

Hydrocephalus Presenting as Precocious Puberty

We report a case of hydrocephalus presenting as central precocious puberty in an 8-year-old female child.

An eight-year old female child was referred to us for Magnetic Resonance Imaging (MRI) of the brain to exclude any central cause for it. The scans were performed on 1.5 Tesla system (Magnetom, Siemens) in head surface coil. Sagittal, axial and coronal T1 weighted (TE/TR/Ac 22/500 msec/2) and axial T2 weighted (TE/TR/Ac 90/2000 msec/1) scans were obtained using spin echo sequence. The images were reconstructed on 256×256 matrix. The study revealed dilatation of the lateral and 3rd ventricle with aqueductal stenosis and collapsed 4th ventricle (Fig. 1). Brain parenchyma revealed an ischemic infarct in the left parietal lobe in the territory of left middle cerebral artery. The patient gave past history of tubercular meningo-encephalitis.

A variety of central nervous system (CNS) lesions have been found to be responsible for central precocious puberty. With the advent of computed tomography and MRI, hamartoma of the tuber cinereum is being increasingly recognised as an important cause of precocious puberty. Post encephalitic scars, tuberculous meningoencephalitis, hydrocephalus and severe head trauma have been reported as cause of precocious puberty on rare occasions(1,2). How these lesions result in



Fig 1. Axial T1 weighted image showing dilated lateral and 3rd ventricle. An ischemic infarct in the left parietal lobe can also be seen.

precocious puberty is not very clear but probably hypothalamus gets involved by scarring, invasion or pressure depending upon the etiological factor.

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HIV Serosurveillance in Multi-transfused Thalassemic Children

Human immunodeficiency virus (HIV) infection has emerged today as the most dreaded transfusion related complication. Both HIV seropositivity as well as acquired immunodeficiency syndrome (AIDS) have been known to result from transfusion of infected blood or blood products(1). In fact transfusions may be responsible for as many as 22% of all cases of AIDS in children(2).

The thalassemia gene is prevalent in North-West and betathalassemia major is a common hematological condition seen in Pediatric practice. These children, being transfusion dependent, constitute an important risk group for HIV infection. The reported HIV seroprevalence rates in such children vary from 1.09-38.5% in different parts of the world(3-5).

We screened 100 thalassemic children for evidence of HIV infection. These patients had been on a regular transfusion programme for periods varying from 1-12 years. Blood that is used in our hospital is procured from the Blood Bank Society which obtains its supplies from voluntary donors only. Chandigarh is perhaps the only city in India where professional donors are not allowed to donate blood.

There were 77 boys and 23 girls in the study population and the ages varied from 1.5-14 years (mean 6.8 years). A cumulative total of 6971 units had been transfused

to these children over the preceding 12 years with the number of transfusions per child varying from 8-250 (mean 69.71).

Serum was tested for HIV-1 antibody by the competitive ELISA method using Welcozyme kits. It was heartening to note that all children were seronegative for HIV-1 antibody. These results also indirectly reflect the absence of, or a very low HIV seropositivity in voluntary donors of the region. It may be noted that at the time when this study was conducted (1989-1990) our blood bank was not routinely screening all donors for HIV antibody.

Needless to say, absence of HIV seropositivity should not slacken our surveillance programme. All attempts should be made to ensure screening of donor blood for both HIV-1 and HIV-2 antibody. Timely institution of these control measures would go a long way in preventing HIV transmission by this route.

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