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Acute Congenital Non-Traumatic Ischaemia of the Lower Limb and Subrenal Ischaemia Secondary to Congenital Thrombophilia in a Newborn: A Case Report and Review of the Literature

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Abstract

Acute ischaemia (AI) is an extreme medical and surgical emergency. It is rare in newborns but also extremely serious due to its functional and life-threatening complications. Diagnosis can sometimes be difficult, which is why this case is being presented. This is a newborn hospitalised in the paediatric ward of La Paix Hospital in Ziguinchor for lower limb and subrenal ischaemia extending to the common iliac arteries. On admission, the examination revealed coldness in the left foot with a painful, flat, monomorphic purpuric area on the anterior-external aspect of the left leg, the lower third of the leg, the back of the foot, and the 2nd, 3rd, and 4th toes. There was also no pedal pulse, normal temperature and cyanosis of the left foot. The rest of the clinical examination was unremarkable. The Doppler ultrasound performed on admission was normal. The CT angiogram performed after five days of hospitalisation showed total thrombosis of the subrenal aorta extending to the common iliac arteries and complete thrombosis of the distal two-thirds of the leg arteries extending to the foot. Laboratory tests showed hyperleukocytosis at 15,490 (neutrophils: 67.4%), negative CRP, thrombocytopenia at 147,000, and normal renal function. Blood count was normal. Cardiac ultrasound was normal. Protein C levels returned to 27.52% functional activity and protein S to 35% functional activity. We believe this is likely to be a congenital thrombophilia. However, a

follow-up assessment is required to confirm the congenital origin of the deficiency. Treatment consisted of two injections of 100 units/kg of low molecular weight heparin combined with 0.1 mg/kg of acenocoumarol. The condition progressed to dry necrosis of the affected area, with dry, hard skin and a cardboard-like appearance forming a well-defined black patch. Secondarily, he presented with loss of substance, exposing the bones and tendons. An amputation was performed on day 20 of hospitalisation. The postoperative course was uneventful. Acute ischaemia in newborns is still a rare condition and constitutes a medical and surgical emergency. Symptoms remain important in diagnosis, but imaging is crucial. Conservative treatment is recommended, but in certain situations, minimally invasive treatment or amputation must be performed.

Keywords

Newborn, Ischaemia, Thrombophilia, Ziguinchor

1. Introduction

Acute ischaemia (AI) is an extreme medical and surgical emergency. It is rare in newborns but also extremely serious due to its functional and life-threatening complications. AI is a potentially devastating condition in children. Its aetiopathogenesis is complex and often poorly understood, but current knowledge of constitutional and acquired haemostasis abnormalities can explain certain vascular thromboses that occur during foetal and neonatal life.

The aetiology is dominated by arterial trauma related to catheterisation [1]. Congenital thrombophilia remains very rare in the literature. Few studies have been conducted on this subject [1] [2].

The rarity of acute congenital ischaemia makes this case unique.

We report a case of acute congenital non-traumatic renal and lower limb ischaemia in a newborn with congenital thrombophilia and discuss the diagnostic, therapeutic and evolutionary aspects through a review of the literature.

2. Patient and Observations

This is a male newborn, born to a 31-year-old mother, one pregnancy (IV), one parity (IV), pregnancy monitored with six prenatal consultations, complete tetanus vaccination, supplemented with iron and folic acid, negative Emmel test, negative serology, an ultrasound scan performed on 16/04/2025 showing a progressive intrauterine monofetal pregnancy of 20 weeks of amenorrhoea (SA) + 1 day. She did not present any particular pathologies during pregnancy and did not take any specific treatments other than iron supplementation and intermittent preventive treatment against malaria.

The delivery took place at 39 weeks gestation via vaginal birth, cephalic presentation, immediate cry without resuscitation, APGAR score of 6 then 8, birth weight:

2900 g, height: 51 cm, head circumference: 33 cm, and essential care provided.

From birth, he had a reddish mark on his foot, which was mistakenly considered to be a benign birthmark. The next day, his mother noticed that the reddish mark had spread and that he had a fever, prompting her to take him to the maternity ward, where he was given paracetamol syrup. The fever persisted until the fifth day of life, with worsening of the lesion on the lower third of the leg, prompting a visit to another health centre, which referred him for better care.

On admission, the examination revealed coldness in the left foot with a painful, flat, monomorphic purpuric area on the anterior-external aspect of the left leg, the lower third of the leg, the back of the foot, and the 2nd, 3rd, and 4th toes. There was also no pedal pulse, normal temperature and cyanosis of the left foot **(**Figure 1 and Figure 2**).** The rest of the clinical examination was unremarkable.

