CYSTIC MALFORMATION OF SEMINAL VESICLE: CASE REPORT
T M Kalekar¹, G J Khadse²

ABSTRACT: Cystic malformation of the seminal vesicle is rare congenital urogenital tract anomaly. It can be isolated anomaly. It can be associated with other urinary tract anomalies. Clinical presentation may be extremely variable. In symptomatic patients surgical intervention is required so radiological features and clinical aspects are very important for patient’s management.

CASE REPORT: A 32 yr male patient was admitted with complaints of dysuria, frequency of micturition and intermittent febrile episodes since last two months. A long course of antibiotics was given to him with clinical diagnosis of urinary tract infection. At the time of admission urine examination was normal. Per rectal examination revealed a firm mass located just superior to the prostate.

Ultrasound of the abdomen and pelvis was performed which revealed absence of right kidney and hypertrophy of contralateral kidney which was also malrotated and improperly ascended. There was a cystic lesion at the anatomical site of seminal vesicle which is not seen separately, the prostate also appeared smaller in size. Transrectal ultrasound was performed for further characterization which revealed a large tubular cystic lesion entirely replacing the seminal vesicle. There was no solid component. The prostate was well visualized however it was smaller in size.

Patient was further investigated with IVU, CT scan and MRI. Intravenous urography revealed absent right kidney and compensatory hypertrophy of left kidney. Left kidney was malrotated and incompletely ascended however it showed prompt and normal excretion. The left ureter and urinary bladder were normal.

Plain and post intravenous contrast CT scan of the abdomen was performed. There was hypodense mass lesion of water attenuation in the pelvis at the bladder base more to the right side with non visualization of the seminal vesicles separately. There was no contrast enhancement.

Plain MRI was performed using dedicated pelvic coil on 1.5 T GE units. Axial, coronal and sagittal T1W and T2 W sequences were performed. It showed a tortuous tubular structure of fluid signal intensity entirely replacing the seminal vesicles. It measured approximately 8x7x6 cms in size. There was no solid component. The prostate was small in size however appeared normal in morphology and signal characteristics. The rest of the pelvic structures were normal.

So based on the imaging findings diagnosis of congenital cystic anomaly of the seminal vesicle was made.

DISCUSSION: Congenital cysts of seminal vesicles can be subcategorized as isolated cysts, cysts associated with upper urinary tract anomalies and cysts associated with autosomal dominant polycystic kidney disease. Cysts of seminal vesicle usually become apparent during second to third decade of life, perhaps related to onset of sexual activity at this age(1). Cysts are mostly smaller than 5 cm in diameter and are often discovered incidentally, but they may present with a wide variety of
symptoms such as hematuria, hematospermia, epididymitis, prostatitis, infertility and urinary tract infections. (1-4) Symptomatic cysts can be excised. Sometimes elective excision can be performed when cysts are detected prenatally or at an early age to prevent future complications.

Seminal vesicle cyst or cystic malformation can be seen on imaging as retro vesicular mass of fluid characteristics depending on imaging modality that arises from seminal vesicle cephalic to the prostate gland(5). Reported findings have been variable ranging from a cystic pelvic mass with thick walls to apparent enlargement of the seminal vesicle (3). On MRI, seminal vesicle cysts are of variable signal intensity on T1W images, are generally of fluid signal intensity on T2W images and are non enhancing after iv gadolinium administration. Increased T1 signal intensity is thought to reflect hemorrhage or an increased concentration of proteinaceous fluid (5).

Cysts of seminal vesicle are associated with ipsilateral renal agenesis or dysplasia in two third of patients, presumably reflecting maldevelopment of distal mesonephric duct and faulty ureteral budding (leading to renal agenesis or dysplasia) and atresia of ejaculatory duct (leading to obstruction and cystic dilatation of seminal vesicle (4.5)

Cystic dilatation of seminal vesicle may also occurs along with ectopic ureteral insertion into seminal vesicle, presumably due to ureteral budding occurring from the portion of the mesonephric duct destined to be the seminal vesicle(5). The ipsilateral kidney is frequently malrotated as well. Both CT and MRI can show the urinary tract anomalies, but sometimes CT cannot show the opening of ectopic ureter. MRI is better for showing the connection between the ectopic ureter and seminal vesicles. MRI is also better tool for accurately defining anatomical relationships when one is planning to excise a seminal vesicle cyst or if one is considering a difficult differential diagnosis. MRI may also help depict the opening of associated ectopic ureter. Seminal vesicle agenesis is always associated the anomalies vas deferens. Ectopia of vas deferens can be confirmed only by invasive vasography (6)

The differential diagnosis of the seminal vesicle cystic lesions include true cysts of seminal vesicles or prostate, ejaculatory duct cysts, mullerian duct cysts, prostatic utricle cysts, hydronephrotic pelvic kidney and ureteroceles. Differentiating features may include position (median, paramedian or lateral), content, associations (renal agenesis or anomalies of external genitalia) and imaging characteristics (7)

REFERENCES:

AUTHORS:
1. T M Kalekar
2. G J Khadse

PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Radiodiagnosis, B J Medical College and Sassoon General Hospital, Pune.
2. Professor, Department of Radiodiagnosis, B J Medical College and Sassoon General Hospital, Pune.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:
Dr. T M Kalekar,
11, Suvarna Housing Society,
B Gagangarina wing,
Behind Khadki Railway Station,
Pune - 411020.
Email - dr.tushar.kalekar@gmail.com

Date of Submission: 01/09/2013.
Date of Peer Review: 04/09/2013.
Date of Acceptance: 10/09/2013.
Date of Publishing: 20/09/2013