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Case Report

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Neonatal arterial thrombosis at birth

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ABSTRACT

Neonatal thromboembolism occurs with various predisposition and triggers. Early diagnosis of the thrombosis is challenging and essential for therapeutic interventions. We herein report one newborn who presented with transient patient with arterial thrombosis underwent arterial Doppler and received low molecular weight heparin (LMWH) subcutaneously. Newborns suspected having arterial thrombosis may need urgent surgical intervention along with thrombolytic and anticoagulant therapy to prevent organ ischemia and amputation of extremities.

Keywords: Thromboembolism, Vascular anomaly, Heparin, Antithrombin

INTRODUCTION

Thromboembolism is a multifactorial disease involving genetic predispositions, underlying disorders, and acquired triggers. The established genetic risks for thrombosis beyond the race/ethnicity include protein C (PC), protein S (PS), and antithrombin (AT) deficiencies.[1] PC deficiency is a major critical thrombophilia of the newborns who presents with purpura fulminans, intracranial thromboembolism, and fetal hydrocephalous.[2,3] Newborn infants are at the highest risk of thrombosis throughout childhood even without having the inherited thrombophilias.[4,5] The incidence of embolism has been increasingly diagnosed in infants and children, as a complication of sepsis, cancer, congenital heart disease, therapy-related events of drugs, and intravenous catheters.[6] Recent advances in the neonatal medicine may raise the chance of developing thromboembolism.[7]

Early diagnosis of thromboembolism is challenging in the newborn because of the physiological increase of the natural anticoagulants and the difficulty of the imaging analysis to determine the vascular occlusions, although an outline of the management has been proposed.[8] Another concern is the limited use of the expensive PC agents for replacement therapy.[2]

CASE REPORT

A male infant was born at 34 weeks of gestation through a normal vaginal delivery from a primi mother. The pregnant course had been complicated with pregnancy induced hypertension, and the family history did not reveal any thrombotic and ischemic diseases. The male infant was small for gestational age (SFD) phenotype and the birth weight was 1025 gm. The Apgar scores were 9 and 9 points at 1 and 5 minutes, respectively. The umbilical cord and placenta did not demonstrate either ischemic or thrombotic lesions.

After birth, we noted the infant's left lower limb to be quite pale on the initial evaluation (Figure 1). No pulsations were detected in left femoral, popliteal, and posterior tibial arteries. Urgent Doppler ultrasonography was done which did not show flow in anterior tibial artery, posterior tibial artery and dorsalis pedis artery. The initial laboratory examination fibrinogen level 173, D-dimer level 0.684, prothrombin time (PT): 12.6, activated partial thromboplastin time (aPTT): 86.6, international normalized ratio (INR): 1.13, factor 8: 154.1%, factor 9: 76.6%, platelets: 185000. We there for speculated the occurrence of arterial thrombosis as a possibility of the ischemia and therefore administered low molecular heparin (LMWH) @1 mg/kg/day subcutaneously.

After that circulatory disturbance of affected side was recovered. The prothrombotic state thereafter gradually improved, and administration of LMWH @1.5 mg/kd/day subcutaneously given for 4 weeks. A slight decline in platelet count was observed after treatment. At the time the infant discharge from the hospital Doppler ultrasonography was normal flow in affected left lower limb. 2D echocardiography and cranial ultrasonography showed normal results. No inherited deficiency of PC, PS, and AT was determined.

Figure 1 showing left lower limb transient ischemia in neonate immediately after birth due to complication of circulatory disturbance due to arterial thrombosis.

Figure 2 shows image of neonate at time of discharge showing improvement in left lower limb circulation.