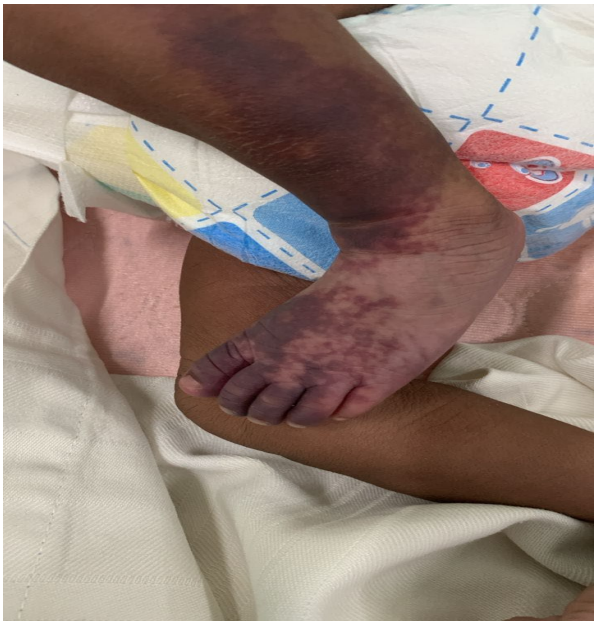

Figure 1. Appearance of the foot upon admission.

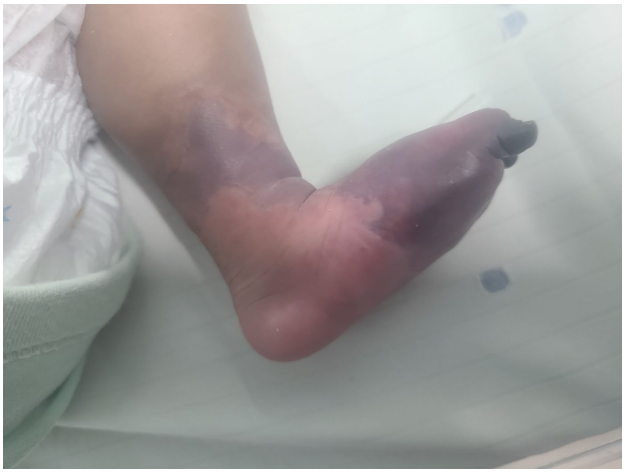

Figure 2. appearance of the foot after 5 days of hospitalization.

The Doppler ultrasound performed on admission was normal, probably due to the early stage of the disease. The CT angiogram performed after five days of hospitalisation showed total thrombosis of the subrenal aorta extending to the common iliac arteries and complete thrombosis of the distal two-thirds of the leg arteries extending to the foot (**Figure 3** and **Figure 4**). Laboratory tests showed hyperleukocytosis at 15,490 (polymorphonuclear neutrophils: 67.4%), negative CRP, thrombocytopenia at 147,000, and normal renal function. Blood count was normal with a prothrombin level of 74%, an international normalised ratio of 1.21 and an activated partial thromboplastin time of 15.3. Cardiac ultrasound was normal.

