Spontaneous Rupture of Urinary Bladder

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SUMMARY

Three cases of spontaneous rupture of the urinary bladder are presented. Histology of the bladder in two cases showed extensive schistosomiasis. The aetiology and symptomatology of spontaneous rupture of the bladder are discussed. The possible causal role of Schistosomiasis in spontaneous rupture of the bladder is discussed.

INTRODUCTION

Rupture of the Urinary bladder usually results from accidental or iatrogenic trauma. Spontaneous, as opposed to traumatic rupture is uncommon (INNIS 1961). There is a high mortality associated with this condition, due primarily to an invariable delay in the diagnosis (STONE, 1931, BACON 1943 and BASTABLE et al 1959). Three cases of spontaneous rupture of the bladder have been recently observed at Ndola Central Hospital. All presented with the features of non specific acute abdomen. The diagnosis of intraperitoneal bladder rupture was not entertained in any of these cases. Histology of the bladders in two cases showed extensive Schistosoma haematobium infection.

CASE REPORTS

Case 1
A 44 year old Zambian female was admitted with 3 day History of lower abdominal pain and inability to pass urine.

The abdomen was minimally distended, and vaguely tender with normal bowel sounds. Investigations included Hb 5gm%, Blood Urea 115mgm% and potassium 5m.eq/L. Catheterization drained 500ml of urine. On the 3rd Hospital day the patient developed marked hypotension and tachycardia. The abdomen was more distended with increased tenderness. A laparotomy performed on the same day revealed generalized purulent peritonitis with free peritoneal fluid and the rupture at the dome of the bladder. The Catheter was in the peritoneal cavity. The perforation was repaired. The patient expired on the 2nd post OP Day. Histology of the bladder showed extensive Schistosomiasis.

Case 2
A 39 year old Zambian male was admitted in a poor condition with the History of not being able to pass urine for “sometime”. The abdomen was minimally distended. A tender mass was present over the suprapubic region. Investigations included Hb 10mg%, Blood Urea 120mg% and potassium 5.8eq/L. Catheterization was done with slight difficulty due to stenosis of external meatus. Only a few ML of blood stained fluid was drained. Patient’s condition failed to improve on symptomatic measures and expired 24 hours after hospitalization. Autopsy showed generalized purulent peritonitis with free fluid and a large tear on the dome of the bladder. The suprapubic mass was formed by the Oedematous Omentum. Histology of the bladder showed “Non specific Cystitis”.

Case 3
A 53 year old Zambian male was admitted with 2 day history of lower abdominal pain and extreme urgency of micturation with passage of small amount of blood stained urine of 2 days duration. Abdomen was moderately distended with suprapubic tenderness. Investigations included Hb 10mg%, Urea 70mgm%. The patient was initially treated conservatively. Due to progressive tenderness an exploratory laparo-
tomy performed on 2nd day, revealed purulent peritonitis, free fluid and a small perforation of the bladder dome. The perforation was repaired and after a protracted post operative course he made a complete recovery, the blood urea taking to 26ngms%. Histology of the Bladder showed severe Schistosomiasis.

SYMPTOMATOLOGY

The symptoms of intraperitoneal rupture of bladder are variable. Lower abdominal pain is always present but the abdominal signs are minimal. The patient is usually unable to pass urine, whilst in the case of a smaller rent extreme urgency of urination with passage of small amount of urine may be the presenting feature.

Left untreated peritonitis develops due to the chemical irritation of the extravasated urine. Later bacterial infection supervenes due mainly to transmigration of bacteria along the chemically damaged bowel wall. Shortly after the peritoneal extravasation of urine the phenomenon of peritoneal self dialysis sets in (WOOK KO et al 1964). The extravasated urine acting as a dialyzate leads to the loss of sodium and chloride and partial retention of urea and potassium. This condition contributes considerably to the high mortality associated with spontaneous rupture of the bladder (LICHTENHELD and LANCASTER 1962).

DISCUSSION

Although spontaneous rupture of normal bladders have been reported (HERSHAM and ALLEN 1954 YARWOOD 1959), it appears that this is more likely to occur in a diseased bladder even in the absence of outlet obstruction (THOMPSON et al 1965, LICHTENHELD and LANCASTER 1962). The rupture usually occurs at the bladder dome. Bastable et al (1959) by over distension of cadaver bladders have demonstrated that the dome is the weakest part of this organ.

Due primarily to the non specific nature of the initial symptoms, the diagnosis is often delayed. Urethral catheterization and immediate return of a large amount of urine does not negate the diagnosis of the ruptured bladder.

Urine in such a situation may be withdrawn from the peritoneal cavity as a result of the catheter traversing the rent in bladder. A rising blood urea due to its absorption through the peritoneum and an apparent absence of urinary output often leads one to the erroneous diagnosis of acute renal failure, which results in a further delay in the surgical correction of the lesion. Preoperatively the diagnosis in a suspected case can be made with certainty by retrograde cystogram or cystoscopy.

It appears that the association of bilharziasis with spontaneous rupture of the bladder has not been given due attention. Only a single case has been reported in the English Literature (ELEM - 1977). It is probable that in areas where bilharziasis is endemic the incidence of ruptured bladder is much higher than the literature tends to indicate. Awareness, a high index of clinical suspicion and employment of cystogram and cystoscopy in cases of obscure acute abdomen will reveal its true incidence. Early diagnosis and prompt surgical correction alone can improve the unacceptably high mortality, associated with spontaneous rupture of bladder.

REFERENCES


