However, in certain aspects the case differs from the typical picture. Thus, it occurred in a girl: of the cases recorded only 10 to 15% have occurred in females. There was an immediate preceding history of abdominal pain of three days' duration: though this does occasionally occur, it is not common. The acute colicky pain complained of on the night before admission was probably due to haemorrhage into the bowel with distension of the ileum. Gray stressed that there is a sudden change from relative well-being to an acute illness in any acute lesion of Meckel's diverticulum. In my case there was no sudden change and it is difficult to state when the perforation occurred.

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Reversible Papilloedema in Pernicious Anaemia

It is recognized that anaemia may be a factor in the causation of papilloedema, yet reports of its occurrence in Addisonian pernicious anaemia are extremely rare.

CASE REPORT

A ship's boatswain aged 58 was admitted to hospital on February 1, 1949, with severe pernicious anaemia of one month's duration. In July, 1946, he first became ill, but was inadequately treated. His vision had been blurred for three weeks, and three days before admission he noticed swelling of the ankles.

On admission he was gravely ill, grossly anaemic, and slightly jaundiced. There was slight pitting oedema over the sacrum. Enlargement of the liver, spleen, or lymph nodes was not observed. The nutritional state was good and no source of blood loss was discovered. The blood pressure was 120/50. There was no abnormality of the heart, lungs, abdominal organs, or nervous system. The urine contained an excess of urobilinogen but no bile salts or pigments and no albumin.

A blood count on February 2 was: red cells, 1,030,000 per c.mm.; haemoglobin, 4.9 g. per 100 ml.; M.C.H., 37.7 $\gamma\gamma$; white cells, 3,000 per c.mm. Films showed macrocytosis, poikilocytosis, polychromasia, reticulocytes 0.2%, and no malaria parasites.

"Anahaemin" produced a dramatic clinical and haematological response with a reticulocyte "crisis" of 29.2% nine days after the beginning of treatment. On March 19 his blood count was: red cells, 4,880,000 per c.mm.; haemoglobin, 14 g. per 100 ml.—a gain of 3,850,000 red cells and 9.1 g. of haemoglobin in 45 days. On February 27 a thrombocyte count was 533,170 per c.mm., with coagulation time 5 minutes 10 seconds and bleeding time 2 minutes 15 seconds. The sacral oedema had disappeared by the twelfth day of treatment. Serial blood pressures were normal after the first week, and the Hess test for capillary resistance was consistently negative throughout his stay in hospital. He was discharged on April 4 for repatriation to England.

OPHTHALMIC REPORT

On admission both pupils were contracted, ocular movements and intraocular tension were normal, and visual acuity (right 6/24, left 6/36) was not improved by lenses.

Bilateral papilloedema was more marked on the right than on the left; venous engorgement and multiple retinal haemorrhages were observed. The arteries seemed to be normal. In both fundi, haemorrhages overlapped the disk margins and, more peripherally, multiple flame-shaped haemorrhages in the nerve-fibre layer were scattered in the nasal and temporal areas. A deep oval-shaped haemorrhage with clearly defined edge was seen one disk diameter supertemporally from the right optic disk. An inverted-comma-shaped haemorrhage projected anteriorly from the lower margin of the left macula, and one disk diameter below this a circular haemorrhage was seen in the deeper retinal layers.

During the next month the papilloedema gradually subsided. On March 1 visual acuity was: right, 6/18, with +2.25 D correctable to 6/6; left, 6/12 not improved with lenses. The haemorrhages were being absorbed on both sides and the swelling of the nerve heads had almost subsided.

On March 15 the patient complained of blurred vision with the left eye. Examination next day revealed a fresh macular haemorrhage. Papilloedema had subsided and the disks were pale.

On March 31 visual acuity was: right, 6/12, correctable to 6/6; left, 6/24 not correctable by lenses. Nearly all the haemorrhages originally seen had been absorbed and their previous sites were discovered with difficulty. The more recent left macular haemorrhage was being absorbed but was still evident, and the pallor of the disks was more pronounced on the right side than on the left.

COMMENT

The pathogenesis of papilloedema, as of most pathological types of oedema, is obscure. Watkins *et al.* (1941) reported three cases of thrombocytopenic purpura and a case of anaemia due to bleeding gastric ulcer in all of which papilloedema was noted. In discussing the mechanism, they postulated a local reaction of the tissues of the optic nerves to anoxaemia. A general oedema of the brain due to the same mechanism is also mentioned as a possible factor. They did not enlarge on this, but presumably increased capillary permeability due to oxygen lack is responsible for the abnormal accumulation of extracellular fluid. It is not clear why this process should affect one optic nerve more than the other (in their fourth case the left showed elevation of 4 to 5 dioptres, the right only 1).

Simpson (1948) reported three cases of emphysema with papilloedema. He attributed the latter to a rise of cerebrospinal fluid pressure from cerebral vasodilatation due to oxygen lack and carbon dioxide accumulation in the arterial blood. It is possible that such a mechanism may operate in anaemia. However, these theories imply a threshold level of anoxaemia and cerebrospinal fluid pressure beyond which one would expect papilloedema to occur. Yet C.S.F. pressure may be high and many cases of severe anaemia may be seen without papilloedema being observed. Furthermore, the swelling may be much more prominent on one side than on the other. It would seem, therefore, that there must be some local factor the nature of which is not apparent.

This paper is submitted by permission of Mr. A. C. Turner, O.B.E., F.R.C.S.Ed., Chief Medical Officer, Anglo-Iranian Oil Company.

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