Diphallia with Duplicated Urethra and Urinary Bladder: A Rare Congenital Genitourinary Tract Anomaly

A 5-month-old boy presented to the outpatient department with complaints of two separate phalluses [Figure 1a]. On examination, the upper phallus was smaller and had normal spontaneous urine excretion, while the lower phallus was larger and exhibited occasional urine excretion. A retrograde urethrogram study using an infant feeding tube (IFT) revealed opacification of two distinct and separate penile and bulbar urethras, each showing a normal course, caliber, and outline. Subsequently, when the IFT was gently advanced across the right and left posterior urethra sequentially, it revealed opacification of two small, non-communicating contrast-filled structures, suggesting complete urinary bladder duplication. Additionally, there was reflux of contrast into the left ureter and pelvicalyceal system from the left-sided urinary bladder [Figure 1b-1e]. Ultrasonography and magnetic resonance imaging confirmed complete duplication of the urinary bladder and a small left kidney [Figure 1f-1h]. The final diagnosis was diphallia with duplication of the urinary bladder and urethra, accompanied by left-sided vesicoureteral reflux (VUR) was made.

Diphallia is a rare anomaly, with an incidence of 1 in 5–6 million live births. It is an embryological malformation with complex etiologies, often related to gene alterations.[1] Diphallia can be associated with various abnormalities, including genitourinary, gastrointestinal, spinal, cardiovascular, and musculoskeletal abnormalities. Genitourinary anomalies may include duplicated urethra and urinary bladder, ectopia vesicae, VUR, single functioning kidney, and pelvic kidney.[2]

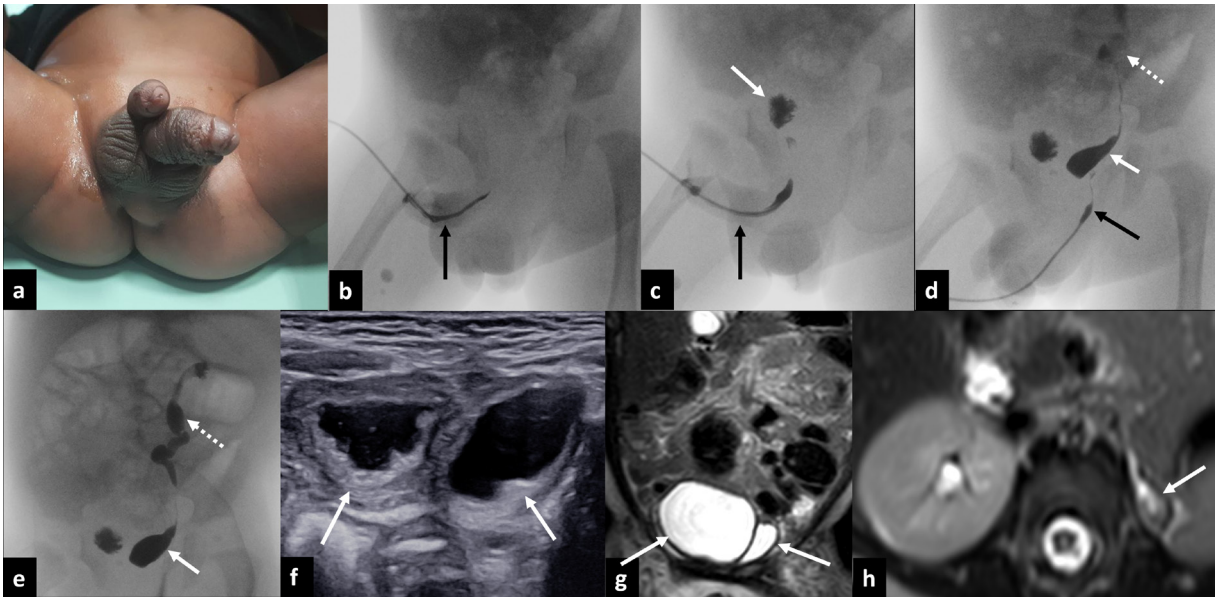

Figure 1: (a) Clinical photograph showing diphallia. (b-c) Retrograde urethrogram of the upper phallus reveals opacification of a distinct urethra (black arrows) and communication with one of the two separate urinary bladders (white arrow in c). (d-e) Retrograde urethrogram of the lower phallus shows opacification of the second distinct urethra (black arrow in d) and its communication with the second urinary bladder (white arrows in d and e). There is also contrast opacification of the dilated and tortuous left ureter and left pelvicalyceal system, indicative of vesicoureteral reflux (dotted arrows in d and e). (f) Gray-scale ultrasound showing duplicated urinary bladders (white arrows). (g-h) Magnetic resonance imaging confirming duplicated urinary bladders (white arrows in g) and a small left kidney (white arrow in h).

Conflicts of interest: There are no conflicts of interest.

