

Median Canaliform Dystrophy of Heller

A 5-year-old boy presented with single dystrophic thumb nail since last seven months. Mother gave history of constant biting of thumb nails. On examination, there was median split of right thumb nail with transverse furrows extending from longitudinal split (*Fig. 1*). All other investigations were non-contributory. He was also diagnosed to have attention deficit hyperactive disorder (ADHD).

Median canaliform dystrophy of Heller (*solenonychia, dystrophia unguis mediana canaliformis, nevus striatus unguis*) is an uncommon and rare dystrophic condition of nail plate which usually affects thumb nails symmetrically and is characterized by a midline defect of the nail plate leading to longitudinal splitting in the midline with canal formation. It extends from cuticle of nail and shows transverse furrows extending laterally from longitudinal splitting giving characteristic inverted fir tree appearance. It may rarely involve toe nails and other finger nails.

The proposed etiopathogenesis is repeated self inflicted trauma in form of habitual nail biting. In the majority of cases the cause is unknown and it may revert back to normal after many months to years. Other proposed that there is also absence of keratinocytes adhesions within nail matrix with dyskeratosis which is responsible for formation of longitudinal groove with splitting of nail plate due to weaker tensile strength. There



FIG.1 Dystrophic thumb nail with median split.

are some case reports of median canaliform dystrophy of Heller after using retinoids and with familial occurrence. Subungual tumors like glomus tumor, myxoid tumors, papilloma, squamous cell carcinoma, and melanoma may form longitudinal groove and then splitting the nail plate in the middle portion leading to median canaliform dystrophy.

The treatment includes avoidance of repetitive nail trauma through behavioral counselling, and topical tacrolimus.

GANESH AVHAD AND PRIYANKA GHUGE

Department of Dermatology,

L.T.M. Medical College and General Hospital, Sion, Mumbai,

400 022, India.

g.avhad84@gmail.com

Childhood Bullous Mastocytosis

A 1-year-old girl presented with numerous pruritic bullae all over the body. There were complaints of recurrent episodes of diarrhea and vomiting. Development was normal. She started developing episodes of intense itching at 8 months of age, followed by appearance of erythematous macules, plaques, and tense bullae at the sites of itching as well as other sites (scalp, trunk and upper extremities) (*Fig. 1 and 2*). Bullae were present on a non-urticated base containing clear fluid. Few of the urticarial plaques showed *peau-d-orange* appearance. Darier's sign was positive. The palms, soles and mucosae

were free. Routine investigations and urine analysis were normal. Skin biopsy showed sub-epidermal bulla and an upper dermal inflammatory infiltrate comprising lymphocytes and many mast cells. Toluidine blue staining showed metachromatic granules and a diagnosis of bullous mastocytosis was made. The patient was treated with antihistamines for itching and topical as well as systemic antibiotics for preventing secondary infection. Parents were counseled regarding the prognosis and course of disease and the importance of avoiding certain medications that may provoke mast cell degranulation.

Bullous mastocytosis is a severe variant of mastocytosis, 30% cases manifesting within 6 months of age. The typical childhood disease is linked to Glu-839-



FIG. 1 Multiple tense bullae and erosions over the trunk.



FIG.2 Bullae, plaques and erosions on face and trunk.

Lyc c-kit mutation. Commonest clinical variants are telangiectasia macularis eruptiva perstans, mastocytoma, diffuse cutaneous mastocytosis and urticaria pigmentosa. Many agents stimulate the degranulation of mast cells, such as bacterial toxins, physical stimuli, poisons, biological peptides, polymers and drugs like aspirin, codeine, morphine, quinine etc. The close clinical differentials include chronic bullous disease of

childhood, epidermolysis bullosa and staphylococcal scalded skin syndrome (SSSS). Combinations of H1 and H2 blocking agents have been the mainstay of treatment for most of the uncomplicated cases.

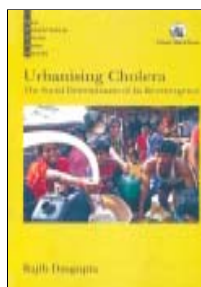
DIPTI DAS, ANUPAM DAS AND SWAPAN SARDAR

Department of Dermatology

Medical College and Hospital, Kolkata, West Bengal, India.

anupamdasdr@gmail.com

BOOK REVIEW



Urbanising Cholera: The Social Determinants of Its Re-emergence

RAJIB DASGUPTA

Orient Black Swan; New Delhi, 2012

Pages: 348; Price: Rs. 429/-.

This work needs to be seen in the framework that goes much beyond cholera, the title notwithstanding. Seven inter-connected sections reignite much needed debate and bring something new to the public health discourse in four cardinal domains: unfinished agenda of water, sanitation, and associated inequities; specter of unthoughtful urbanization; a transition of waterborne infections from

epidemic to endemic/focal outbreaks scenario; and application of social determinants approach in an acute communicable disease. There is a unique analysis of the spatial distribution of cases within Delhi, making a compelling argument of 'area effects' as an epidemiological approach; and a deconstruction of the historical 1988 episode in Delhi, which would be variedly classified as 'epidemic', 'outbreak' or 'clustered events' (a perpetually unsettled debate). The flipside is that the title and the key words punctuating the narrative limit a big picture to a cholera centric construct.

SANJAY CHATURVEDI

Professor of Community Medicine,

University College of Medical Sciences,

Delhi-110095, India. cvsanjay@hotmail.com