Renal artery thrombosis: an exceptional etiology of arterial hypertension in new born

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ABSTRACT

Renal artery thrombosis is rare in new-borns. It is mostly related to umbilical arterial catheterization associated with thromboembolic risk factors. We report here both aortic and right renal artery thrombosis in a female premature new-born declared as an arterial hypertension. Diagnosis was confirmed by Doppler ultrasound. The progression was favourable with anticoagulation therapy. This condition is associated with a low mortality but long-term kidney dysfunction is common and patients require a long term follow up. To avoid arterial thrombosis, we need to rationalize the use of catheters and identify the risk factors for thrombosis.

INTRODUCTION

Neonatal thromboembolic disease is rare and severe. Artery thrombosis is less frequent than venous thrombosis and it rarely affects aorta. The incidence of symptomatic arterial thrombosis is reported to be approximately 0.25/10 000 live births (1–3). Almost 80% of aorta thrombosis is related to umbilical artery catheter (4). Mortality is heavy and renovascular or functional sequelae are usually severe, imposing urgent diagnosis and effective treatment. We rapport here an arterial renal thrombosis in a term newborn caused by an umbilical catheter.

CASE REPORT

Our patient was a female newborn. She was born to a 35 years old mother and from a pregnancy complicated by gestational diabetes. She was born at 34 weeks of gestation by an emergency caesarean section for acute pancreatitis stage E. At birth, the APGAR score was 1 and 3 at 1 and 5 minutes respectively with a 2350 g birthweight. She was resuscitated and then transferred to the neonatal intensive care unit for management of perinatal asphyxia and respiratory distress secondary to the perinatal asphyxia and a hyaline membrane disease. She required mechanical ventilation, placement of an umbilical catheter and the use of vasoactive drugs for hemodynamic disturbances. At 9 days of age, she presented an arterial hypertension. She developed an oligoanuric renal failure with a creatinine level at 54µmol/l without electrolytic disorders. A renal etiology was suspected, so a doppler ultrasound of the renal arteries was realized and showed a floating thrombus of the abdominal aorta at the level of the renal arteries extended to the origin of the right renal artery. Within these results a continuous infusion of heparin was begun. She received 7 days of Unfractionated Heparin (20UI/kg/h), then 3 weeks of Low Molecular Weight Heparin at a rate of 160 IU/kg/12h, then 15mg/day (5mg/kg/day) of acetylsalicylic acid. The patient was normotensive under 1mg/kg/day nicardipine. The control ultrasound, on day 5 of heparin therapy showed the absence of visualization of the thrombus in the abdominal aortic and renal artery previously described. The decision was an early withdrawal from treatment. In view of the re-ascension of the blood pressure figures during the evolution, an ultrasound control was indicated and showed a recurrence of the renal artery thrombus. She was then transferred to the pediatric nephrology department for management and further exploration of her high blood pressure. At the initial examination the infant was eutrophic for age. The neurological examination was without abnormalities. The cardiac auscultation did not show excessive noise. The pulse was present and symmetrical. Blood pressure was 80/50 mmHg for all four limbs. The urine sediment test was free of abnormalities, especially no hematuria or proteinuria. Heparinotherapy was resumed and then relayed an anticoagulant treatment based on anti-vitamin K adapted according to the level of INR (objective between 2 and 3). As part of the impact assessment of high blood pressure: the neurological examination was without abnormalities; the examination of the fundus was normal, cardiac echography showed no left ventricular hypertrophy

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or pulmonary arterial hypertension with retained FeVG. But the eGFR was decreased with a CKD stage 2, and the right kidney was hypoplastic on ultrasound and not functional on DMSA scan. In front of this thrombosis, an etiological balance was carried out. The thrombophilia workup found a normal level of antithrombin III at 106% and a decreased level of Protein C at 49% due to immaturity with normal rates for the parents. Levels of free protein S at 99.8% and fibrinogenemia at 2.42 were normal with respectively 99.8% et 2.42 g/l with no mutation of factor II or factor V Leiden. The neonatal lupus and anti-phospholipid syndrome were eliminated in front of no circulating anticoagulant antibodies for the infant and negatives antinuclear and anti-cardiolipin antibodies mother testing . Normal profile of hemoglobin electrophoresis (Hb A:82.7%, Hb A2:2.6%, Hb F:1.7%) allowed us to eliminate sickle cell disease. Given the negativity of the etiological work-up for thrombophilia in the patient and her mother, renal artery thrombosis was linked to acute foetal distress and umbilical catheterization. The anticoagulation therapy was stopped after 18 months. Renal ultrasound after 7 months showed no renal artery thrombus or other abdominal localization. As well as blood pressure and antihypertensive medication was stopped and renal function was normalized with creatinine clearance > 90/ml/mn/1.73 m2 SC. After one year, our patient was eutrophic with normal psychomotor development, normal blood pressure and normal renal function.