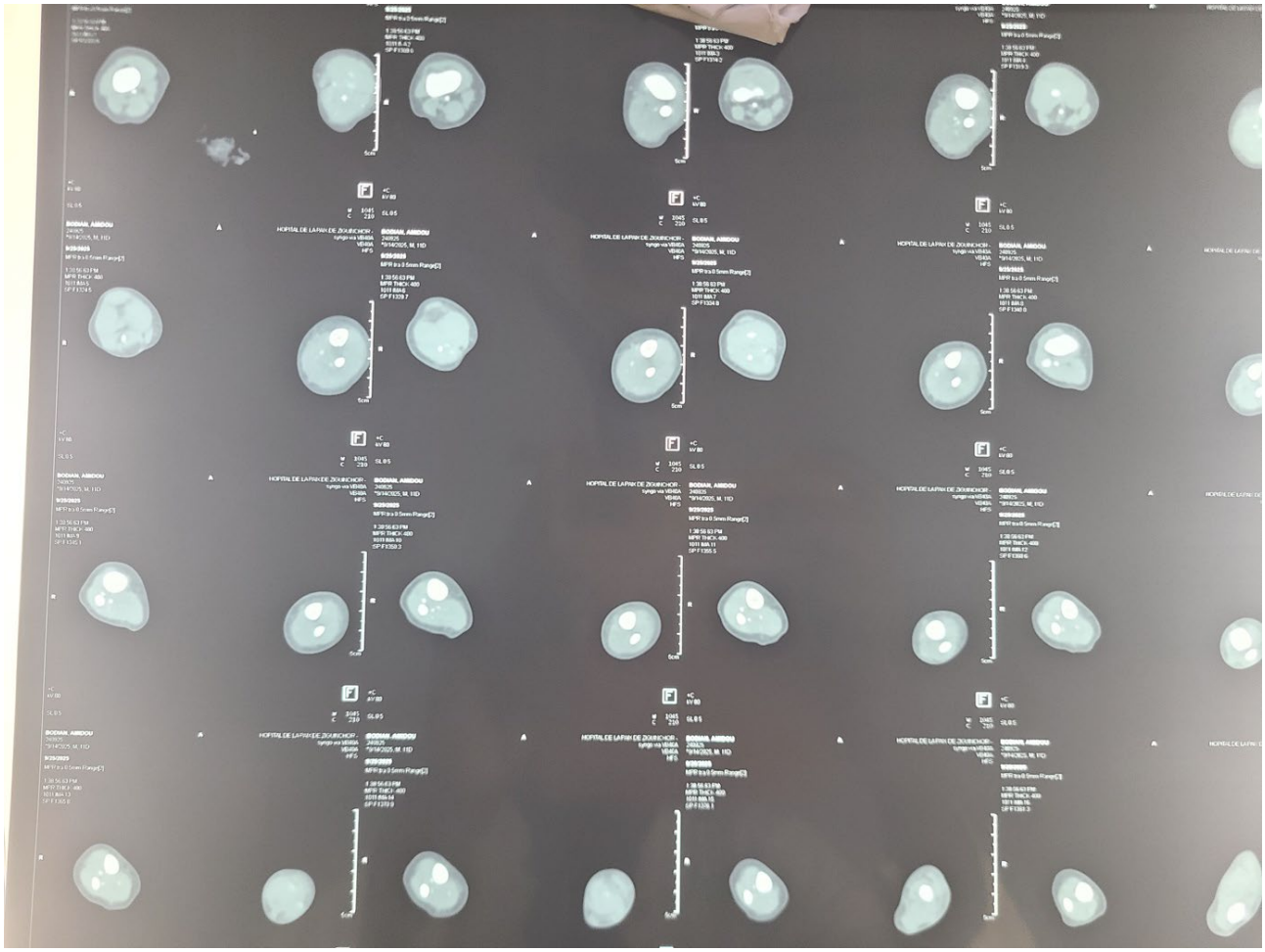

Figure 3. Complete thrombosis of the distal two-thirds of the leg axes extending to the foot.

Protein C levels returned to 27.52% functional activity and protein S to 35% functional activity. We believe this is likely to be a congenital thrombophilia. However, a follow-up assessment is planned to confirm the congenital origin of this deficiency.

Treatment consisted of two subcutaneous injections of 100 units/kg of low molecular weight heparin per dose, combined with 0.1 mg/kg of acenocoumarol.

