RENAL VENOUS THROMBOSIS IN THE NEWBORN

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Thrombosis of the renal vein was first described by Rayer in 1837. This was followed in 1877 by Hutinel’s account of forty-two cases of renal thrombosis, in thirty of which the condition was bilateral. Abeshouse (1945), in reviewing 228 cases of renal thrombosis of all varieties, found ninety-eight of these in children less than a year old, and of this total ninety occurred in infants during the first two months of life. Barenburg et al. (1941) reported five cases in a series of twenty-five necropsies carried out on infants with gastro-enteritis—a figure which suggests a rather high incidence in this selected group of cases. At the Birmingham Children’s Hospital over a period of five years and out of a total of 1,430 routine post-mortem examinations, six instances of renal haemorrhagic infarction were found. In four of these cases the main renal trunk was thrombosed. In the remaining two cases the thrombosis was confined to the interlobular veins and was therefore detected only on microscopic examination (figs. 1 and 3). A survey of the literature was made by Morison in 1945, with particular reference to renal thrombosis and infarction in the newborn, and he includes a description of a series of fatal cases.

Renal thrombosis may be primary, that is, the thrombotic process originates within the renal venous system; or secondary, resulting from extension of a thrombus along the vena cava or tributaries of the renal vein. This latter variety is rare in childhood. Sandblom (1948) has further subdivided the cases arising during the neonatal period.

1. A primary, rare form with no obvious etiology, and predominating urinary symptoms.

2. The usual variety encountered in infancy, which is secondary to infection with dehydration, and in which the urinary changes are minimal.

Ante-mortem diagnosis of the condition is rare, due probably to the rapid dissolution which almost invariably follows the onset of symptoms. A case of unilateral thrombosis, of his first type, was described by Sandblom (1948), in which nephrectomy appeared to be a life-saving measure in a five-day-old infant. Campbell and Matthews (1942) had previously described two similar cases with recovery following nephrectomy. One of these was unique in that a correct pre-operative diagnosis was made.

So far as can be ascertained, there is no authentic account in the literature of survival following bilateral renal venous thrombosis. Such a case is here recorded, with the notes of two fatal cases.

Case Records

Case 1. W.K., a full-term male infant, weight 7 lb. 4 oz., was delivered with forceps on March 24, 1948. He made good progress until the twelfth day, when he became drowsy, fed reluctantly, and developed diarrhoea. His condition deteriorated rapidly during the next four days, the diarrhoea increased, and all feeds were refused during the twenty-four hours before his admission to hospital on April 9, 1948. He was by now extremely collapsed, the temperature being 100° F., the pulse 160, and respiration 40 per minute. His weight had fallen to 5 lb. 7 oz.; the features were sunken; the colour was ash, and the extremities were cyanosed; the fontanelle was depressed; respirations were shallow and distressed. Pus discharged from a forceps wound over the right parietal region. No enlargement of the abdomen was detected. The child was put into an oxygen tent and intravenous administration of plasma and saline was begun. Penicillin was given intramuscularly at three-hourly intervals. Oral fluids were withheld during the first two days.

Progress. No urine was passed until eighteen hours after admission, when a few cubic centimetres of almost pure blood was collected. The respirations became laboured; both kidneys were now considerably enlarged, the left kidney more than the right. During the following forty-eight hours the condition deteriorated. The stools became watery and offensive, numbering between eight and ten a day. Gross haematuria persisted. The size of the kidneys by this time had increased even more and the abdomen had become distended. On the fourth day after admission the respiratory rate rose to 65 a minute and there was marked dyspnoea, presumably related to the uraemia, since the blood urea had reached the high level of 329 mg. per 100 ml.

By the seventh day the scalp infection had resolved and the urine was macroscopically clear. The blood urea level had fallen to 292 mg. per
100 ml. Weak breast milk feeds, an ounce at a time, were now tolerated. The diarrhoea improved gradually but the condition of the child remained critical until towards the end of the second week. Then definite improvement set in. He gained weight; the stools became normal, and the size of the kidneys decreased. The blood urea fell to 62 mg. per 100 ml. By the end of the third week the left kidney was still somewhat enlarged but the right was no longer palpable. The urine was now microscopically clear. He was discharged from hospital six weeks after admission in good general condition. The left kidney was still palpable. The urine was clear and the blood urea was 52 mg. per 100 ml. Weight on discharge was 7 lb. 4 oz.

His condition was reviewed at the age of four months. Intravenous pyelography showed a normal pelvic shadow with normal excretion on both sides. The blood urea level was then 41 mg. per 100 ml., and clinical examination was negative.

**Diagnosis.** Although post-mortem examination or laparotomy are the only means of establishing an absolute diagnosis, a clinical diagnosis of bilateral renal thrombosis may reasonably be accepted in this case on the following grounds:

1. The occurrence of frank haematuria in the absence of urinary infection. The urine remained sterile and free from pus throughout the acute phase of the illness. The haematuria persisted until the end of the third week in spite of adequate dosage with vitamin K at the onset. A diagnosis of haemorrhagic disease of the newborn could therefore be excluded. It has been shown that infants with renal haemorrhage resulting from birth injury invariably succumb within the first twenty-four hours of life (Cruikshank, 1930).

2. Gross enlargement of the kidneys with their subsequent return to normal size. This, along with intravenous pyelography at a later date, eliminated the possibility of a congenital condition.