DISCUSSION

At birth, the components of the neonatal haemostatic system are similar to those in older children and adults, but plasma concentration and activity are significantly different. This system continues to develop during the first year of life to reach adult values. Actually, it is influenced by the age of gestation and postnatal age. Levels of vitamin k dependent clotting factors and inhibitors of coagulation are lower in preterm infants than term infants. This condition exposes them to a higher risk for developing bleeding or thrombotic complications in response to perinatal risk factors or iatrogenic events (5,6). This foetal neonatal risk factors include inherited thrombophilia, prematurity, polycythaemia, congenital heart disease, respiratory distress syndrome, sepsis and low birth weight. In case of prolonged umbilical catheterism the risk of thromboembolism event increases in association with these risk factors (7,8). In our case, prematurity, perinatal asphyxia, respiratory distress syndrome and umbilical catheterism are the main risk factors of the aorta and renal artery thrombosis.

Clinical features of renal vascular thrombosis are extremely variable. It varies from venous to artery thrombosis and it largely depend on the location and degree of involvement of each one. This presentation can vary from asymptomatic to life threatening conditions. While the majority of patients with renal venous thrombosis present with at least one of the three cardinal signs (macroscopic haematuria, large palpable kidney, thrombocytopenia), patients with renal artery thrombosis are usually asymptomatic and the diagnosis should be suspected whenever a newborn having risk factors presents with transient hypertension like our patient. Acute renal failure in this setting is usually rare, except in cases of aortic thrombus that extends to both renal arteries (8–10). Although our patient had a unilateral renal artery thrombosis, she presented with oligo-anuric renal failure. Perinatal asphyxia, associated with haemodynamic disorders alongside the thrombosis may explain this condition.

Once suspected renal vascular thrombosis should be confirmed by imaging. Contrast angiography is the gold standard for the diagnosis of vascular thrombosis but it exposes the newborn to radiation and contrast agent. The access to angiography is generally difficult in these population because they are usually severely ill. So, this exam is rarely used. The Doppler ultrasound is more accessible in neonatology units making it the most used method to confirm the diagnosis of arterial thrombosis in newborns (11).

Medication should be started as soon as the diagnosis is established. Therapeutic indications are not codified in neonates and they are based on extrapolation from adults. They are dependent on the extent of thrombosis, its location, the effectiveness of the circulation of supplement, existence of kidney failure, general condition of the newborn, the experience of the care team and the availability of the different treatments. The American College of Chest Physicians (ACCP) guidelines advocate that treatment (anticoagulation, thrombolytic therapy and surgery) should be individualized based on the extent of thrombosis and the urgency of the clinical situation(6,12). The benefit/risk ratio should be calculated before deciding whether or not to start anticoagulant therapy because of the major bleeding risk, especially in premature neonates and those at risk of intracerebral haemorrhage. Sometimes it is necessary to adopt a conservative approach (armed abstention). Thrombolysis is only indicated in cases of bilateral arterial thrombosis with acute renal failure. In all cases umbilical catheter should be removed. In addition to renal revascularization, attention should be paid to supportive care (the regulation of acid-base and electrolyte disorders) and anti-hypertensive therapy. Renal function monitoring is necessary and consideration should be given to when to indicate extra-renal purification in these patients. Current guidelines recommend continuous monitoring of the thrombus by US and continuing therapy until resolution of the clot for a total duration of between 6 weeks and 3 months (11). Although renal artery thrombosis in neonates is associated with low mortality, it is associated with high morbidity. In the long term, there may be progressive deterioration of renal function, hypertension and proteinuria, requiring long-term renal monitoring (7–9).

CONCLUSION

Although rare, neonatal arterial thrombosis is serious. The aetiology is dominated by arterial catheterization. The presence of sepsis, perinatal asphyxia or dehydration is an additional risk factor for the occurrence

of thrombosis, and an abnormality of homeostasis is rarely found. The diagnosis of thrombosis must be rapid as well as the management. The best treatment remains prevention which aims to identify the situations at risk and to limit the indications for catheterization.

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