

Fig. Tuberculides: Reddish-purplish papular lesions that are symmetrically distributed but more marked on the extensor surfaces.

miliary opacities in both lung fields with a right paratracheal lymphnode. Gastric aspirate for acid fast bacilli was negative.

The histological examination of skin biopsy revealed tubercle formation with many giant cells. Special staining by the Ziehl Neelsen technique, demonstrated acid fast bacilli which were sparsely distributed. Liver function tests, cerebrospinal fluid examination, blood urea and serum electrolytes were within normal limits.

A diagnosis of miliary tuberculosis with skin tuberculides was made and the child was put on antitubercular treatment (streptomycin, isonex and rifampicin). The skin lesions disappeared rapidly over the next few days and the child improved.

Discussion

Skin tuberculides occur preferentially in children and young adults(1). A deep

focus of tuberculosis is present in more than one third of cases and a prompt response to antitubercular therapy is generally seen(2,3). Morrison and Fourie(2) believe that from a tuberculous focus, bacilli periodically enter the circulation where they are opsonised with antibodies and complement and settle out preferentially in slow flowing capillaries in the skin. They suggest that the tuberculide represents an arthus reaction followed by a delayed hypersensitivity response to mycobacteria.

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Prolonged Traumatic Transient Cortical Blindness Following Head Injury

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Cortical blindness occurs in children after head injury. In typical cases, blindness

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Received for publication March 16, 1990; Accepted February 11, 1991 occurs after 15-45 minutes of head injury without loss of consciousness. There is total recovery in few hours. A patient is reported in whom blindness was noticed after 24 hours of unconsciousness and lasted for about 48 hours.

Case Report

A 9-year-old girl, fell from 10 feet high roof. Within 10 minutes she was brought to my clinic. She was in extreme distress and appeared disoriented. She was admitted and all routine investigations including X-ray skull were normal. After thirteen hours, she became unconscious and the neurological examination at that time did not show any localizing sign. She was treated on the lines of head injury and regained consciousness after 24 hours.

At that time she was agitated and was unable to see. Examination showed inability to perceive any objects in the visual field, the pupillary reaction and both fundi were normal. There was no other neurological finding. Forty eight hours after regaining consciousness, her vision recovered fully.

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Transient traumatic cortical blindness in children may have vague visual symptoms. Some children simply cannot see, others report that all appears white, and palinopsia has been recorded. The child may not complain of visual loss, but appear restless, irritable, or disorientated, with an unsteady gait. The most important feature is the absence of any other symptoms or signs. There may be a family history of migraine(1-5). The condition often goes unrecognized since the pupillary light reflexes are normal.

The incidence of transient cortical blindness following head injury is unknown. There is great disparity between our findings and reported by other workers(1-6). Earlier workers reported a typical history of minor head trauma, blindness noticed within an hour and complete recovery within 0-12 hours.

In this case the patient was unconscious for 24 hours, Blindness was noticed after regaining consciousness and complete recovery after 3 days. Keyes and Herskowitz also reported prolonged post traumatic cortical blindness(6).

Little is known about the pathophysiology of the condition. Various hypothesis include (i) interruption of the blood supply to optic nerves and chiasma(3); (ii) an effect on the occupational cortex due to compression of the posterior cerebral arteries against tentorium(4,7); (iii) local cerebral edema is a result of focal trauma(8); (iv) transient spasm of basillar artery. This disorder has also been associated with migraine and epilepsy(1,5,9). Local cerebral vasospasm may be important. Most favor a cortical etiology. The transient hemiparesis following head injury is identical to cortical blindness.

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Prune-Belly Syndrome in a Female

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Guthrie and Parker reported the first case of Prune-Belly syndrome in mid 1890s(1). The syndrome occurs primarily in boys with an incidence of one in 35,000

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Received for publication June 8, 1990; Accepted February 15, 1991 to 50,000 live births. Only about 3% of patients with Prune Belly syndrome are females. Rabinowitz and Schillinger reported 17 female subjects with Prune-Belly syndrome(2). We report a case of Prune-Belly syndrome in a female because of its rarity.

Case Report

A full term, Hindu female, first born of non-consanguinous marriage was referred immediately after birth with complaints of abnormal appearance of abdomen and increased respiratory rate. The maternal and paternal ages were 22 and 25 years, respectively. The mother had regular antenatal check up and had oligohydramnios but was otherwise normal. Antenatal sonography was not done. On examination, the body and umbilical cord were stained with thick meconium. Cord had two arteries and one vein. The abdomen was lax and wrinkled like a wizened prune due to abdominal muscle deficiency (Fig. 1). Both kidneys were palpable. External genitalia and anal opening were normal. Per rectal examination revealed a palpable bladder. The



Fig. 1. Photograph showing baby with prune like abdominal wall.