## **Transverse Testicular Ectopia**

We recently encountered a seven-year-old boy having type I transverse testicular ectopia (TTE). He presented with left inguinal hernia and contralateral impalpable undescended testis; the left testis was normally located in the ipsilateral hemiscrotum. During left herniotomy, a second testis was unexpectedly delivered through the deep inguinal ring. The two testes shared the common patent processus vaginalis. The two vasa deferentia were fused for a short length about 4 cm. above the testes. The vascular supply to the ectopic testis originated from the appropriate ipsilateral side. Presence of mullerian ductal structures was ruled out by laparoscopy; optical camera could be easily introduced through the open hernial sac. Because of the ductal fusion, the ectopic testis was brought down on the left side to avoid damage during separation and placed in either hemiscrotum through transceptal window.

TTE, also named testicular pseudoduplication, unilateral double testis and transverse aberrant testicular maldescent, is an uncommon anatomical abnormality in which both the gonads migrate towards the same hemiscrotum; only about hundred cases have been reported in literature. The ectopic testis may lie in opposite hemiscrotum, in the inguinal canal or at the deep inguinal ring. An inguinal hernia is invariably present on the side to which the ectopic testis is migrated. Variations in the anatomical position of the vasa deferentia and abnormalities of insertion of the vas into the testis can occur. Fusion of vasa deferentia has been described previously also(1).

Based on the presence of various associated anomalies, TTE has been classified into 3 types: (i) associated with inguinal

hernia alone (40-50%); (*ii*) associated with persistent or rudimentary mullerian duct structures (30%); (*iii*) associated with other anomalies without mullerian remnants (inguinal hernia, hypospadias, pseudohermaphroditism and scrotal abnormalities) (20%)(2).

The mean age at presentation is 4 years(2). In most of the cases, the correct diagnosis was not made pre-operatively and the condition was revealed during herniotomy. Recently, MRI and MR venography have been suggested for preoperative location of impalpable testis(3). Laparoscopy is useful for both diagnosis and management of TTE and the associated anomalies(4). Treatment includes transceptal orchidopexy or extra-peritoneal transposition of the testis, search for mullerian remnants and other anomalies and long-term postoperative follow-up. Like all dysgenetic infertility and progression to testes. malignancy are relatively frequent with TTE(5).

We emphasize that associated mullerian ductal structures, if any, should be ruled out by laparoscopy or mini-laparotomy in the same sitting.

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## Neonatal Hypocalcemia Due to Asymptomatic Maternal Primary Hyperparathyroidism

Neonatal hypocalcemia resulting from maternal primary hyperparathyroidism (MPH) is usually detected clinically in the first 2 weeks of life. Occasionally, diagnosis of primary hyperparathyroidism in a young asymptomatic mother is made when the infant presents with hypocalcemia. We present an infant with late onset hypocalcemia resulting from a combination of transient hypoparathyroidism due to asymptomatic MPH and vitamin D deficiency.

A thirty-five-day-old infant was admitted to Pediatric Emergency Service because of recurrent tonic-clonic convulsions for 36 hours. He was a full term male baby born to a 22 year old G2, P2 woman by normal spontaneous vaginal delivery with a birth weight of 3500 g. He was only breast fed. The family history revealed that his brother was hospitalized and treated because of neonatal hypocalcemic convulsion but no further investigations were carried out.

On physical examination his anthropometric measurements were normal. He had no dysmorphic features. Initial laboratory evaluation showed hypocalcemia, hyperphosphatemia slightly increased alkaline phosphatase (ALP), inappropriately low PTH level for concurrent degree of hypocalcemia and sligthly low 25-OH vitamin D<sub>3</sub> (Table I). The remainder of the laboratory findings were unremarkable. Based on these findings, he was diagnosed as hypoparathyroidism and started on calcium boluses. After his calcium level reached 7mg /dL, oral calcium (75 mg/g/day) and vitamin D<sub>3</sub> supplements (800 IU/day) were given. He remained asymptomatic after second day on admission. Since he had no findings consistent with DiGeorge syndrome and he had a positive family history, maternal hyperparathyroidism was investigated. The mother had high levels of calcium and PTH and low level of phosphorus. She was diagnosed as hyperparathyroidism and referred to endocrinologist. A technetium scan showed an area of increased uptake on the right side of neck suggesting a parathyroid adenoma. It was removed by surgery.

The presentation of asymptomatic maternal hyperparathyroidism by convulsion in an infant is exceedingly rare. Hyperparathyroidism in asymptomatic mothers might easily have been missed if the maternal calcium status had not been investigated(1-4) like in our patient's mother. Suppression of the fetal parathyroid gland by maternal hypercalcemia often causes transient neonatal hypocalcemia(5). Low vitamin D levels of the patient might have exacerbated the hypocalcemia observed in this infant which may due to several causes : he has not received

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