injecting into the sac 4 c.cm. of lithocaine (lithium salicylate 30% and tutocain 1%), followed by 2 c.cm. of quinine-urethane. The parts are thoroughly massaged after the injection, and the patient is ordered to wear a testicular support. A week later the fluid which accumulates in the tunica vaginalis as the result of chemical irritation of the delicate endothelial lining is cautiously aspirated.

The following case is of great interest, as it proves conclusively that a hydrocele of enormous proportions and of many years' standing can be treated successfully by the sclerosing method.

Case Report

The patient, aged 42, consulted me on August 2, 1939, complaining of an "enlarged testicle." He had first noticed the swelling in 1928, but it had not increased much in size until the last three years. It then grew rapidly and caused a great deal of embarrassment on account of its weight and gigantic size. In spite of the fact that he was short, stout, and very stockily built, with consequently loose-fitting clothing from the waist down, it was apparent the moment he entered the room that there was something very abnormal amiss, and when he sat down it looked as if he was carrying a football in his left trouser pocket.

Examination revealed a giant hydrocele of the following dimensions: length 10¾ inches, circumference 17½ inches. When tapped, 64 oz. of typical straw-coloured hydrocele fluid was evacuated. After the tapping was completed 5 c.cm. of quinine-urethane was injected, followed by 10 c.cm. of lithocaine. This mixture was thoroughly agitated inside the sac, and the patient was then sent home and told to retire to bed early in the evening.

On September 16 a second tapping was carried out, and 24 oz. of fluid was withdrawn. This time I injected 2 c.cm. of quinine-urethane and 8 c.cm. of lithocaine into the hydrocele sac. On October 23 a further 12 oz. of fluid was withdrawn, and another combined injection was given. The patient was told to return in six months' time, and when he did so, on April 24, 1940, I was disappointed to find that the hydrocele, although much smaller, was still present and appeared to have a very thick wall. He was tapped once again, 10 oz. of fluid being obtained, and a further injection of the two sclerosing media was undertaken. I did not see him again until May, 1941, when I was agreeably surprised and pleased to find that I had at last effected a complete cure by the injection method. The testicle on the left side was then of normal size, and there was no undue thickening or discomfort subsequent upon the extensive fibrosis which had occurred.

The patient was examined by Mr. Rodney Maingot at the Southend General Hospital in July, 1941, when it proved impossible on clinical examination to ascertain on which side the hydrocele had been, thus suggesting that a complete cure had been achieved.

Hadleigh, Essex. W. L. James, M.R.C.S., L.R.C.P.

Transient Blindness following Blood Transfusion

The following is an interesting case of blindness after blood transfusion.

Case Report

A man aged 42 was admitted to hospital on May 29, 1941, complaining of flatulent dyspepsia, loss of weight, and weakness. Clinical examination showed marked anaemia and obvious loss of weight. There were no abnormal physical signs in the respiratory, cardiovascular, or central nervous system. There was, however, an indefinite mass palpable in the epigastrium with some tenderness and muscular guarding over this region. He was put on Meulengracht's diet with alkalis.

The reports of the radiological and laboratory investigations were as follows. Barium meal: marked delay in emptying of stomach; pathological stenosis of the pyloro-duodenal region, nature not manifest, but could be the result of ulceration or of neoplasm, the former being the more likely. Blood picture (May 30): Red cells 2,700,000; white cells 8,000; haemoglobin 50%; film showed anisocytosis and central pallor of the red cells. Fractional test meal showed low total acidity, rising to a maximum in two and a half hours: free HCl 0.2%; total acidity 0.45%. Wassermann reaction negative. Blood pressure 108/62. Urine: deposit consisted of urates only; no albumin or sugar present.

On June 9 the patient was given a blood transfusion of Group O (IV) blood with a view to improving his condition preparatory to laparotomy. The blood was given at the rate of 2 c.cm. per minute approximately, without incident. Fifteen minutes after the completion of the transfusion he had a very slight rigor, but no rise in temperature; this lasted about ten minutes, after which the patient slept. An hour later he awoke and complained that he was blind. On examination there was no perception of light, his pupils were dilated but reacted to light; there was no evidence of arteriospasm or of retinal haemorrhages on examination of the ocular fundi, and the disks appeared normal. Fifteen minutes later perception of light returned, and after a further thirty minutes he could perceive objects. His vision then improved steadily until one and a half hours after the onset of the condition it was normal. The patient's general state remained very good throughout, and no other physical signs were discovered in his central nervous system. On the following day the urine contained a considerable quantity of blood and albumin, but this cleared up in twenty-four hours.

Clinically, the patient appeared to be very much improved after the transfusion, but the blood count showed: red cells 2,000,000, haemoglobin 50%, blood urea 64 mg. per 100 c.cm., blood non-protein nitrogen 55 mg. per 100 c.cm.

Unfortunately it was not possible to test the donor blood against that of the recipient. The recipient's group, however, was O (IV), as was the donor's blood.

Discussion

The possible causes of this reaction, in view of the transient nature of the blindness and the absence of positive findings by ophthalmoscopic examination, were considered to be: (1) oedema of the optic tracts; (2) arteriospasm associated with hypertension; (3) arteriospasm following severe blood loss; (4) air embolus; (5) uraemic blindness.

1. It is difficult to see how oedema of the optic tracts would have affected the optic nerves without producing other evidences of oedema in the central nervous system and elsewhere, and one would expect to find some evidence of such a condition on ophthalmoscopic examination.

2. Arteriospasm associated with hypertension can be excluded, since the patient's blood pressure was 108/62 before the transfusion and not materially different twenty-four hours later.

3. If arteriospasm following severe blood loss was the cause one would expect to find evidence of arteriospasm in the retinal vessels, but this was not present; also there was no sign of recent severe haemorrhage.

4. While air embolus is a possibility, air which enters the circulation during transfusion is almost certainly absorbed before reaching the arterial circulation and usually produces symptoms in the right ventricle or pulmonary circulation. Further, the bilateral blindness in the absence of other symptoms is difficult to explain on these grounds.

5. Uraemic blindness is also a possibility in view of the somewhat raised blood urea and N.P.N., but no other signs or symptoms of uraemia developed subsequently.

From these observations it will be seen that no definite aetiological factor has been decided upon. It would be interesting to hear if any readers have had similar cases, and whether they have been able to explain them. One fact should, however, be noted—that is, in this case vision was normal in about three hours, whereas in cases of transient blindness previously reported it has persisted for longer periods, even up to twenty-four hours.

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