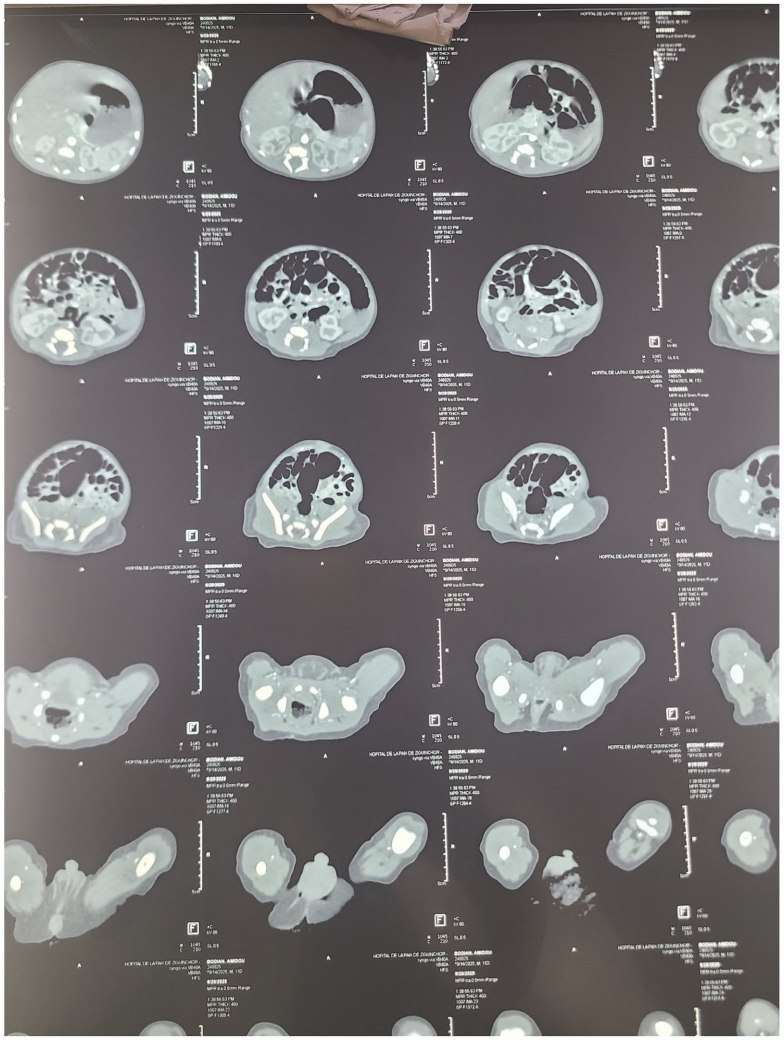

Figure 4. Total thrombosis of the subrenal aorta extending to the common iliac arteries.

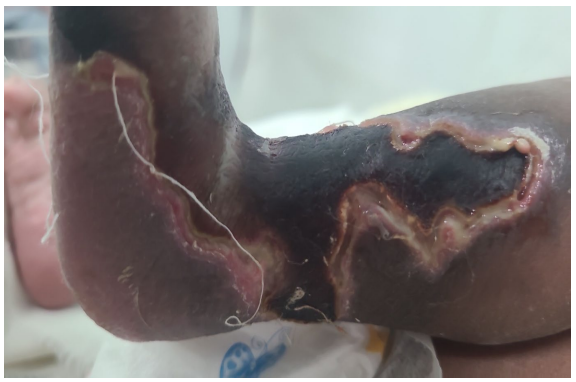

Figure 5. Appearance of the foot during hospitalisation (day 12 of treatment).

The progression is marked by dry necrosis of the affected area with dry, hard skin and a cardboard-like appearance forming a black, well-defined plaque **(**Figure 5 and Figure 6**).** Antibiotic therapy with cefotaxime at 200 mg/kg/day was added.

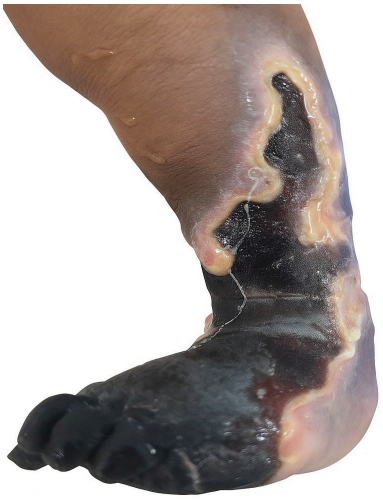

Figure 6. Appearance of the foot (dry necrosis) during hospitalisation (day 14 of treatment).

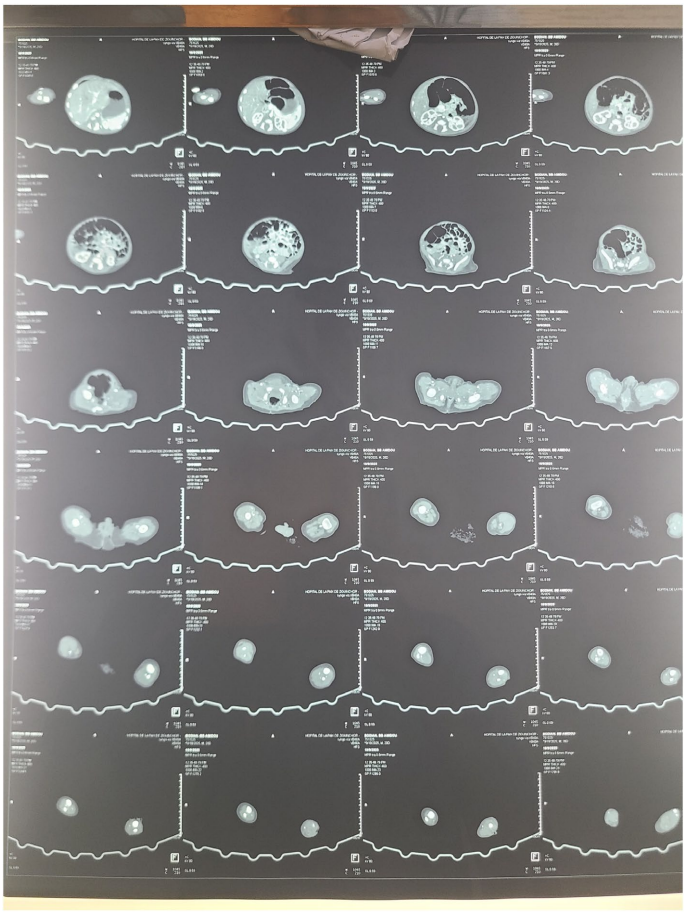

Figure 7. Follow-up angiogram showing reperfusion of the infrarenal aorta, common iliac arteries, and left anterior tibial and pedal arteries. The left posterior tibial and fibular arteries remain thrombosed in their distal two-thirds.

