A 7-month-old girl presented with history of fever, on and off, since 4 months and irritability since 8 days. She had received treatment for the same, records of which were not available. Birth history was unremarkable. Vaccination was adequate for age. There was no neurological deficit; however, neck stiffness was present. The rest of the clinical examination was normal. She was referred for a transcranial USG with a clinical diagnosis of meningitis. Transcranial USG revealed echogenic stripes in both the basal ganglia regions (Figure 1). These echogenic stripes were periarterial in distribution as evidenced by their branching nature and the presence of arterial signals on colour Doppler examination (Figure 2). Velocity within these branches was within normal limits. The rest of the examination was unremarkable. Cerebrospinal fluid (CSF) study and the haemogram were within normal limits. The patient also underwent a computed tomographic (CT) scan of the brain, which was normal. Serological study for congenital infections (TORCH) was positive for Cytomegalovirus (CMV) IgG antibodies indicative of past or recent infection (1:32). Titre for IgM antibodies indicative of current infection was equivocal (1:16). Since the titres were not significant for active infection and also since there were no overt clinical manifestations, no specific therapy was instituted for CMV infection. A clinical diagnosis of partially treated pyogenic meningitis was made and she received antibiotics and antipyretics for 10 days. Her course in the ward was uneventful. At discharge she was completely asymptomatic. At 6 months follow-up the patient had normal neurological development and had attained all her milestones for that age.

Discussion

The detection of echogenic stripes in the basal ganglia and its significance in the diagnostic context have changed considerably since it was first reported by Grant et al in 1985.1 So much so that there has been a proposed change in nomenclature from mineralising vasculopathy to lenticulostriate vasculopathy (LSV) as the neuropathological correlation of mineralisation has not been proven in all cases. We try to determine the significance of the detection of LSV in our case in the light of the current literature.

Normally, on transcranial sonograms of neonates, it is unusual to detect the branching arteries that supply the basal ganglia...
and thalami. Typically, they are inconspicuous within the grey matter or indistinguishable from it.\(^2\)

Several causes of these echogenic stripes in the basal ganglia have been reported in the literature. Amongst the various causes are CMV infection, Rubella, congenital syphilis, Herpes, Trisomy 13, Trisomy 21, corpus callosal agenesis, exposure to drugs, perinatal asphyxia, streptococcal meningitis and many others.\(^2\) Basophilic deposits (mineralisation) in arterial walls were thought to be the likely reason for the echogenicity of these vessels on sonography. Prussian blue positive stain for iron has been demonstrated in a neonate having CMV infection.\(^2\) In the early weeks of foetal development, the germinal matrix regions near the caudothalamic grooves are active in rapid cellular mitosis. Because of the active proliferation occurring in these regions, the blood flow in the lenticulostriate arteries that supply these regions must be high.\(^3\) This probably is the reason for the involvement of these vessels in congenital infections.

Wang et al reported echogenic stripes in the basal ganglia of 34 patients. They suggested that intracranial haemodynamics play an important role in the development of LSV and suggested the term sonographic LSV rather than mineralising LSV as neuropathological evidence of mineralisation has not been proven in all cases.\(^4\)

The underlying pathogenesis of LSV seems to be almost as diverse as the causes. Apart from the perivascular infiltrates caused by congenital infections, vasoconstriction produced by maternal cocaine produces infarction and can produce LSV.\(^5\) Coley et al reported 63 cases of LSV in which hypoxic/ischaemic conditions accounted for 33 cases. They have speculated upon a common etiologic pathway of asphyxia, hypoxia and ischaemia producing these changes. In addition, the studies of the sonographic course of LSV are limited to period till the anterior fontanelle remains open as none of the changes of LSV are mirrored on CT and MRI. There have been only 4 cases in which on coronal T2 MRI showed lesions corresponding to LSV.\(^6\) Wang has suggested that the exact pathogenesis of LSV can be satisfactorily explained only after the establishment of a suitable animal model.\(^7\)

Thus it can be safely said that the detection of LSV on transcranial USG does not specifically point to a condition or a group of conditions. However, although causes such as perinatal asphyxia and hypoxia are clinically evident, conditions such as the congenital TORCH group of infections can exist without manifesting overtly.\(^5\) These congenital infections often have devastating effects on the developing brain.\(^8\)

Wang et al have reported the progression of the sonographic change of LSV in as many as 85% of their patients, but there was no deterioration in the clinical course. They suggested that the progression demonstrated on sonograms does not have clinical significance, because most prenatal brain insults should be inactive after birth except in those cases with inborn errors of metabolism.\(^2\)

In conclusion, detection of LSV can alert the sonologist and clinician to the presence of an underlying insult or pathology to the infant brain in the absence of overt manifestations of the causes of LSV mentioned above, as evidenced by our case.

References