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Fetal Urinary Ascites - A Case Report.

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ABSTRACT

The most common cause of fetal ascites is posterior urethral valve. It is an obstructive developmental anomaly in the posterior urethra and genitourinary system of male newborns. Diagnosis of posterior urethral valve is made antenatally by the presence of keyhole ureter in Ultrasound and confirmed postnatally by voiding cystourethrogram. Here I present a case of fetal urinary ascites secondary to rupture of posterior urethral valve obstruction.

Keywords: Fetal urinary ascites, posterior urethral valve, keyhole sign.

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INTRODUCTION

The most common cause of fetal ascites is posterior urethral valve. Other causes are hemolytic diseases, severe anaemia, thoracic duct obstruction, cardiac diseases and congenital infections [1]. Mechanisms involved in ascitic fluid formation are urinary tract obstruction, abnormal lymphatic drainage, decreased plasma oncotic pressure, venous return obstruction and increased capillary permeability [2, 3]. Posterior urethral valve is an obstructive developmental anomaly in the posterior urethra and genitourinary system of male newborns, which increases voiding pressure and alters normal development of fetal bladder and kidneys [4].

Case Report

26 years old female, G2P1L0, Rh +ve with 30 weeks gestation, Booked & Immunised elsewhere referred to SBMCH, Chrompet with history of previous preterm normal vaginal delivery with recently diagnosed Gestational Diabetes Mellitus around 29 weeks not on Treatment. Patient came in active labour with an Ultrasound finding of Fetal Ascites. Patient delivered spontaneously a Preterm, Large for Gestational Age, Boy baby of 3 kgs with generalized edema (Facial puffiness, ascites and bilateral pedaledema) which did not cry after birth even after stimulation. Hence baby was intubated and put on ventilator. Surfactant was given. Baby was started on IV antibiotics. Ascitic tapping done and fluid sent for analysis and was reported as urinary ascites. On further evaluation baby was provisionally diagnosed to have posterior urethral valve obstruction and fetal urinary ascites was secondary to rupture of unilateral Hydroureteronephrosis. Baby voided urine freely after temporary catheter insertion and removal. Baby had minimal renal dysfunction, which improved after couple of days. On day5 of life, Baby was weaned off ventilator, on day8 of life catheter was removed, baby passed urine well and had no obvious ascitic fluid collection. Pediatric surgeon opinion was obtained in view of suspected urethral obstruction, as per his advice further work up was postponed, as the baby was voiding urine and feeding well. Danger signs (abdominal fullness, decreased urine output, refusal of feeds and irritability was explained to the parents and baby was discharged subsequently. Postnatally mother had elevated blood sugars and started on Oral Hypoglycaemic agents.

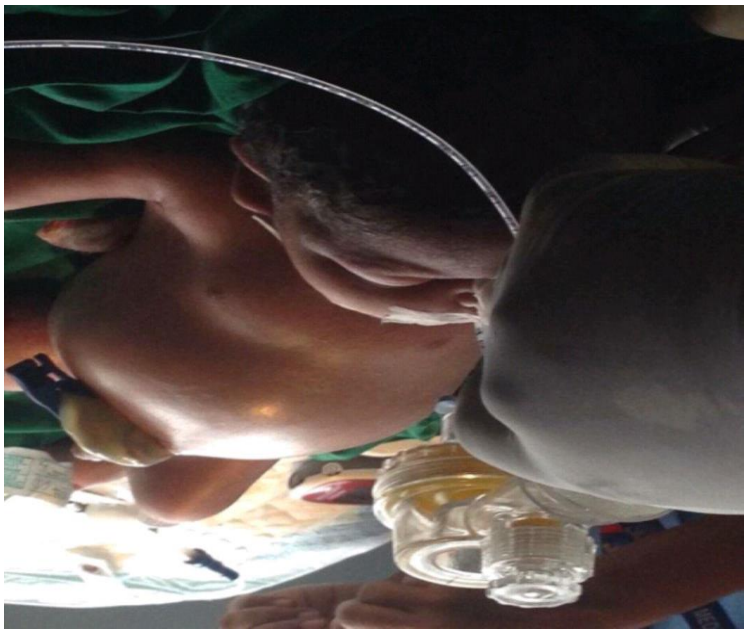

Figure 1: Baby with ascites and facial puffiness.

