Bilateral central retinal vein occlusion in a neonate secondary to atrial septal defect and patent ductus arteriosus

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DESCRIPTION

A 15-day-old late preterm male baby was admitted in the neonatal intensive care unit (NICU). He was delivered at 36 weeks of gestation by normal vaginal delivery and weighed 980 g at birth. He was admitted in the NICU due to severe intrauterine growth restriction (IUGR) and had multiple comorbidities like very low birth weight, birth asphyxia, respiratory distress syndrome, atrial septal defect (ASD) with patent ductus arteriosus (PDA), abdominal distension due to ascites and thrombocytopenia (78 000/mm³). Ophthalmic examination was requested as a part of the protocol for IUGR evaluation. The anterior segment evaluation of both eyes was unremarkable with well-dilating pupils to topical mydriatic (tropicamide 1% and phenylephrine 2.5%). Lens was clear in both eyes. Fundus examination revealed the presence of dilated and tortuous veins in both eyes with extensive intraretinal flame-shaped and dot haemorrhages at the posterior pole and in all the four quadrants (figure 1). The foveal reflex was dull and the contour seemed elevated on indirect ophthalmoscopy. A diagnosis of central retinal vein occlusion (CRVO) with macular oedema in both eyes was made. Both eyes underwent intravitreal bevacizumab one-third dose of adult dose (0.4 mg/0.015 mL) under aseptic condition in the presence of a neonatologist in the NICU. At postinjection day 3, the haemorrhages and venous tortuosity were on reducing course and a favourable response was noted. However, the baby succumbed to the comorbidities a week later due to which the subsequent follow-up was not possible.

Infants with congenital heart diseases (CHDs) like septal defects, PDA, pulmonary stenosis, coarctation of aorta, transposition of great vessels and cyanotic heart diseases are at a higher risk of developing retinal vascular occlusion and retinal haemorrhages.1–5 These CHDs can cause volume overload with alteration in hematocrit values, hypoxia due to the admixing of arteriovenous blood and also cardiovascular stress due to the obstruction imposed on the blood flow.1–5 In fetal circulation, ductus arteriosus is an essential communication between pulmonary artery and aorta, which closes spontaneously within 48 hours of birth.4 5 In full-term babies soon after birth, the rise in arterial oxygen tension and reduction in circulating prostaglandins results in the constriction of the ductus arteriosus.4 5 Failure of closure of ductus arteriosus is more common in preterm newborns, and this results in volume overload on the pulmonary blood flow.4 5 CRVO in newborns has been previously reported due to thromboembolic episodes due to deranged haematological parameters like protein C deficiency and hyperhomocysteinemia, postlaser photocoagulation for retinopathy of prematurity, CHDs and intraocular haemorrhage.4 6–9 In our case, we could not evaluate other haematological parameters before the death of the baby although thrombocytopenia was present. We believe the severe volume overload attributed to the ASD and PDA is the primary cause for bilateral CRVO. To the best of our knowledge, there is no case report on bilateral CRVO secondary to ASD and PDA in newborns. This report sends a message regarding the importance of fundus examination in newborns with CHD irrespective of gestational age of delivery.

Learning points

- Newborns with congenital heart disease (CHD) like atrial septal defect and patent ductus arteriosus are at risk of developing central retinal vein occlusion.
- Fundus examination in newborns with CHD is important irrespective of their gestational age at delivery.
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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

REFERENCES