3. The high degree of renal insufficiency indicated by the uraemia.

Two outstanding features of the case described are: first, the remarkably high blood urea level attained; secondly, the return of the kidneys to apparently normal function within a few weeks, following such gross enlargement with almost complete suppression of function. So complete was the recovery that it seems probable that both recanalization of the thrombi within the renal vein or its tributaries, together with the development of a collateral circulation from the extrarenal veins, were the factors responsible.

This case is unusual in that it had the presenting signs and symptoms of both groups described by Sandblom.

**Case 2.** A male infant was admitted to hospital on the twelfth day with a history of failure to gain weight since birth and gastro-enteritis of twenty-four hours’ duration. He was semi-comatose. The urine contained a moderate amount of albumin. The prothrombin time was 30 seconds (control 18 seconds). Death occurred three days later.

Autopsy revealed an enlarged right kidney which was grossly congested. The right renal vein was distended and contained a recent thrombus which filled the whole renal vein and extended into the inferior vena cava. The lungs showed patchy consolidation. Both middle ears contained pus.

**Case 3.** A female infant aged four weeks became fretful and sleepless on the day before admission. The stools were watery. The child succumbed the following day. Post-mortem examination showed a haemorrhagic infarction of the whole right kidney and of the lower pole of the left kidney. Recent thrombi were present in both main renal veins and in the longitudinal sinus.

**Case 4.** A prematurely born female infant was admitted to hospital on the first day of life. The birth weight was 2 lb. 7 oz. The general condition was feeble, and there was respiratory distress. Death occurred eighteen hours after delivery. At autopsy the lungs were found to be almost completely airless. There were numerous asphyxial subepicardial haemorrhages. The heart and great vessels were normal. The liver was grossly congested. Both kidneys showed areas of early haemorrhagic infarctions, 2 to 5 mm. in diameter. There were multiple subarachnoid haemorrhages over the cerebellum. The cerebral venous sinuses were normal.

**HISTOLOGY.** The medulla showed gross congestion but no necrosis. In the cortex there were areas of haemorrhagic infarction. In some places the renal parenchyma was completely replaced by red cells and numerous white blood cells. In other areas there was only disintegration of the tubular epithelium, the first convoluted tubules being more affected than the convoluted and Henle loops. The glomeruli were better preserved than the tubules (fig. 2). In several places thrombosis of the small veins was seen. Some were completely obstructed. In others a layer of hyalinized platelet thrombus was seen lining the wall of the vein (fig. 3).

**Etiology.**

Sepsis and dehydration were the probable precipitating factors in the first and second cases described. In the third case dehydration was the only cause of thrombosis. Focal infection has been shown to be a frequent accompaniment of renal thrombosis, and in Case 1 the scalp infection may well have given rise to bacteraemia or septicaemia. The occurrence of dehydration, in conjunction with the normal relatively low venous pressure in the newborn, predisposes to thrombosis and accounts in part for the high incidence of the condition during the neonatal period.

Case 4 is unusual in that both infection and dehydration were absent. The thrombosis in this instance was presumably caused by the asphyxiated
Fig. 1.—Thrombosis of renal vein in one of six cases discussed. ×120.

Fig. 2.—Haemorrhagic infarction of renal cortex in Case 4. ×120.
condition of the infant, which resulted in general venous congestion with stagnation. This etiology contrasts with cases hitherto described. Cruikshank (1930) in an analysis of eight hundred neonatal deaths, was unable to demonstrate any relationship between maturity, birth asphyxia, or marasmus and renal (as opposed to intracranial) thromboses.

Toxaemia per se has not been proved to play any significant part in the process.

**Treatment**

It is of interest that in Campbell’s two cases treated surgically the prothrombin level, pre-operatively, was well below the average normal low level at that period of life. He advocates the administration of vitamin K in such cases. In Case 2, referred earlier by the author, the prothrombin time was found, forty-eight hours before death, to be 30 seconds, a figure which may also be regarded as low, especially in view of the fact that 10 ml. of whole blood had been given intramuscularly on the day before admission to hospital.

As pointed out earlier, vitamin K did not influence the haematuria in Case 1 here reported. It is possible that the raising of the prothrombin level may, in fact, increase the risk of an extending thrombosis. It would seem reasonable, therefore, to avoid the use of vitamin K in any case of renal thrombosis which is to be treated medically, unless the child shows a general haemorrhagic tendency.

Since the diagnosis of renal thrombosis is usually retrospective, there has been no opportunity for observing the effect of the anti-coagulant drugs.

The place of surgery in the treatment of renal thrombosis requires further consideration. Marshall and Whapham (1936) describe a case conforming to Sandblom’s first group, in which the thrombosis is bilateral, and many similar cases are to be found in the literature. When a decision is made to remove a kidney in which thrombosis is suspected, a grave risk is being taken, since the possibility of a thrombotic process already having started in the other kidney is considerable. Unless there is a supplicative condition within the affected kidney it is difficult to foresee that nephrectomy, during the acute phase, will be of any benefit.

The criterion for surgery would appear to be, therefore, a heavily infected urine in a case of unilateral thrombosis.

The treatment of renal venous thrombosis may be summarized as follows:

1. Surgical treatment: nephrectomy is of value in certain selected cases of unilateral thrombosis.
2. Medical treatment: this should be directed towards (a) the relief of dehydration; (b) treatment of co-existing focal infection; (c) the prevention or treatment of urinary infection.

**Summary**

The literature concerning renal thrombosis is briefly reviewed.

A case of bilateral thrombosis with recovery is described in detail. Three fatal cases are described, including one of interlobular thrombosis due to neonatal asphyxia. The diagnosis, etiology, and treatment of the condition are discussed.

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**References**


**Fig. 3.**—Early thrombosis of a small renal vein in Case 4. × 480.