DISCUSSION

The most common cause of bladder outlet obstruction is posterior urethral valves. Incidence -1 in 8,000 to 25,000 live births**.** 10% in utero urinary obstruction. 1 in 1250 fetal USG Screenings.[5] Urine extravasates across a renal fornix enters the retroperitoneum and travels across the peritoneum as a transudate, this is subjected to the large absorptive mesothelial surface that quickly normalizes the

creatinine and electrolytes values, masking the identity of ascitic fluid as urine [6]. Langenbeck is credited with first reporting of congenital obstruction of the prostatic urethra in 1802 (Dewan et al, 1999). Hugh Hampton Young, more than a century later, named it as posterior urethral valves (Young et al, 1919).Valves have occurred in siblings, in twins, and in successive generations (Farkas and Skinner, 1976 ; Hanlon-Lundberg et al, 1994 ; Morini et al, 2002). PUV is due to Abnormal is insertion of mesonephric duct into fetal cloaca, mesonephric ducts enter cloaca more anteriorly than normal, during unfolding of cloaca migration of mesonephric duct is impeded and fusion of these ducts lead to formation of valves[7].

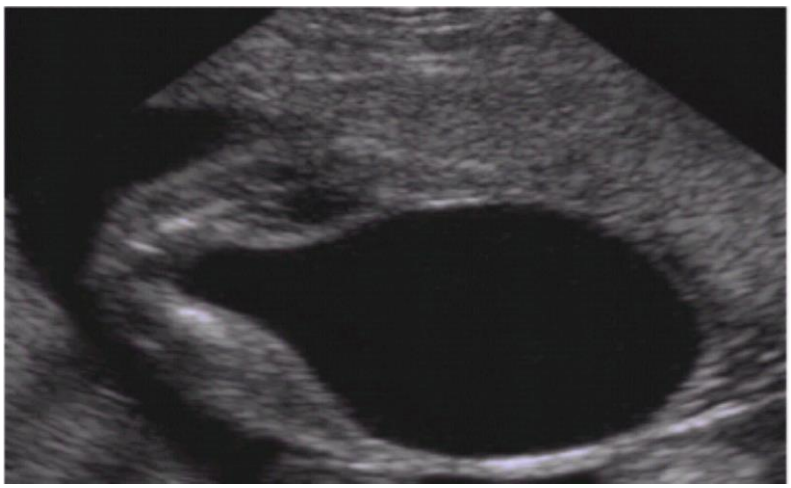

Figure 2: USG showing keyhole sign.

CLINICAL SIGNS [8]:

Neonatal period:

- Potters morphology
- Respiratory distress
- PUV: Dilated palpable bladder
- And / ascites

Later presentation:

- Voiding difficulty
- UTI
- End Stage Renal Disease

DIAGNOSIS:

- Ante natal USG: 40 – 60 %
- Valve develops at 8 weeks and detected after 14 weeks
- The accuracy of ultrasound diagnosis is proportional to fetal age, and sometimes valves may be missed in scans done before 24 weeks of gestation
- (Skolder et al, 1988 ; Abbott et al, 1998)
- DIAGNOSIS- USG-after 24 weeks more accurate.
- Prenatal USG shows B/L hydronephrosis, thickened bladder, dilate d prostatic urethra- key hole sign (Dilated bladder above and Dilated prostatic urethra, oligohydraminos.[9]
- MRI – Confirms Diagnosis

AFTER BIRTH:

USG, VOIDING cystourethrogram, Endoscopy, Renal Scintigraphy, Urodynamic evaluation

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DIFFERENTIAL DIAGNOSIS:

- Posterior urethral valves
- Prune-belly syndrome
- Bilateral ureteropelvic junction obstruction
- Bilateral high-grade vesicoureteral reflux
- Bilateral ureterovesical junction obstruction
- Congenital urethral atresia
- Anterior urethral obstruction.

CONCLUSION

PUV is the most common cause of bladder outlet obstruction presenting as fetal ascites. The earlier it manifests in utero the worse is the fetal and neonatal prognosis early antenatal sonographic diagnosis is important in view of dismal prognosis and early termination of pregnancy[1]In my case the anomaly scan at 20 weeks did not reveal any pyelectasis. First presentation was only at 30 weeks(late onset) and there was no associated anomalies, no oligohydraminos, hence the post natal prognosis was good.

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