Delayed Dehiscence of Repaired Bladder Rupture

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Key Word
Bladder rupture

Abstract
A case of dehiscence of a previously repaired bladder rupture occurring 3 years after the original repair is reported. There was no obvious predisposing cause to this event. As this occurrence is very rare, it brings into question the need for long-term review of patients with conservatively treated bladder rupture.

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Introduction
Bladder rupture following blunt injury is relatively common. Case reports of spontaneous bladder rupture have usually been associated with some underlying pathology [1]. This case could be the first report of recurrent bladder rupture unassociated with trauma following previous repair.

Case Report
In 1986, a 32-year-old man underwent laparotomy for blunt abdominal trauma causing peritonitis. An intraperitoneal 5-cm tear of the dome of the bladder was discovered and repaired by a general surgeon in two layers using chromic catgut. The urethral catheter was removed on the 8th post-operative day, and the patient made an uneventful recovery and was discharged. He was admitted 17 months later with generalised abdominal pain having been unable to pass urine for 12 h. He had drunk 5 pints of beer that evening. Urethral catheterisation drained 300 ml of residual urine. His pain settled rapidly on bladder drainage and the catheter was removed 2 days later with no recurrence of the pain. He was therefore discharged without further investigation.

He was readmitted in December 1989 with generalised abdominal pain, with no history of trauma but having had 4 pints of beer prior to the onset of the pain. Investigations showed uraemia and ‘ascites’ on ultrasonography. Once again, his pain settled on catheter-isation, and a cystogram (fig. 1) showed a diverticulum of the dome of the bladder from which there was an intraperitoneal leak of contrast.

Cystourethroscopy did not show any evidence of outflow obstruction and confirmed a diverticulum in the area of the previous repair.

Laparotomy revealed a diverticulum of the bladder dome surrounded by fibrinous exudate. Excision of the diverticulum of the bladder dome was followed by full-thickness bladder repair in two layers using polyglycolic acid sutures. When reviewed in the outpatient department 6
weeks following the operation, he was asymptomatic with normal blood urea, electrolytes and creatinine and a peak urinary flow rate of 52 ml/s. Histology of the diverticulum showed a diverticulum lined by normal urothelium which dips into the muscle coat.

Comments
Spontaneous perforation of the bladder has been previously described usually associated with a pathological condition of the bladder or outflow tract [1] or following recurrent albeit trivial trauma [2]. A recent paper has also reviewed the literature on spontaneous rupture and found 20 cases associated with substance abuse (mainly alcohol). However, the paper does not define what amounts to substance/alcohol abuse [3]. In this case, the only precipitating factor appears to have been a moderate intake of alcohol.

It is presumed in this case that the reason for the acquired diverticulum was a previous inadequate repair. This case also raises the question of what length of follow-up we should accept in assessing the results of any treatment of bladder rupture particularly in view of the recent trend in treating some cases of extraperitoneal rupture by more conservative means [4].

References

L. Lynch/Stewart
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