their suffering; but, a call to the research community to generate evidence so that any potential benefits are not discredited. The science must provide evidence to dispel myths and focus efforts on evidence-based management of dengue.

**NEETU SHARMA AND *DEVENDRA MISHRA*

*Departments of Pediatrics, GR Medical College, Gwalior, and *Maulana Azad Medical College, Delhi, India.*

drneeetuagarwal@gmail.com

**REFERENCES**


---

**Reversible Corneal Clouding in Neonatal Hyperglycemia**

Corneal clouding, a rare observation in neonates, has been linked to causes such as infections and metabolic disorders. We describe a case of an extreme preterm with bilateral, reversible corneal clouding – possibly due to hyperglycemia.

This preterm (25 weeks) male infant was born vaginally to a primigravida mother; birth weight was 840 g. The child was managed with surfactant, ventilation, parenteral nutrition, cefotaxime and amikacin. On 4th day, baby developed seizures requiring phenobarbitone. Neurosonogram revealed grade-I germinal matrix hemorrhage.

On 6th day, infant developed hyperglycemia, which was managed by insulin infusion. Hyperglycemia lasted for 22 hours (highest blood glucose: 890 mg/dL). Antibiotics were changed to vancomycin and meropenem. Child also developed bilateral diffuse corneal clouding suggestive of corneal edema. There was no conjunctival or circumculari congestion or eye discharge. View to the anterior chamber and fundus was hazy; a red glow was present. Iris details could not be visualized. Cornea became clearer in the next few days. Mild haze persisted till 28 days and cleared completely by 39 days. The infant was diagnosed to have retinopathy of prematurity (ROP) at 28 days which progressed bilaterally requiring laser treatment at 41 days. At discharge (81st day) and at 6 months, the cornea remained clear.

Opacification of the cornea in a newborn may occur in congenital glaucoma, corneal dystrophies, Peter’s anomaly, sclerocornea, infection, trauma, limbal dermoids and metabolic disorders like mucopolysaccharidoses [1]. The case presented had no signs of ophthalmic infection or dysmorphism. Corneal clouding was reversible unlike in mucopolysaccharidoses. ROP is an unlikely cause since cornea had cleared when ROP had progressed. Drugs given to this infant are also not known to cause corneal edema.

In this infant, severe hyperglycemia could have caused endothelial dysfunction and corneal edema. The Na,K-ATPase in the basolateral membrane of the cornea, is responsible for the pump function [2]. Animal studies show that hyperglycemia reduces Na,K-ATPase activity of cornea [3,4]. In adult humans, acute hyperglycemia affects corneal hydration control [5]. Another mechanism could be hydration of lens, leading to angle closure and rise in intra-ocular pressure resulting in corneal edema. However, digital tonometry was normal in this infant.

We could not find any reported case of corneal clouding in neonates due to hyperglycemia. Although 60-80 percent of extreme preterm develop hyperglycemia, very high levels as observed in this case are rare. We hypothesize that severe hyperglycemia may have an
adverse effect on the cornea of extreme preterms. With early optimization of blood glucose levels, the corneal prognosis appears to be good.

Acknowledgement: Dr Maria Sajini Durairaj, Department of Ophthalmology, CSI Kalyani Multi Specialty Hospital, Chennai, Tamil Nadu, India.

S Sreedhar Raju and Betty Chacko
Department of Pediatrics, CSI Kalyani Multi Specialty Hospital, Chennai, Tamil Nadu, India.
bettychacko@gmail.com

REFERENCES