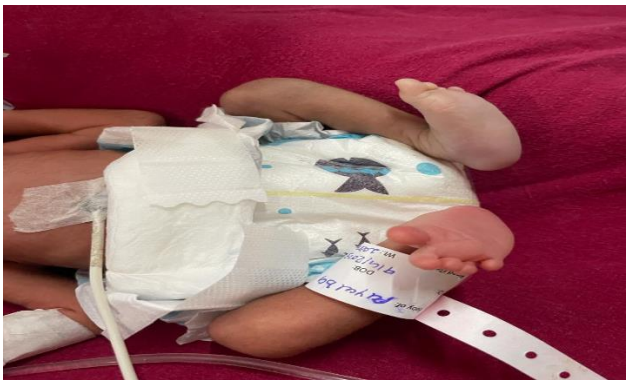

Figure 1: Left lower limb transient ischemia in neonate immediately after birth due to complication of circulatory disturbance due to arterial thrombosis.

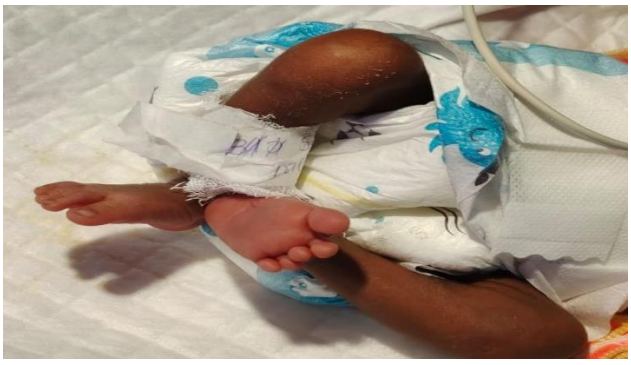

Figure 2: Neonate at time of discharge showing improvement in left lower limb circulation.

DISCUSSION

Neonatal arterial thrombosis occurs due to changes in prothrombotic tendency; increase procoagulant effect (prothrombin gene mutation, elevated factor 8, hyperhomocysteinemia) or decrease anticoagulant effect (factor 5 leiden mutation, deficiency of protein C and S or antithrombin 3).

Risk factors are pre term babies (decrease level of antithrombin 3), birth asphyxia, polycythemia, CHDs, IDM.

Clinical sign

Thrombosis of peripheral artery: decrease perfusion, pallor, reduced pulsation, ischemic skin changes.

Aortic thrombosis: reduced pulsation and perfusion of lower extremities, increase differential in BP in upper limb and lower limb, hypertension, oliguria, and CHF.

Diagnosis is done by Doppler colour flow (non-invasive) and radiographic contrast study (confirmatory).

Treatment

Heparin: loading dose – 75 units/kg over 10 min, continue infusion – 25 units/kg/hour, LMWH: 2 mg/kg/dose subcutaneously every 12 hourly, urokinase can be given, fresh frozen plasma (FFP) @ 10 ml/kg, tissue plasminogen activator (tPA): treatment of choice due to reduced risk of allergic reactions and short $t_{1/2}$.

If life threatening bleeding due to thrombolytic therapy give; cryoprecipitate (1 unit/5 kg) or amino caproic acid (100 mg/kg IV every 6 hourly). Surgical thrombectomy is avoided due to high risk of mortality.

The primary goal of early intervention for the neonatal thrombosis in those guidelines was to prevent to prevent organ ischemia and amputation of extremities. Unfortunately, few randomized clinical trials have addressed the management of neonatal thromboembolism emergencies. A prompt diagnosis is quite essential to achieve an improvement in such patients, and the successful therapeutic strategies have been reported to be the intravenous administration of high-dose urokinase, low-molecular-weight heparin, and the combination of unfractionated heparin and AT concentrate.[9,11,12] Expectant management is a reasonable alternative considering that recommendations and dosing regimens for anticoagulant/thrombolytic therapy in neonates are based on those reports. Meanwhile, the potential for serious complications (intracranial hemorrhage) must be considered in any neonate before initiating antithrombotic therapy.[10]

CONCLUSION

We reviewed the infants who exhibited ischemia of the lower limbs immediately after birth. Newborns suspected of having arterial thrombosis may need a prompt diagnosis and proper intervention to prevent organ ischemia and amputation of extremities.

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