SPONTANEOUS RUPTURE OF THE BLADDER

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Spontaneous rupture of the bladder is the term applied to rupture occurring without trauma, resulting from either pathological changes in the wall of the bladder, or obstructive lesions at the neck of the bladder irrespective of the presence or absence of pathological changes in the bladder wall.[1]

Stone[1] reported that Pierus recorded the first case in 1279 and Johnstone the next one in 1773.

Bastable[2] reviewed 71 cases of spontaneous rupture of the bladder in 1959, 66 of which were intraperitoneal and 5 extraperitoneal. In the intraperitoneal cases, tuberculosis was the commonest pathological lesion of the bladder wall associated with the rupture, while in those in which obstruction was a factor, neurological lesions and urethral obstruction from an enlarged prostate or a stricture, were the commonest associated conditions. There was also a miscellaneous group which included 4 puerperal patients, 1 patient in the postoperative phase after a herniorrhaphy, and 1 patient with bronchopneumonia.

To my knowledge, since 1959 there have been 6 further reports in the literature of spontaneous rupture of the bladder,[3-8] 2 of which are in journals not available to me.[5,6]

I wish to report a further case of spontaneous rupture of the bladder occurring during the puerperium. There were associated neurological signs.

CASE REPORT

A Bantu female, Grissel, aged 22 years, was admitted to Baragwanath Hospital with a history of having given birth to a stillborn infant at home after a protracted labour, 4 days before admission.

The patient had felt reasonably well until 48 hours after confinement, when she suddenly developed severe abdominal pain. This was a constant pain and was situated in the lower abdomen. She had vomited several times since the onset of the pain. She had not had a bowel action for 8 days before admission, and had not passed urine for the 4 days since her confinement.

Examination revealed a young female in obvious distress, pulse rate 120 per minute and blood pressure 120/90 mm.Hg.

On examination of the abdomen, there was marked distension, with some generalized tenderness. There was dullness in both flanks, and shifting dullness was present. Bowel sounds were absent.

The leucocyte count was 18,000 per c. mm., and the haemoglobin level was 14·9 G. per 100 ml., the blood-urea level was 142 mg. per 100 ml., the serum-potassium level 5·7 mEq./litre, serum sodium 120 mEq./litre, CO_2 content 15·8 mEq./litre and chlorides 90 mEq./litre.

The 'ascitic fluid' was tapped, giving approximately 2 litres of clear fluid with an SG of 1015. A Foley's catheter was introduced into the bladder, producing 100 ml. of faintly blood-stained urine.

I saw the patient for the first time after the paracentesis. She was somewhat dehydrated and in shock, with a blood pressure of 90/60 mm. Hg. The abdomen was moderately distended, and the uterus was enlarged to about the size of an 18 weeks' pregnancy. There was generalized tenderness with guarding, but no rigidity; there was still some free fluid in the peritoneal cavity and the bowel sounds were absent. Rectal examination was non-contributory.

Laparotomy was decided upon with a tentative diagnosis of ruptured bladder; torsion of an ovarian cyst was also considered.

Operation

A right lower abdominal paramedian incision was made. There were at least 2 litres of clear fluid in the peritoneal cavity. A hole, 3 cm. in size, was found in the vault of the bladder; this was closed in 2 layers with plain catgut. The urine was sucked out of the peritoneal cavity and the abdominal wound was closed in layers without drainage. The bladder was drained by an indwelling Foley's catheter.

Postoperative Course

Tetracycline was given intravenously in a drip for 3 days postoperatively and this was followed by oral tetracycline for a further 2 days.

On the second postoperative day the patient complained of pains in both legs; it was found that she had bilateral foot drop, more marked on the left side. The ankle jerks were absent and there were areas of anaesthesia over the saddle area and the posterior aspect of the left upper thigh, in the region of the gluteal fold. The patient was treated for this condition with physiotherapy in the form of active and passive exercises. Light plaster-of-Paris splints were applied each night.

The Foley's catheter was removed for the first time after 12 days, but it was found that the patient passed urine with great difficulty and there was a large volume of residual urine present. Culture of the urine revealed a growth of *E. coli* sensitive to streptomycin, 'kantrex' and polymyxin. A cystometrogram revealed an atonic bladder.

Continuous drainage of the bladder was continued for 3 months, the catheter being changed every 10 days. The cystometrogram was repeated twice and showed gradual improvement in the tone of the bladder. Urine cultures were done at intervals and the infection was treated with the specific antibiotic indicated, both systemically and by instillation into the bladder.

Attempts were made to remove the catheter on several occasions, but it was found that the patient was unable to empty her bladder completely. Only after a full 3 months of continuous drainage had the bladder tone returned almost to normal and the residual urine became less than 60 ml.

The neurological signs in the legs improved gradually, the anaesthesia disappearing first. The foot drop remained, but also improved.

On discharge from hospital the patient was walking well, having been fitted with orthopaedic boots with side irons and a back-stop.

