May 2009

Primary vesical varices: a cause of gross haematuria

Fauzia Ahmad Bawany  
_Aga Khan University_

Rajab Ali Ghirano  
_Aga Khan University_

Syed Raziuddin Biyabani  
_Aga Khan University_

Follow this and additional works at: [http://ecommons.aku.edu/pakistan_fhs_mc_surg_urol](http://ecommons.aku.edu/pakistan_fhs_mc_surg_urol)

Part of the [Surgery Commons](http://ecommons.aku.edu/pakistan_fhs_mc_surg_urol), and the [Urology Commons](http://ecommons.aku.edu/pakistan_fhs_mc_surg_urol)

**Recommended Citation**  
Available at: [http://ecommons.aku.edu/pakistan_fhs_mc_surg_urol/4](http://ecommons.aku.edu/pakistan_fhs_mc_surg_urol/4)
Abstract

We report a case of isolated bladder varices that manifested with sudden onset, gross hematuria, in a 44-year-old male with no co-morbid conditions. Varicosities were discovered on cystoscopy. Conditions to look out for in such a situation are discussed.

Introduction

Enlarged veins are called varicosities. Varicosities in the bladder are uncommon. They are typically associated with other conditions such as portal hypertension, shistosomiasis, pregnancy, Ataxia Telangectasia and Klippel Trenaunay syndrome.\(^1\)\(^-\)\(^5\) We report a case of bladder varices that manifested with sudden gross hematuria, in an otherwise healthy male.

Case Report

A 44 year old healthy male from Afghanistan, presented for evaluation of a single episode of gross, total haematuria with clots six months ago. There was associated dysuria. There were no triggering factors, no associated fever or any symptoms of a urinary tract infection. The haematuria resolved spontaneously, without any intervention.

He complained of a discharging perianal sinus since 3 years. He had undergone appendectomy at the age of nineteen years. He had no co morbid conditions and there was no history of hospital admissions or blood transfusions in the past. He had not been on any medications. He was a smoker, having smoked 15 pack years (one pack per day for the last 15 years). He used to consume alcoholic beverages occasionally. His family history was positive for hypertension on the paternal side. There were no known allergies. He was an architect by profession. His weight was stable in the past few years, and he had no urinary or bowel complaints.

A detailed general physical examination revealed nicotine staining of his tongue and finger tips of the right hand, secondary to his habit of smoking. He had no pallor or icterus. On examination of the chest he had a 0.5x0.5 cm sebaceous cyst in his right axilla in the mid clavicular line at the level of fourth inter-costal space. He also had a 7x7 cm soft mass most probably a lipoma, over the right hypochondrium and right iliac fossa scar of his appendectomy. Rest of the systemic examination was unremarkable. To evaluate the problem, lab tests, abdominal ultrasound and cystoscopy was done.

The laboratory investigations showed: haemoglobin; 16.3gm/dL (normal: 13.7-16.3), haematocrit; 48.4% (41.9-48.7), white blood cell count; 9.1x10^9/dL (4-10^9) platelet count: 235,000/mm^3 (150-400 x 10^3). His Fasting Blood Glucose was 89mg/dL (normal: 65-110). Liver function tests showed a total bilirubin of 1mg/dL (normal = 0.2-1.25), Gamma Glutamyl Transferase of 52 IU/L (normal = 3-50) and Alanine Amino Transferase of 56 IU/L (normal = 0-55). His serum uric acid level was 7.5 mg/dL (normal = 4.1-8), serum cholesterol was 220 mg/dL (normal = <200), triglyceride was 495 mg/dL (normal = 46-236). His serum High density lipoproteins were 31mg/dL (>35) and LDL was 110 mg/dL (<130). His serum creatinine was 0.9 mg/dL (normal=0.85-1.35). Urinalysis and urine microscopy did not reveal any abnormality and stool analysis and microscopy was essentially normal. His viral serologies showed both Hepatitis B Surface Antigen as well as Hepatitis C Virus Antibodies to be non-reactive.

Ultrasound of the abdomen revealed mildly increased echogenicity of the liver parenchyma, suggestive of fatty infiltration. No focal mass was seen. Margins of the liver were smooth and regular. The intra-hepatic biliary channels were normal with portal vein measuring 10 mm and common bile duct 3.4 mm in diameter. The rest of the organs also displayed no abnormality. There was no ascites or lymphadenopathy seen. Urinary bladder appeared normal with a pre void volume of 140 ml and post void of nil.

Flexible cystoscopy revealed a group of dilated sub mucosal veins, 5x5 cm in area, on the right lateral wall of the bladder [Figures 1 & 2]. There was no active bleeding seen during cystoscopy follow-up.

Discussion

Enlarged and tortuous veins are called varices. Bladder varicosities are rare. Of the few cases that have been reported, majority of them have been associated with severe portal hypertension. Literature search suggests an association of bladder varices with shistosomiasis, pregnancy, Ataxia Telangectasia and Klippel Trenaunay syndrome.\(^1\)\(^-\)\(^5\) The pathogenesis and management of vesical varices varies in each situation.

The possible mechanisms of formation of vesical varices: Portal hypertension is a common consequence of cirrhosis and may lead to dilated venous collaterals. Usually,
varices due to portal hypertension develop in the lower esophagus, stomach, or rectum and rarely in other parts of the digestive tract. Extra-intestinal ectopic varices are extremely rare. Bladder wall is an unusual collateral route for the splanchnic venous blood. Vesical varices may appear when the usual splanchnic bed collaterals cannot develop thus allowing venous blood to flow through the venous system of the bladder. A patient, who had portal hypertension, underwent sclerotherapy, band ligation, and abdominal surgery, causing her usual venous collaterals to be interrupted, leading to formation of bladder varices and presentation with gross haematuria.¹

In the case of bladder varices secondary to shistosomiasis, the patient also had associated portal hypertension secondary to portal thromboses, which lead to the development of ileal varices, another ectopic site. As a consequence of this, an unusual ileo-vesical shunt was created due to infiltrating ova of the parasite. The patient was treated by ileal resection.² Other than this, there are singular reports associating bladder varices with pregnancy,³ Ataxia Telangetasia⁴ and Klippel-Trénaunay syndrome.⁵

Our patient had none of the mentioned conditions, or any other co-morbidities, which could have contributed to the pathophysiology of the bladder varices and this makes our case unique. After cystoscopy, he was advised expectant treatment and annual follow ups. He returned for his first follow-up recently and reported being asymptomatic during this time period.

**Conclusion**

The presence of varicose vessels in the bladder is a very rare phenomenon. When they exist, it is usually in the presence of severe portal hypertension. In a stable patient, with no attributable etiology, we suggest expectant management.

**References**