Renal failure and suprarenal calcification after secondary haemorrhagic disease in a newborn baby

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Summary
A newborn baby who was severely asphyxiated at birth developed secondary haemorrhagic disease which was treated successfully with intravenous heparin. However, she was later found to have supra-renal calcification, chronic renal insufficiency and hypertension. It is felt that prompt treatment of conditions that predispose to secondary haemorrhagic disease is very important as, once this has occurred, treatment may not prevent permanent damage to vital organs.

Introduction
Secondary haemorrhagic disease of the newborn occurs mainly in association with severe birth asphyxia, hypothermia and rhesus haemolytic disease (Chessells and Wigglesworth, 1970) and often results from disseminated intravascular coagulation (Boyd, 1967). Treatment with heparin (Whaun,Ormson and Oski, 1971) or exchange transfusion (Gross and Melhorn, 1971) has been advocated, but the mortality is 60% (Whaun et al., 1971). Although fibrin deposits have been found in many organs at autopsy (Boyd, 1967) we have not found reports of permanent damage to organs other than the brain in the survivors (Bryant et al., 1970). Case report
This baby girl was born at 42 weeks' gestation after an antepartum haemorrhage by normal vertex delivery in a nearby maternity home and weighed 3.85 kg. She failed to breathe and after 10 min was treated with positive pressure ventilation via an endotracheal tube, regular respiration being estab-
blood transfusion, she was treated with intravenous heparin which was continued for 4 days. With heparin therapy her platelet count rose rapidly and thereafter remained normal, and the level of fibrin degradation products declined. The course of this part of her illness is illustrated in Fig. 1.

**Fig. 1.** Haematological findings in the first 2 weeks of life in a baby with secondary haemorrhagic disease. T = blood transfusion; ET = exchange transfusion.

At the age of 12 days she had more fits, and her weight had increased by 260 g in 24 hr. Results of investigations were: blood urea 108 mg/100 ml; serum bicarbonate 12 mEq/l; sodium 100 mEq/l; creatinine 5.1 mg/100 ml. She was treated with normal saline and sodium bicarbonate intravenously, fluid restriction and one injection of frusemide. By the next day her weight had fallen by 100 g, and the serum sodium had risen slightly to 104 mEq/l and 24 hr later she had lost a further 60 g and the serum sodium was 145 mEq/l. Thereafter she improved steadily and began bottle feeding. She gained weight satisfactorily on SMA feeds, muscle tone became normal, her blood urea fell to 69 mg/100 ml and she was discharged home taking additional sodium bicarbonate.

Radiographs 4 months after birth showed calcification of the suprarenal glands and small irregular kidneys (almost 1 s.d. below the mean for her length) with an area of medullary necrosis in the left kidney. Creatinine clearance a month later was 20 ml/min/1.73 m² and plasma cortisol, which was 37 μg/100 ml before an injection of ACTH, rose to 57.5 μg/100 ml at 30 min and 70 μg/100 ml at 90 min. She has gained weight steadily despite a blood urea level of 70 mg/100 ml. At 8 months of age her psychomotor development was equivalent to 7 months and she had developed hypertension which was treated with bethanidine.

**Comment**

This baby had evidence of disseminated intravascular coagulation, bleeding, thrombocytopenia and elevated fibrin degradation products, after severe birth asphyxia and acidosis, known precipitating factors of secondary haemorrhagic disease. Exchange transfusion with fresh heparinized blood, as advocated recently (British Medical Journal, 1971) caused only temporary improvement but after starting heparin therapy there was prompt and sustained resolution of the haematological abnormalities. We feel that in severe cases of secondary haemorrhagic disease where therapy may be needed for several days, heparinization is more efficacious than reperfused exchange transfusions.

The low sodium levels found when the baby was having fits at 12 days of age were probably due mainly to overhydration, for she had gained 260 g in the preceding 24 hr, but may also have been due partly to urinary loss of sodium which is known to occur in renal papillary necrosis. Unfortunately urinary sodium was not measured.

The baby survived, but now has supra-renal calcification and chronic renal insufficiency. Though cortisol production is normal at the moment, adrenal failure may occur in later life (Cathro, 1969). The nature of the kidney lesion is not certain. Bilateral renal vein thrombosis is unlikely, as neither kidney was palpable, and recovery is extremely uncommon (Verhagen, Hamilton and Genel, 1965). The radiological appearance in the left kidney suggests that partial medullary necrosis occurred. This causes sodium loss, but not usually massive haematuria, and renal function generally returns to normal (Crispin, 1972). We believe, therefore, that, in addition, widespread glomerular capillary thromboses occurred, as in the haemolytic uraemic syndrome, causing chronic renal insufficiency.

Fibrin deposits are widespread at autopsy in babies who have had secondary haemorrhagic disease and, as Chad et al. (1971) point out, it cannot be assumed that survivors escape unscathed. However, apart from the finding that some babies, who probably had disseminated intravascular coagulation, showed evidence of brain damage (Bryant et al., 1970), we have not found reports of permanent injury in those who have recovered. With better recognition and treatment of this condition the number of survivors will rise and impaired function of vital organs is likely to be detected more often.

We feel that prompt treatment of birth asphyxia and acidosis is important in the prevention of secondary haemorrhagic disease, for once disseminated intravascular coagulation has occurred it may be too late to prevent damage to vital organs.
Case reports

The treatment of priapism by corpus-saphenous by-pass

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Summary

Two cases of priapism treated by corpus-saphenous by-pass are presented. Priapism responded to treatment successfully in both cases. Satisfactory erection and sexual intercourse followed 6 weeks after operation in one case. The second patient failed to have further erections. It was thought that this was due to the length of time (6 weeks) between onset of priapism and operation.

Introduction

Priapism is a pathologically prolonged erection not associated with sexual desire and usually painful. Priapism is due either to increased neurogenic stimulation or more usually clotting in the corpora cavernosa and prostatic plexus and obstruction of the venous outflow of the penis (Hinman, 1960). Thus in 1964 Grayhack et al. devised a method of diverting the venous outflow via a long saphenous shunt to the femoral vein. As the corpora cavernosa communicate distally with each other a unilateral shunt is usually sufficient. In this paper, two cases of priapism treated by corpus saphenous by-pass are presented.

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References


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