DISCUSSION

Causation

Postpartum intravesical photography[9] and cystoscopy[10] have both demonstrated that definite changes occur in all deliveries; these vary from slight oedema in mild cases to massive areas of ecchymosis, frank haemorrhage and marked oedema, these changes being most obvious in the trigone and floor of the bladder. The factors that determine the degree of trauma are the length of time the presenting part is in contact with the bladder and the amount of force exerted on the area. The degree of oedema in the trigone

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may be such as to cause retention of urine. Derangement of nerve function postpartum may contribute to retention of urine.[10]

In the present case there was evidence of nerve-function derangement in that the patient had bilateral foot drop and anaesthesia in sacral segments 2 and 3.

The mechanism of obstetric foot drop is not known with any certainty.[12] The usual explanation given is that a form of neuropraxia is produced by pressure on the lumbosacral trunk, arising from lumbar segments 4 and 5, where it crosses the pelvic brim. Involvement of this nerve will result in paralysis of the peronei muscles; the anaesthesia in this case showed a more widespread nerve involvement than would have resulted from pressure on the lumbosacral nerve only. The sacral segments involved are also the nerve roots through which the parasympathetic fibres pass to the detrusor of the bladder.

Pressure on sacral segments 2, 3 and 4 could have resulted in the anaesthesia and could have disturbed the parasympathetic outflow to the bladder, thus contributing to the urinary retention. An added factor in rupture of the bladder in the postpartum period is the tendency of the bladder after delivery to be hypotonic, with decreased sensation, reduced muscle tone and increased capacity.[11]

Diagnosis

Pain and haematuria or retention of urine are the usual symptoms of intraperitoneal rupture of the bladder, the pain usually coming on in association with some act which raises the intra-abdominal pressure, such as defaecation, micturition or the lifting of some heavy object.

The findings in most cases reported are of generalized peritonitis with tenderness and rigidity, although Bastable *et al.*[2] reported that in 10 of the 71 cases that they reviewed no abdominal tenderness or rigidity was present. I have noted in 3 previous cases of intraperitoneal rupture of the bladder following trauma, that abdominal tenderness may be minimal and rigidity may be completely absent. One case had obvious free fluid in the peritoneal cavity following trauma, but abdominal tenderness and rigidity were not present. The diagnosis of ruptured bladder was made only after it was noticed that palpation of the abdomen caused 3 litres of urine to drain out of a Foley's catheter, which had previously been introduced into the bladder.

Rigidity is dependent on the presence of urinary infection. Where there has been trauma to the abdomen it is unlikely that there is infected urine, so that signs of peritoneal irritation may be completely absent in a patient who has a ruptured bladder, particularly in the first 24 hours. Blood in the peritoneal cavity may similarly produce little or no peritoneal irritation, as has been observed in cases of traumatic rupture of the spleen.[13]

The diagnosis of intraperitoneal rupture of the bladder is usually suspected on obtaining blood-stained urine after passage of a catheter. A cystogram will usually confirm the diagnosis, but the patient may have to be tilted into the Trendelenburg position to allow loops of bowel, which may be occluding a hole in the bladder, to fall away. Cystography is usually not necessary in the diagnosis of intraperitoneal tears, but is of more use in the demonstration of extraperitoneal tears. Cystoscopy may be time-consuming and may miss small tears.

Attempting to recover a measured volume of fluid, after injection through a catheter, may give false negative results and is inferior to cystography.

Treatment

The first laparotomy with suture of a bladder hole was performed by Willett in 1876, but the patient died.

Laparotomy with two-layer closure of the bladder wall and an indwelling Foley's catheter for 10 - 14 days is the treatment of choice.

Stone[1] stated that it was unnecessary to suture the rent in the bladder, provided a large cystostomy drain was inserted. This method is, however, only applicable to the extraperitoneal tears and those in the region of the trigone.

In cases of intraperitoneal rupture of the bladder, the rent is usually situated in the vault of the bladder in the midline, which has been shown to be a site of weakness developmentally, and it is furthest from the support of surrounding structures.[2]

The prognosis of intraperitoneal rupture of the bladder is favoured by the absence of associated disease of the bladder and the absence of infection. The mortality for spontaneous rupture was given as 45·4% in 1931[1] and 47% in 1959,[2] two-thirds of the mortality in 1959 being attributed to the presence of tuberculosis, carcinoma of the bladder or an enlarged prostate.

SUMMARY

A case of spontaneous rupture of the bladder, occurring in the puerperium and associated with neurological signs in the lower extremities, is described. The aetiology of the condition is discussed and the literature briefly reviewed.

OPSOMMING

'n Geval van spontane ruptuur van die blaas word beskryf. Die geval het na 'n moeilike kraamgeval in 'n Bantoevrou voorgedoen. Daar was gepaardgaande neurologiese letsels. Die meganisme van die blaasruptuur word verklaar en melding word kortliks gemaak van vorige gevalle wat in die literatuur verskyn het.

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