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Extensive Tumoral Calcinosis and Acral Osteolysis

A 39-year-old male on hemodialysis developed painful swelling at the tips of the right index and middle fingers, left elbow and wrist, and right shoulder. X-rays showed calcified densities (blue arrows) in the right index and middle fingers [Figure 1a and b], left wrist [Figure 1c], bilateral shoulders [Figure 1d and e], left elbow [Figure 1f], and left femur [Figure 1g]. Acral osteolysis affected the distal phalanx of right thumb [Figure 1a], right long finger [Figure 1b], and left index finger [Figure 1c]. Labs showed normal serum calcium, elevated phosphate, and elevated parathyroid hormone (PTH) of 1762 pg/mL. Nuclear parathyroid scan revealed hyperplasia of three parathyroid glands. Patient was started on cinacalcet with improvement in PTH level and was referred to endocrine and orthopedic surgery. This case depicts the effects of severe tertiary hyperparathyroidism on the bone resulting in acral osteolysis and tumoral calcinosis.

Tumoral calcinosis is characterized by extraosseous calcium deposits in periarticular soft tissue regions. Lesions may be asymptomatic or cause local pain, ulceration, and neurovascular symptoms due to compression with risk of secondary infections.[1] Typically, the lesions are in the periarticular regions, especially around the shoulder, hip, elbow, and wrist. Primary forms occur due to excess phosphate reabsorption in the kidney secondary to mutations in genes involving proteins that promote phosphate excretion, for example, FGF23, GALNT3, and KL.[2] Secondary tumoral calcinosis occurs due to

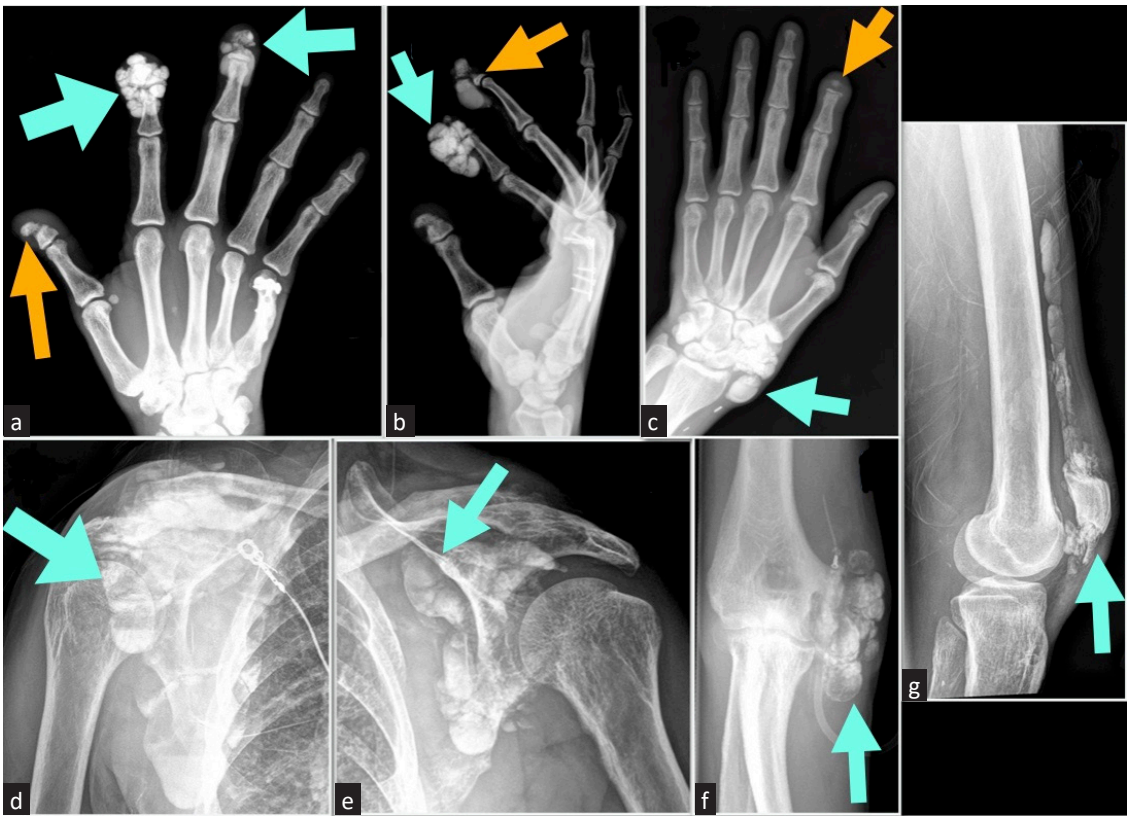

Figure 1: Plain radiograph showing calcified densities (blue arrows) in the (a and b) right index and middle fingers, (c) left wrist, (d and e) bilateral shoulders, (f) left elbow, and (g) left femur. Orange arrows represent acral osteolysis that affected the distal phalanx of (a) right thumb, (b) right long finger, and (c) left index finger.