Parathyroid Carcinoma Presenting as Genu Valgum

A 11-years-old male child was brought to our hospital with presenting complaints of progressive bowing of legs with mild pain 2 months. Past history and family history were not significant. Vitals and anthropometry were normal.

X-rays wrist showed fraying but no cupping. Investigations showed Ionized calcium level 1.627mol/l (normal-1.12-1.23mmol/l), 24 hour urine calcium 420 mg/day, serum phosphorus 2.9gm/dL, serum alkaline phosphatase 2820 IU, and vitamin D3 level 18.88 ng/mL (normal 11.1-42.9 ng/mL). Abdomen ultrasound showed cystitis. Tubular reabsorption of phosphate was 92 (Max 100- min-82). ABG pH was normal. Ultrasound neck was done for a mass in neck, which showed enlarged parathyroid gland (right) 2.5 cm 1×1.5 cm. To rule out multiple endocrine neophasis syndrome MRI brain and abdomen with contrast was done which revealed no adrenal/pituitary abnormalities. His parathormone level was 1630 pg/mL (normal 10-69). Tc-99m-MIBI static study of the neck/mediastinum showed features suggestive of functioning parathyroid lesion (suggestive of adenoma) in the region of lower pole of right lobe of thyroid. Excision of the adenoma was done. Child developed hypocalcemia symptoms on first post operative day and was treated with parentreal calcium gluconate. He was discharged on 7th POD in good health. Pathologic specimen was found to be parathyroid carcinoma. Subsequently underwent right hemithyroidectomy for the carcinoma. Currently child is doing well and is under orthopedic follow up for corrective surgery.

Childhood hyperparathyroidism is rare. Onset during childhood is usually as a result of a single benign adenoma. Manifestations are usually after 10 years and have an autosomal dominant pattern of inheritance. Parathyroid carcinoma is rare in children, and typically presents with significant hypercalcemia and a palpable neck mass [1]. At all ages, the clinical manifestations of hypercalcemia of any cause include muscular weakness, fatigue, headache, anorexia, abdominal pain, nausea, vomiting, constipation, polydipsia, polyuria, loss of weight, fever and nephrocalcinoisis. Renal calculi may occur and may produce renal colic and hematuria. Osseous changes may produce pain in the back or extremities, disturbances of gait, genu valgum, fractures, and tumors. Bone presentation is very rare [2]. This case gains importance not for its rarity but for the fact that parathyroid carcinoma should also be kept in the back of our mind for onset of genu valgum in an adolescent.

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Congenital Splenorenal Shunt: A Dilemma

We report a two years old girl who was born preterm at 36 weeks with a birth weight of 2.6 kg to a primi mother by emergency cesarean section due to uncontrolled hypertension. Mother's age was 24 years and she was hypothyroid, hypertensive and had Type 2 diabetes mellitus. She was on thyroxine, nifedipine and oral hypoglycemics, which was changed to insulin during pregnancy.

Baby was hypotonic and lethargic. TSH was >100mlU/L. She was started on oral thyroxine and discharged on 7th day of life. At 6 months, baby presented with fast breathing. Heart rate was 180/min, respiratory rate was 68/min and the baby looked flushed. Thyroxin

induced hyperthyroidism was suspected. Blood pressure recording in right upper arm was 140/100 mmHg. Her thyroid profile was within normal limits.

Child was put on propranolol and was investigated for secondary causes of hypertension. Renal function test, plasma adrenaline, noradrenaline; urine 24 hours metanephrine, 24 hours VMA; serum cortisol, aldosterone, and renin were within normal limits. CT abdomen showed normal sized kidneys and normal appearing liver and spleen. Echocardiography was with in normal limits. CT renal angiography showed single renal artery on both sides with no coarctation or aneurysm, single renal veins on both sides, abnormal large spenorenal shunt between splenic vein and left renal vein, left renal vein dilated measuring 1.1 cm, shunt measured 0.6 cm, portal vein narrowed to 0.2 cm (***Fig.*1**).

Presently the child is on propranolol 5 mg twice a day, and L-thyroxine 50 µg daily. Her growth and milestones are with in normal limits. Her BP and thyroid status is with in range.

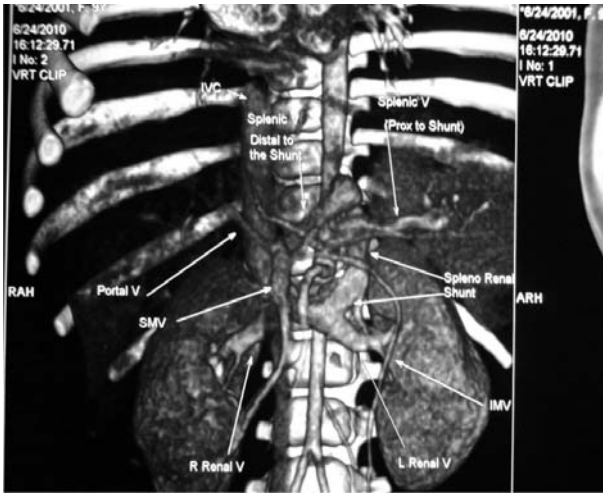

Fig.1 *CT renal angiography showing large splenorenal shunt.*

Our dilemma was whether hypertension is due to the splenorenal shunt and the future management of this shunt. Pulmonary hypertension has been reported in cases with congenital porto-systemic venous shunt but we could not get any literature reporting systemic hypertension in this condition. Extremely hypoplastic kidneys have been reported in an aborted fetus with congenital splenorenal venous shunt. But ultrasonography shows bilateral normal size kidneys in this child.

It is reported that patients with congenital splenorenal shunt may be asymptomatic in the initial years but develop portosystemic encephalopathy at later stage. Hepatic cirrhosis has also been reported. We are following her with serial ammonia levels which is with in normal limits. CT guided liver biopsy shows normal histology.

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Biliary Atresia with Cytomegalovirus

We read with interest "Evolving biliary atresia with cytomegalovirus" in the August issue [1], as the etiology and pathogenesis of biliary atresia (BA) still remains as an enigma. Instead of answers, we are left with several questions in our mind. It was not clear whether the author is implementing CMV as a cause for biliary atresia in these two cases or as a red herring? Though, there are several speculations about CMV causing biliary atresia, so far there is no hard evidence implementing the virus as a causative agent [2,3]. In the first case early liver decompensation and low gamma-glutamyl transferase (GGT of 10 IU/L) in the presence of severe cholestasis raise the suspicion of an underlying metabolic problem