Swyer-James Macleod Syndrome

Swyer-James Macleod Syndrome (SJMS) is a rare condition characterized by unilateral hyperlucency of part of whole of lung which usually follow bronchiolitis/pneumonitis in early childhood(1). Despite this, the majority of cases have been diagnosed at a later age.

A two-year-old male child presented to us with chronic cough for past 6 months. The cough started following an acute respiratory illness of unknown etiology for which he had been hospitalized for two weeks. Examination revealed a well-preserved child with normal vital parameters and a SpO2 of 97% in room air. There was diminished breath sounds on the left side in the chest while rest of the physical examination including an ENT examination was unremarkable. Hemogram with ESR was normal. ELISA for HIV and Mantoux test were also negative. Chest radiograph revealed a hyperlucent lung with oligemic lung fields on the left (Fig.1). CT scan of the chest revealed similar findings and there was no evidence of any bronchiectasis. A lung perfusion scan with Tc99m labeled



Fig.1. X-ray chest showing unilateral hyperlucent lung (L)

MAA showed markedly decreased tracer uptake in left lung with normal homogenous distribution on the right. Ventilation scan and pulmonary function test could not be carried out, as the child was very small to cooperate for the same. He obtained no benefit with a trial of bronchodilators, has been administered pneumococcal and influenza vaccines apart from routine immunization and is on regular follow up.

Swyer and James first described the syndrome in 1953 followed by macleod in 1954(2). The disease starts as an obliterative bronchiolitis with concomitant vasculitis commonly following infections organisms such as adenovirus, measles or pertussis. The damage to the terminal or respiratory bronchioles in early childhood possibly prevents normal development of their alveolar buds(1). One study of bronchoalveolar lavage in SJMS suggests an ongoing inflammatory process in the lung(3). The condition should be differentiated from congenital anomalies of airway/pulmonary vessels and bronchial obstruction due to mucus plug or foreign body(1). Management is based on (i) pulmonary function test, which may reveal mixed restrictive and obstructive airway disease (ii) prevention and/or follow up for infection and bronchiectasis and (iii) rarely surgery(4,5).

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Parathyroid Adenoma

Primary hyperparathyriodism (PHPT) is associated with either an adenoma or hyperplasia of chief cells of the parathyroid gland. It may occur sporadically, as a single autosomal dominant familial disorder, or as a part of the autosomal dominantly transmitted complex of multiple endocrine neoplasia (MEN). PHPT is relatively rare in children and adolescents(1,2). Patients of PHPT in India are comparatively young and they suffer from overt skeletal and renal manifestations.

A 12-year-old boy presented with a pain in abdomen, decreased appetite, and constipation for three months and severe nausea and vomiting since eight days. Physical examination of the child was unremarkable. Investigations revealed: blood urea 34 mg/dL, creatinine of 1.5 mg/dL, serum calcium level was 19.5 mg/dL, phosphorus 3.2 mg/dL, and serum alkaline phosphatase 563 IU/L. Parathyroid hormone levels were 380.0 pg/ mL (12-72 pg/mL). USG abdomen showed small renal calculi in left kidney. Bone changes of hyperparathyroidism were not seen. CT neck showed a well-circumscribed enhancing lesion 1 cm \times 0.9 cm at the level of the thyroid lobe suggestive of parathyroid adenoma. Parathyroid scintigraphy with technetium-99m-MIBI showed evidence of tracer activity in the region of upper pole of

left lobe of thyroid confirming parathyroid adenoma in that region. Patient subsequently underwent surgery.

PHPT most often associated with parathyroid adenoma(3,4). Clinical manifestation of hypercalcemia includes muscular weakness, anorexia, nausea, vomiting, constipation, polydipsia, polyuria, loss of weight and renal calculi. Osseous changes may include pain in back or extremities, fractures and decrease in height due to compression of vertebra. Surgical removal is recommended. Prognosis is good if the disease is recognized and treated early. When extensive osseous lesions are present, deformities may be permanent(3).

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