Idiopathic Intracranial Hypertension due to Intralesional Triamcenolone Acetate

Systemic corticosteroid therapy and its withdrawal is one of the causes of idiopathic intracranial hypertension. Intralesional steroids have not been reported as a cause.

A 3-year-old boy presented with complaints of irritability for one month, nonprojectile vomiting for 4 days and medial deviation of both eyes for 3 days. There was no history of fever, headache, seizures or neck stiffness during this period. The child had scalds over 75% of the body surface area one year previously. For the hypertrophic scar due to scald, the child was receiving intralesional injections of triamcenolone every month for last 7 months. Magnetic resonance imaging (MRI) of brain showed buckling of bilateral intra-orbital optic nerve with partial empty sella turcica, without dilatation of ventricles, and without any structural abnormality or meningeal enhancement. Magnetic resource venography was also normal. He was treated with intravenous mannitol, oral glycerol and oral acetazolamide. His complete blood counts were within normal limits. His lumbar puncture done on 3rd day of admission, revealed normal CSF pressure. CSF gram staining, cytology and biochemistry were normal.

Although the lumbar puncture and CSF pressure recording was not done at the time of acute episode but the acute presentation of vomiting, papilledema, medial deviation of both eyes with normal blood pressure, and MRI findings makes the diagnosis of idiopathic intracranial hypertension likely. Triamcenolone acetonide is a fluorinated prednisolone derivative, with four times the potency of hydrocortisone [1]. Due to fluoridation, it is less soluble than its parent compound. This allows it to remain at the site of injection for longer periods of time, establishing a prolonged duration of action [2]. This facilitates a pooling effect that can result in the slow release of steroid, potentially increasing systemic levels of steroid [1,2]. High systemic levels of corticosteroids are known to cause idiopathic intracranial hypertension [3]. We suggest monitoring of signs and symptoms of raised intracranial pressure in children receiving prolonged intralesional steroids therapy.

ABHISHEK ARYA AND ATUL JINDAL
Department of Pediatrics,
All India Institute of Medical Sciences,
Raipur, Chhattisgarh, India.
dratuljindal@gmail.com

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Are Concerns about Folic Acid Supplementation in Children with Acute Lymphoblastic Leukemia Justified?

The issue of folic acid supplementation to children with acute lymphoblastic leukemia (ALL) remains unresolved pending adequate clinical data. Folic acid supplementation is believed to reduce chemotherapy related complications and improve tolerance allowing adequate drug dosages, particularly for methotrexate, but the fear of rescuing leukemic clones prevents routine supplementation [1]. However, folic acid is unlikely to interfere with anti-neoplastic action of methotrexate as: (*i*) there is apparently no competition between folic acid and methotrexate as the former preferentially utilizes the human folate receptor for entry into the cell whereas the latter and its antagonist folinic acid (reduced folic acid) use reduced folate carrier for their uptake (***Fig.* 1**); (*ii*) Folic acid needs to be reduced by dihydrofolatereductase (DHFR) (an enzyme blocked by methotrexate but can be circumvented by folinic acid) in order to take part in DNA synthesis; (*iii*) Folic acid gets active upon regeneration of the DHFR enzyme only after methotrexate is eliminated from the system; (*iv*) methotrexate and folinic acid are administered at thousand-fold higher dosages as