The follow-up angiogram performed on day 15 of treatment showed repermea-

bilisation of the subrenal aorta, common iliac arteries and left anterior tibial and pedial arteries (Figure 7). The left posterior tibial and fibular arteries are still thrombosed in their distal two-thirds.

Secondarily, he presented with tissue loss, exposing the bones and tendons (Figure 8). An amputation was performed on day 20 of hospitalisation. The postoperative course was uneventful.

Figure 8. Appearance of the foot with loss of tissue and exposure of bone.

3. Discussion

Due to the rarity of this condition, the literature is limited to clinical cases [2]. Kayssi's study in Canada [1], covering a period of 13 years and involving 151 cases of acute limb ischaemia, found 14 cases (9%) of non-traumatic ischaemia and 57 (38%) aged less than 30 days. The diagnosis remains clinical, and imaging is used to confirm the diagnosis and possibly monitor the patient. These are generally postnatal thromboses, which are iatrogenic in almost 90% of cases due to the increasingly frequent use of intravascular catheters. More rarely, the thrombosis is congenital with antenatal development of ischaemia [3].

Non-traumatic aetiological factors vary depending on age group. In the neonatal period, the most common causes remain genetic abnormalities and problems during pregnancy.

Hakim *et al.* [4] reported two cases of intrauterine arterial thrombosis of the upper limbs linked to heterozygosity for the 677C/T polymorphism of methylene tetrahydrofolate reductase and one case of intrauterine ischaemia of the upper

limbs linked to foetal thrombophilia due to a mutation in the factor V Leiden gene [5].

Doppler ultrasound is often sufficient, but in some cases, arteriography or CT angiography may be required [3]. In our patient, there was strong clinical suspicion, but the Doppler ultrasound performed on admission was normal. This could be related to the early diagnosis but also to the initial distal involvement.

The CT angiogram enabled the diagnosis to be corrected on the fifth day of hospitalisation. Kayssi in Canada [1] found involvement of the lower limbs in 142 patients (94%) and involvement of the common iliac artery in 12 cases (8%). Involvement of the subrenal artery in newborns has not been described in the literature.

Cases of acute idiopathic ischaemia have been described in the literature [1]. Similarly, infections in the neonatal period have been reported, such as Shivani [2], with group A beta-haemolytic streptococcus as the causative agent. In our patient, we diagnosed congenital thrombophilia.

Other aetiologies are sometimes described, certain medications such as indomethacin during pregnancy by Arad *et al.* [6], and hypernatraemic dehydration by Shivani *et al.* in 2011 [7]. In older children, other causes include vasculitis, antiphospholipid syndrome, and a mutation in the prothrombin gene [8].

For the paediatric population, there is still no consensus or protocols on management. Medical treatment is based on the administration of low molecular weight heparin or unfractionated heparin combined with an anti-vitamin K or antiplatelet agent, depending on the clinical picture. Regular monitoring of prothrombin time and international normalised ratio is carried out to verify the effectiveness of the treatment. Conservative treatment should always be used, as children can rapidly develop arterial collaterals, thus increasingly favouring a non-invasive approach as a first-line treatment [1] [3]. Treatment with low molecular weight heparin combined with vitamin K antagonists was initiated in our patient after 5 days of hospitalisation. However, a therapeutic window was observed at the time of surgery to reduce the risk of bleeding.

If this fails, minimally invasive treatment or surgery may be required, consisting of embolectomy or bypass surgery. With early diagnosis and prompt treatment, a good outcome can be achieved [9]-[11]. Otherwise, there is a high risk of amputation. In our case, the outcome was marked by necrosis followed by loss of tissue, exposing the bone and tendons. An amputation was performed. The postoperative course was uneventful.

4. Conclusion

Acute ischaemia in newborns is still a rare condition and constitutes a medical and surgical emergency. Its aetiology remains unclear, with various possible causes. Symptoms remain important in diagnosis, but imaging is crucial. To date, there is still no consensus on treatment. Treatment with unfractionated or low molecular weight heparins should always be initiated with a vitamin K antagonist or a

platelet antiaggregant. Conservative treatment is recommended, but in certain situations, minimally invasive treatment or amputation may be necessary.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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