an animal instinctively attacks a bright flashing object, just as fish will attack a spinner.

Since this is the first reported case of an ocular perforating injury caused by a sparrow it was presumably accidental and, as such, represents an extremely rare hazard of shopping precincts.

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Moorfields Eye Hospital, London WC1V 7AN
J. R. O. COLLIN, M.A., F.R.C.S., Senior Registrar

Treatment of Acrodermatitis Enteropathica with Zinc Sulphate

The effectiveness of zinc for treating acrodermatitis enteropathica^{1 2} is borne out by the following case.

Case History

A man born in 1946 had no family history of skin disease or consanguinity. Breast-feeding was discontinued at 6 weeks because of failure to gain weight, and a rash, diagnosed as eczema, appeared at 3½ months. This persisted until he first presented at age 3½ years with primary baldness, dental caries, episodic diarrhoea, and scaly, erythematous lesions on neck, face, trunk, buttocks, and legs. A few bullae were seen on the legs. Haemoglobin was 9.7 g/dl and there was radiological evidence of healed rickets. A diagnosis of candidiasis was unsubstantiated. In 1953 acrodermatitis enteropathica was diagnosed and di-iodohydroxyquinoline (Diodoquin) 210 mg daily instituted. He remained fairly well on this treatment until he stopped attending in 1959.

In 1964 he returned with a 10-month relapse of his skin lesions together with dystrophic nails, scanty scalp hair, absent pubic and axillary hair, gynecomastia, and undescended testes. Urinary oxosteroids and oxogenic steroids were 8.3 and 12.5 µmol/24 h (2.4 and 3.6 µg/24 h), respectively (normal 24-83 µmol (7-24 µg) and 21-69 µmol (6-20 µg)/24 h, respectively), bone age was 15½ years, and skin biopsy showed non-specific inflammatory changes. Investigations giving normal results included full blood count, urea and electrolytes, serum calcium, liver function tests, cholesterol, faecal fats, urinary amino-acids and porphyrins, x-ray pictures of chest and pituitary fossa, barium meal and follow-through, jejunal biopsy, and buccal smear.

He was treated with di-iodohydroxyquinoline 625 mg thrice daily, chorionic gonadotrophin 1500 units twice weekly for one year, and beta-methasone valerate ointment 0.1% topically. Bone age and gonadal development greatly improved, scalp hair grew, and the skin showed a good response.

When he again stopped attending in 1970, however, his face, hands, and feet were severely affected.

In November 1974 he was referred back with extensive erythematous, scaling, crusted, infected, and malodorous lesions, especially affecting face, neck, hands, feet, and lower legs. He had suffered no recent deterioration in his skin and for the four intervening years had lived as a recluse. His face showed telangiectatic and atrophic changes from the long-term application of fluorinated corticosteroids. His nails were grossly dystrophic and his teeth carious and he had no beard growth, though his scalp hair appeared normal. Skin biopsy showed non-specific changes. *Staphylococcus aureus* was cultured from the skin, and serum zinc estimations on three specimens were 4.9, 8.7, and 5.6 µmol/l (32, 57, and 37 µg/100 ml) (normal range 7.6-15.2 µmol/l; 50-100 µg/100 ml).

He received zinc sulphate 220 mg thrice daily and topical 3% clioquinol ointment and noticed an improvement within a week. After a month his face was clear apart from some residual telangiectases, his hands showed only postinflammatory pigmentation, and his legs, which had been the worst affected, were greatly improved. His beard had begun to grow but there was no change in his dystrophic nails.