

FIG. 1 Multiple tense bullae and erosions over the 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



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

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.

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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 an 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.

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