Dextromethorphan: Problems with Formulations

Dextromethorphan is a commonly used antitussive drug in children. Recommended dose of dextromethorphan is 1-2 mg per kg per day [1]. As such there should be no problem in recommending appropriate doses of cough formula containing dextromethorphan, whenever necessary. Problems may occur due to of the following reasons:

1. IAP Pediatric Drug Formularly, 2012 states 1.25 - 2 mg/kg/dose 4 times a day [2], and not per kg/day in 4 divided doses. It appears to be a printing mistake. Some doctors may inadvertently recommend higher dose.
2. Cough syrups containing dextromethorphan by different manufacturers have different quantity of dextromethorphan per 5 mL of liquid (e.g. 5 mg, 10 mg and 15 mg). Some doctors may not be aware of this fact, which can result in inappropriate dosage of dextromethorphan. Similarly, a pharmacist may substitute the brand which may result in lower or higher dose of dextromethorphan to the child. Lower dose would be in-effective and higher dose can result in adverse reaction.
3. Some manufacturers print recommended dosage according to age group on the bottle. For example, two different brands having different composition of dextromethorphan (Piriton CS 10 mg/5 mL and Piritexyl 5 mg/5 mL) but recommend same dose (2.5 mL 3-4 times a day) for a child between 2 and 6 years. The manufacturers of cough syrups should not mention dosage at all, so that pediatricians calculate it as per weight of the child.

Any drug can cause adverse reaction in any individual, and doctors have to inform the patients or their caregivers about it. In case of dextromethorphan, in addition to the potential side effects of the drug, the doctors and patients are exposed to problems created by three sources *viz.*, by experts in form of wrong dosage recommendation,; by pharmaceutical industry in making formulations with different quantity of dextromethorphan and wrong dosage recommendations; and by pharmacists in substituting one brand with other brand without checking the quantity of dexomethorphan in substituted brand. Regulatory agencies should ensure that all brands have similar composition with the correct instructions on product insert.

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Factitious Bleeding Disorder in a Child: An Unusual Presentation of Munchausen Syndrome

Munchausen Syndrome (MS) is a psychiatric disorder characterized by feigning of symptoms of some physical or mental disorder, by patients [1].

A nine-year-old girl presented with complaints of bleeding from multiple sites. Ten days prior to presentation, the child had complained to her parents of severe pain in the right breast followed by bloody discharge from right nipple. Similar bleeding was also seen by the parents in other places, including eyes (***Fig.* 1**), nose, ears, oral mucosa and umbilicus. The 'bleeding' consisted of few drops and was self-limiting. Rest of the history was unremarkable and there was no obvious psychological stressor. Her vital parameters were stable and the systemic examination did not reveal any abnormal findings.

Blood counts, including hemoglobin, platelet count and peripheral smear were within normal ranges. Liver function tests and coagulation profile were normal. Urine and stool microscopy did not reveal any red blood cells or

occult blood. Screening test for Factor XIII levels was normal and platelet aggregation studies showed normal aggregation with ADP, AA, Collagen and Ristocetin.

It was explained to the parents that there was a discrepancy between their child's clinical profile and her investigation reports. In due course of time, the child revealed to the clinician and the parents that she had been applying her mother's liquid vermillion to fake bleeding. Thus, she was diagnosed as a case of Munchausen Sydrome. She was referred for psychiatric treatment, which the parents refused.

Munchausen Sydrome is an extreme form of factitious disorders, wherein the sufferer simulates illness, to assume patient role [1]. It is characterized by multiple outpatient visits or hospitalizations [2]. Typically, sufferers lie deliberately and may consume drugs like insulin, vitamins, warfarin to produce adverse effects like hypoglycemia, and bleeding symptoms [3]. It is uncommon in children and should be suspected if there is a discrepancy between symptoms and signs/ investigation results.

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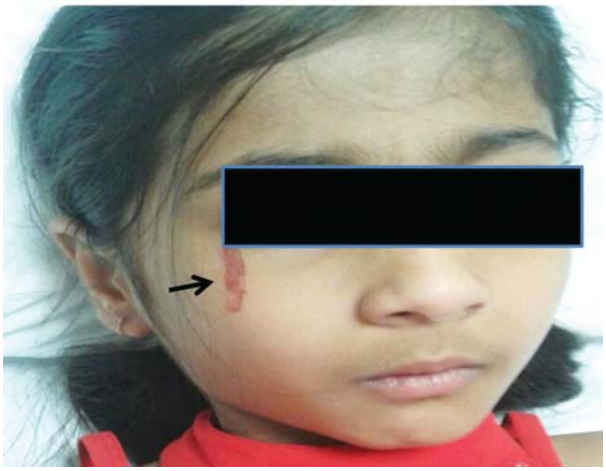

FIG. 1 *Patient showing factitious bleeding from the right eye.*

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Identification of PML/RARá Fusion Gene by RT-PCR Acute Promyelocytic Leukaemia

The genetic hallmark of acute promyelocytic leukemia (APL) is the balanced reciprocal chromosomal translocation of (15;17) leading to a fusion of the promyelocytic leukemia (PML) gene on chromosome 15 and the retinoic acid receptor-á (RARá) gene on chromosome 17 [1]. Identification of PML/RARá fusion gene by reverse transcriptase-polymerase chain reaction (RT-PCR) without evidence of t(15;17) both on conventional karyotype and fluorescence *in situ* hybridization (FISH) is rare [2,3].

An 8-year-old girl was admitted with the complaints of fever and generalized weakness for last 1½ month, and epistaxis and bleeding from gums for last 20 days. She had moderate pallor and hepatosplenomegaly. Routine blood examination revealed hemoglobin 7.4 g/dL, total white cell count 9.8×10^9/L and platelet count was 40×10^9/L. Peripheral blood smear showed 35% neoplastic promyelocytes with cytoplasmic hypergranulation. Coagulation profile revealed normal prothrombin time and activated partial thromboplastin time, but serum fibrinogen level was low. Bone marrow aspiration showed a hypercellular marrow with 66% neoplastic promyelocytes and presence of multiple Auer rods (faggot cells).

Cytochemical staining with myeloperoxidase was strongly positive. Child was diagnosed morphologically as AML-M3 (hypergranular variant). Immunophenotyping analysis of the bone marrow cells was consistent with APL. Conventional cytogenetic analysis showed 46, XY, isochromosome (17q). FISH analysis was negative for t(15;17). RT-PCR was positive for PML–RARá fusion transcripts. She had completed both induction and consolidation phase of all-trans retinoic acid (ATRA) based chemotherapy regimen. Child responded dramatically and went into molecular remission.