Introduction:
Vitamin A is a pivotal biochemical factor required for normal proliferation and differentiation of skin as well as for specialized functions such as vision. It is an important fat-soluble vitamin. Its basic molecule is a retinol or vitamin A alcohol. As vitamin A is fat-soluble, it is stored in a variable degree in the body, making it more likely to cause toxicity when taken in excess amounts.

The daily recommended dose of vitamin A is 500 mcg in an adult, 250-350 mcg in children, and 600 mcg in pregnant women. Acute toxicity of vitamin A occurs within a few hours or days after ingestion of very large amounts as a result of accidental overdose or inappropriate therapy. Children often got attracted to vitamin A capsules due to their attractive colors, thus accidental ingestion may occur frequently. The estimated toxic dose is about 25,000 IU/kg. A single dose of >1 million units of vitamin A can also cause acute toxicity. Chronic toxicity in older children and adults usually develop after doses of >50,000 IU/day taken for months. Both acute and chronic toxicity usually cause headache due to increased intracranial pressure. Here we wish to report this case of a 11 years old girl presented with diplopia and 6th nerve palsy, 24 hours after accidental ingestion of a large dose of vitamin A.

Case Report:
Rekha, a 11 years old girl, 5th issue of nonconsanguinous parents got admitted in pediatric department of Dhaka Medical College Hospital on June, 2010 with complaints of history of ingestion of 6 vitamin A capsules (2 lac IU/capsule) 3 days back. She also complained of headache, vomiting, double vision and desquamation of facial skin 24 hours after ingestion of vitamin A capsule. During National vitamin A campaign in her school, she got attracted on colorful vitamin A capsules and took 6 capsules (equivalent to 1.2 million vitamin A) at a time by herself.

On examination, Rekha was lethargic and mildly anaemic. Her facial skin had desquamation at various places. Her systemic examinations were normal and there was no evidence of focal neurological deficit. She complained of double vision and 6th nerve palsy (Fig-1) was noted. She had bilateral papilloedema.

Complete blood count was normal. Serum calcium level was 8.4 gm/dl. Fundoscopy revealed blurred disc margin of both eye. CSF study was normal and MRI of brain showed normal findings. Serum vitamin A level was not estimated due to lack of facility. We managed her conservatively with Acetazolamide (30 mg/kg/day) and Prednisolone (1.5 mg/kg/day) for 5 days. 1 day after admission peeling of skin of whole body started (Fig.-2).

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She improved with conservative management. Clinical signs and symptoms disappeared after 2 days. We discharged her after 5 days with advice for follow up after 2 weeks.

**Discussion:**

Acute vitamin A toxicity generally occurs in children when a single dose >3,30,000 IU is ingested, although some infants can be adversely affected by single doses as low as 100,000 IU. Our case demonstrates acute toxicity developed after a 1.2 million IU of vitamin A at a single dose. The sixth nerve palsy and diplopia occurred presumably from raised CSF pressure. The cause of raised intracranial pressure is not fully understood but may be due to altered CSF resorption or increased production.

Vitamin A toxicity is more common in developed countries in comparison to developing countries. Woman and children of developing countries suffers from vitamin A and other vitamin deficiencies. The benefits of supplementation with high doses of preformed vitamin A twice in a year in combating vitamin A deficiency in woman and children of developing countries are documented. Dietary vitamin A is obtained from preformed vitamin A (from animal foods, fortified foods and pharmaceutical supplements) as well as from provitamin A carotenoid from plant sources. In Europe and United states up to 75% of dietary vitamin A is preformed which is largely derived from multivitamins, fish liver oil and other fortified food such as milk, butter, margarine, breakfast cereals and some snack foods. So, chance of chronic vitamin A toxicity is high there. In developing country only accidental ingestion of high dose can cause acute vitamin A toxicity. Prolonged use of retinoic acid for acne and other skin disorders may cause chronic toxicity.

Vitamin A supplementation efforts were initiated as an immediate action to control vitamin A deficiency while more long term, sustainable interventions could be developed and implemented. Examples include food fortification, diet diversification and biofortification. Because diet diversification and biofortification typically provide vitamin A carotenoid sources, toxicity will not occur.

**References:**