agenesis (with or without uni- or bilateral lobster clawhand), to bilateral tibial agenesis and bilateral lobster clawhand(2). Our proposita had unilateral hypoplastic pelvic bones and femur with tibial agenesis, hypoplastic 3-toed foot, and cleft hands. Other associations seen in our case are sacral agenesis, anorectal atresia, hemivertebra and congenital heart disease (CHD).

TH-SHFM associated with fatal pulmonary hypertension and congenital alveolar capillary dysplasia is reported in a consanguineous Turkish family(3). CHD seen in our case was a large membranous ventricular septal defect. Lethal congenital cardiac malformations have been described with "Tibial Hemimelia-Polydactyly syndrome", an entity similar but distinct from TH-SHFM(4). Congenital heart disease has not been reported with TH-SHFM.

Features of Tibial hemimelia-split hand/ foot syndrome, in absence of any family history, in this neonate with anorectal atresia, sacral agenesis, hemivertebra and in particular the CHD may either be a rare association or is it possible that there is an unexplored etiopathogenetic correlation which could explain the presence of all these rare conditions in a single neonate!

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Presence of Thyroid Antibodies in a Child with Systemic Lupus Erythematosus

A 6-year-old girl presented with high grade fever daily and painful swelling of all major joints for two months. She had been treated with various anti inflammatory drugs and corticosteroids as a case of chronic arthritis without much relief. She was investigated in detail, diagnosed and confirmed to have systemic lupus erythematosus (SLE) and lupus nephritis based on the following results obtained. Her laboratory investigations were total WBC count -12,100 / cu.mm, differential count-N 62%, E 2%, L 30%, M 6%, ESR 62 mm at 1 hr, Hb

10.5 g%, platelet count 3,92,000/cu mm, reticulocyte count 1.0%, blood picture anisocytosis with hypochromia. Her serum total complement was 40% and antinuclear antibody (ANA) was positive. Her anti dsDNA antibody level was 51 AU/mL (normal <30 AU/mL). Renal biopsy showed lupus nephritis (WHO grade II). Her 24 hour urine protein excretion measured 16 mg in 790 mL. Her serum albumin was 3.8g%. All other investigations were within normal limits.

One month prior to the onset of these symptoms, she had thyroid function tests done elsewhere in view of the strong family history of hypothyroidism with the maternal grandmother, uncle and aunt reportedly affected but whose further details were unknown. She did not have any goitre or features of hyper or hypothyroidism at that time. Her thyroid function tests then were reported to be as follows: serum triiodothyroxine (T_3) 49 ng/dL (normal 70- 200), serum total thyroxine (T_4) 2.77 µg/dL (normal 4-13) and serum thyroid stimulating hormone (TSH) 98.07 mIU/mL (normal 0.3- 6.0). She was started on thyroxine 100 micrograms once daily since then.

Thyroid function tests repeated by us while she was receiving thyroxine replacement therapy showed serum total T_4 14.8 µg/dL, free thyroxine concentration (FT_4 conc.) 2.00 ng/dL (normal 0.8-2.0) and TSH of 2.83 mIU/mL (normal 0.3-4.5). Her thyroid antibody titers were: thyroglobulin hemagglutination antibody (TGHA) 1:3202 and microsomal hemagglutination antibody (MHA) 1:1602 (significant titer >1:102). She was prescribed prednisolone and azathioprine for treatment of lupus nephritis. She was advised to continue thryroxine in replacement doses, in view of her high level of thyroid antibody status, strong family history and prior biochemical evidence of hypothyroidism. She was reviewed three months later, when the repeat thyroid function tests revealed T_4 16.7 µg/dL, FT_4 conc. 2.35 ng/dL and TSH of 0.678 mIU/mL after which thyroxine was stopped because of hyperthyroxinaemia and severe TSH suppression. She was advised followup and monitoring of thyroid function tests to ensure euthyroid status or disordered thyroid function.

Even though high prevalence (32%) of antithyroid antibodies in children with SLE has been reported in the literature, thyroid dysfunction is rare (9%)(1). The corresponding figures in Miller series(2) were 25% and 7.5% and 43% and 18% respectively in the report by Eberbard, *et al.*(3).

B-lymphocyte dysregulation is the immunopathological explanation for the production of autoantibody and other antibodies in SLE. As in SLE, presence of antithyroid antibodies without any other biochemical evidence of thyroid disease has been documented in several other autoimmune endocrine diseases(4,5). Drugs used in the treatment of SLE such as glucocorticoids may either suppress serum T_3 or T_4 values or prevent the development of overt thyroid disease by anti-inflammatory action(3).

In the presence of high level of thyroid antibodies, diagnosis and treatment of thyroid abnormalities based exclusively on biochemical abnormalities in children with SLE require special attention and regular followup.

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