

A case of renal vein thrombosis with adrenal haemorrhage

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Introduction

Renal vein thrombosis (RVT) is the second most common thromboembolism in neonates next to umbilical vein thrombosis and accounts for 10% of neonatal venous thromboembolism with nearly half of them in preterm infants¹. We report a case of RVT with adrenal haemorrhage.

Case report

A term male newborn with an Apgar score of 9 at 1 and 5 minutes presented with jaundice after 6 hours of birth and haematuria, reduced activity and poor feeding since 1 day. There was no history of skin / mucosal bleeds, decreased urine output, fever or abdominal distension. There was no history of umbilical catheterization. Mother was a primigravida booked case with an uneventful antenatal period. Her blood group was O Rhesus negative without previous sensitization. There was no history of gestational diabetes, fever, pregnancy induced hypertension, drug ingestion, epilepsy, chronic illness and no history suggestive of systemic lupus erythematosus (SLE) or oligohydramnios. The baby was the 1st issue of non-consanguineous parents with no history of neonatal deaths, bleeding disorders, renal problems or chronic illness.

On examination, baby was comfortable, icteric, with stable vitals, a capillary filling time less than 3 seconds, a blood pressure of 73/55mm of Hg and a respiratory rate of 56/minute. Weight was 3.5kg. There were no obvious congenital anomalies. There was bilateral subconjunctival haemorrhage and the baby was not pale or oedematous. No petechiae or ecchymoses were seen. Umbilical stump was healthy. Abdomen was soft and lax and a mass was felt in the left loin, 4 x 4cm, firm, ballotable and bimanually palpable with no hepatosplenomegaly.

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The white blood cell count was 23,800/cu mm (polymorphs 76%), the haemoglobin 14.7g%, the platelet count 128,000/cu mm, reticulocyte count 3.6% and the haematocrit 38%. The prothrombin time was 13.5 seconds, the activated partial thromboplastin time (aPTT) 46.3 seconds, the internationalized normalized ratio (INR) 1.22, the bleeding time 2 minutes 09 seconds and the clotting time 5 minutes 05 seconds. The blood group was B Rhesus positive and the direct Coombs test was negative. Blood and urine cultures were negative. Clotting factor, protein C, protein S, Antithrombin III assay, liver enzymes and serum electrolytes were within normal limits. Lupus anticoagulant was negative. The serum bilirubin was 17.3mg/dl, direct 0.76mg%, blood urea was 21.84mg% and the serum creatinine 1.16mg%. Urine analysis showed plenty of red blood cells with no casts. Ultrasonography (USG) of the abdomen showed left renal vein thrombosis with left adrenal haemorrhage and patent inferior vena cava (Figure 1).

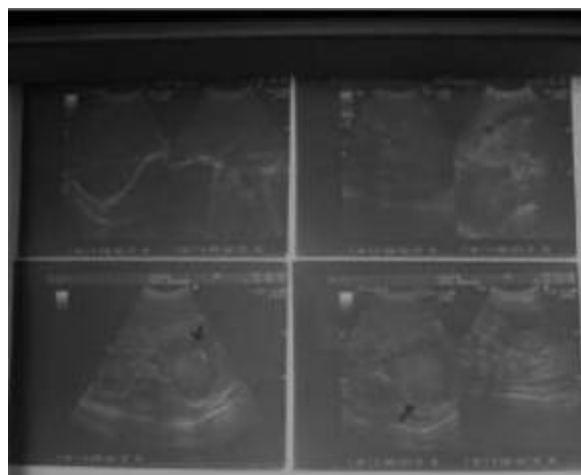


Figure 1: USG showing enlarged kidney with haemorrhage

USG of the brain was normal. DTPA scan showed that the glomerular filtration rate (GFR) on left was 2% and right 98%. The diagnosis was left renal vein thrombosis with left adrenal haemorrhage and neonatal jaundice. Baby was well hydrated, Phototherapy was given, antibiotics administered for

7 days, packed red blood cells & fresh frozen plasma given, Heparin 75units/kg loading with 25 units /kg/hour was titrated according to aPTT and after consultation with the hematologist was given for 5 days. Urine began to clear by 48 hours. Repeat USG showed improved blood flow in the renal vein. Follow up after 6 months DTPA scan showed improvement in GFR of the left kidney from 2% to 30%.

Discussion

RVT in newborn has been reported to occur in 0.5 per 1000 admissions to neonatal intensive care unit according to a large international registry². RVT is the commonest non catheter related thrombosis in infancy and neonates³. In neonates it is often spontaneous, secondary causes being asphyxia, hypotension, polycythaemia, dehydration, sepsis, maternal SLE, gestational diabetes, homocystinuria, central lines, umbilical vessel catheterization, cyanotic congenital heart disease, angiography, congenital deficiencies of anticoagulants - protein C, S, Antithrombin III, factor V Leiden mutation and prothrombin mutation^{4,5}. Haemoconcentration with hypotension leads to sludging in the intrarenal vessels, initiation of thrombus formation and extending to the large vessels⁶.

Ultrasound scan is a useful and convenient tool for the diagnosis of RVT⁷. Approach to an individual infant with RVT must balance risks and benefits. In nearly 40% of the newborns supportive care and close monitoring of the size of the thrombus will suffice. Use of heparin, preferably low molecular weight heparin (LMWH) and fibrinolytic agents for 6-12 weeks are recommended in bilateral RVT and extension into IVC⁸. Surgical thrombectomy is rarely done in newborns, owing to the small size of the blood vessels and clinical instability of newborns⁷. Perinatal mortality from RVT has decreased significantly over the past 20 years⁸.

RVT and adrenal hemorrhage are known to occur simultaneously in neonates^{9,10,11}. The pathogenesis of neonatal adrenal hemorrhage is unknown. Associated factors include birth trauma, asphyxia, sepsis, and coagulopathy¹². None of these factors were present in our patient.

In one study the authors present 4 cases of newborns affected by renal vein thrombosis associated with adrenal hemorrhage and caval thrombosis, evaluated by means of ultrasound¹³. In the present case no caval thrombosis was found.

Neonatal adrenal haemorrhage with RVT, an almost exclusively left-sided phenomenon, may occasionally be bilateral in the presence of inferior vena cava thrombus but has only twice been reported as confined to the right side¹⁴. In our patient both the neonatal haemorrhage and the renal vein thrombosis were left-sided.

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