

Case Reports

A neonate with quadruple cephalhaematoma, anaemia and heart failure

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Case report

A 10 day old male neonate was admitted in a collapsed state to a base hospital. He was gasping on admission and was immediately intubated and ventilated. He was found to be very pale with several lumps on the head. An urgent blood transfusion (45 ml) was given without performing any tests. Physical abuse was suspected. A frantic call was received by the paediatrician close to midnight for a ventilator bed in the neonatal intensive care unit (NICU) and a bed was arranged. He was immediately transferred to the NICU Teaching Hospital Karapitiya (THK).

The child was born on 6/3/2006 at the same base hospital. He was discharged home on 7/3/2006 following a normal routine examination after the BCG and vitamin K injections. Birth weight was 2.8 kg. He was exclusively breast fed and was apparently well till 14/3/2006. However, on inquiry, breast feeding had been poor after going home. On 14/3/2006 the child had poor feeding, breathing difficulty, grunting and reduced urine output. He was taken to the base hospital.

On admission to the NICU at 01.20 am he was still very pale with very poor peripheral circulation. Capillary refill time was 5 seconds. Peripheries were cold and clammy with central and peripheral cyanosis. Heart rate was 160/minute with a triple rhythm. All peripheral pulses were weak. Blood pressure was 40/20 mmHg. There was a soft systolic murmur which was thought to be functional.

There were four large lumps on the head (figure 1). These were cystic in consistency on both parietal regions and both temporal regions. Lumps were limited in extent to the suture lines of these bones. These were considered cephalhaematoma. There were no other lumps elsewhere. There were no

bruises or bleeding manifestations on the body. Anterior fontanelle was normal. No lymphadenopathy was present. There was tender hepatomegaly of 4 cm but no splenomegaly.



Figure 1 Biparietal & bitemporal cephalhaematoma

A provisional diagnosis of heart failure due to severe anaemia following bleeding into multiple cephalhaematoma was made and treatment commenced accordingly. His peripheral circulation was so poor that intravenous access was difficult. Therefore umbilical artery and venous cannulations were done.

A second blood transfusion of 45 ml was given. Ventilation was continued with 85% inspired oxygen. Dopamine infusion was commenced. A septic screen was performed. Empiric antibiotic therapy was commenced with ampicillin and cefotaxime.

He was also commenced on captopril, digoxin and frusemide. Vitamin K1 injection was given. Random blood sugar, serum calcium, serum electrolytes, serum magnesium, liver functions tests, blood urea, bleeding time and clotting time were normal. Initial APTT and PT were prolonged. These returned to normal subsequently. First recorded haemoglobin (Hb) was 16g/dl after 2 blood transfusions.(90 ml=32

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ml/kg). Platelet count was $90 \times 10^9/L$. WBC was normal. Electrocardiograph was within normal limits for the age. The chest radiograph showed cardiomegaly.

His oxygen requirement came down gradually and ventilator support was reduced. He was extubated in 61 hours. The triple rhythm persisted with the systolic murmur. Rest of the cardiovascular examination was normal. The oxygen saturation was normal in room air after extubation. He was transferred out of the NICU. The child started breast feeding slowly and it took some time for him to gain weight on exclusive breast feeding. Weight on discharge was 2.9 kg on day 31 and the first recorded weight after extubation was 2.8 kg.

Blood film and reticulocyte counts were normal. The septic screen was negative. In view of the systolic murmur and heart failure, an echocardiography was performed. This showed pulmonary stenosis, ventricular septal defect, right ventricular hypertrophy and overriding of the aorta compatible with Tetralogy of Fallot. This was confirmed by a second echocardiography. The child was discharged three weeks later after registering for cardiac surgery to be followed up at the base hospital paediatric clinic.

Discussion

Cephalhaematomata are known to be benign in a large majority¹. It is also known to cause several complications. Haemorrhage causing hypotension, anaemia and shock occurring soon after birth has been reported with large cephalhaematomata¹. Other complications include infections, abscess formation and exaggeration of physiological jaundice². Linear fractures underlying cephalhaematomata occurred in 5% of cases in a large series³. Linear fractures can be complicated with osteomyelitis and rarely meningitis³. These complications occur a few days or weeks later.

Slow bleeding leading to anaemia from any cause could cause heart failure. In this child the diagnosis of heart failure was never in doubt because of the clinical features of dyspnoea, weak pulses, triple rhythm tachycardia, hypotension, poor peripheral circulation, and hepatomegaly.

Anaemia was never confirmed by a low haemoglobin record at the THK. Hb was done after two blood transfusions (90 ml) at the two hospitals due to the urgency of the situation. So the Hb would have been much lower than 16g/dl. No other cause was found for anaemia as there was no evidence of haemolysis or bone marrow hypoplasia. Therefore, the only

possibility for the cause of anaemia is bleeding into multiple cephalhaematomata.

Significant bleeding leading to anaemia, hypotension and shock would be detected in the acute stage. Bleeding not large enough to cause hypotension and shock could be easily missed at the routine examination within the first 24 hours of birth like in cephalhaematoma. It is known that cephalhaematomata start bleeding at or soon after birth and continue to bleed for about 12 hours². These are initially soft and fluctuant. Although not reported, there is nothing to prevent these presenting later with heart failure if anaemia was severe enough. Anaemia may have been aggravated because the Hb drops gradually after birth and that may have resulted in the decompensation which led to the development of heart failure toward day 10. An argument could be brought for Tetralogy of Fallot (TOF) to be cause of the heart failure in this infant but it is well known that TOF does not cause heart failure and often does not present in the neonatal period with problems related to the heart. In fact this child was weaned off the ventilator after correcting the anaemia easily and was maintaining normal oxygen saturations in room air. Thereafter his digoxin and frusemide were stopped. This could not have been possible if heart failure was due to congenital heart disease. It also cannot be a duct dependent pulmonary stenosis (critical pulmonary stenosis) because child did not need prostaglandin or intervention in that direction to recover.

Therefore the most likely cause of heart failure in this neonate was anaemia due to bleeding into multiple (four) cephalhaematomata. This is the first case of quadruple cephalhaematomata I have come across in my clinical experience. A literature research did not find any reported cases. The other unusual feature of this case is that two of these cephalhaematomata were on the two temporal bones. These are unusual sites for cephalhaematomata, the commonest being parietal and less commonly the occipital bone. Importance of careful neonatal examination to detect soft cephalhaematomata in the first few days is stressed as it is not always benign.

References

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