NON-PUERPERAL UTERINE INVERSION - A CASE REPORT

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ABSTRACT

A rare case of non-puerperal uterine inversion caused by a large fundal leiomyoma in a 36-year-virgo intacta resulting in intractable haemorrhage was reported. After a myomectomy, attempts to reduce the inversion vaginally by transecting the anterior and posterior cervix was unsuccessful and a laparotomy was performed. The inversion was successfully corrected with return of normal function of the uterus.

Keywords: *uterine inversion, non-puerperal, fundal leiomyoma*

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CASE REPORT

A 36-year-old virgo intacta was admitted with severe dysmenorrhoea and a very heavy menstrual flow.

She had a history of severe spasmodic dysmenorrhoea for many years, worse on days 2 and 3 of her menses but occasionally lasting throughout the menstrual period. It was usually relieved by analgesics. She had also been prescribed Norethisterone for her heavy menstrual flow for the last 3 years. On this occasion, her menstrual flow was heavier and prolonged. Her menstrual pads were thoroughly soaked in the blood which needed changing every half an hour. She also passed out copious amounts of blood clots.

She had no bowel complaints. There was delay in initiation of micturition and the flow was intermittent. She also had strangury.

On examination, her general condition was fair but she was distressed by her lower abdominal pain. She was afebrile and her vital signs were stable. She was markedly pale. The rest of the physical examination was unremarkable. Abdominal examination was normal and no masses were felt.

On rectal examination, the uterus was thought to be enlarged.

Her haemoglobin was 6.1g/dl. Additional investigations confirmed an iron deficiency anaemia. Platelet count was 395 x 10^9/l. Urine microscopic examination and culture were normal.

A transabdominal pelvic ultrasound scan reported the uterus as axial and enlarged with a "fibroid" measuring 7.8 x 6.3 x 7.6 cm in the corpus. Both ovaries were demonstrated and were normal. No other masses were seen in the pouch of Douglas.

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Fig 1 - Fundal myoma being shelled out.

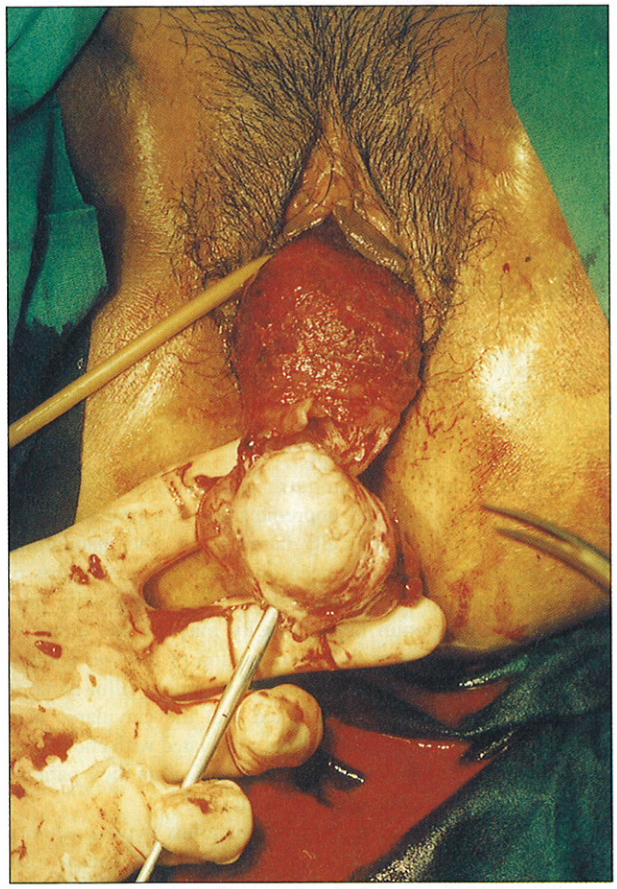


Fig 2 - Round ligaments seen from above

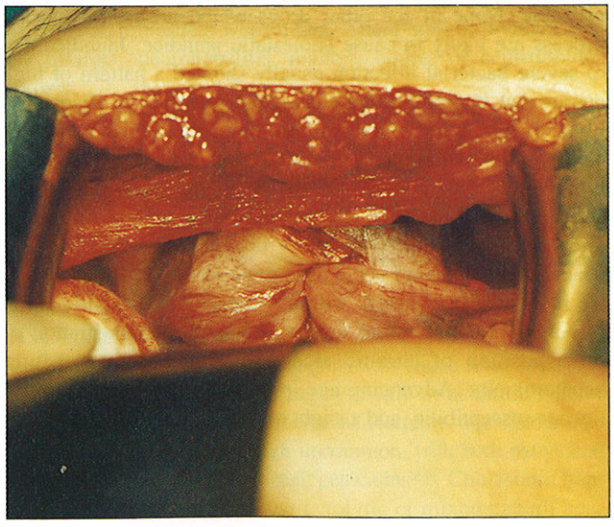


Fig 3 - Uterus inverted back onto itself after an incision was made in the anterior uterine wall.

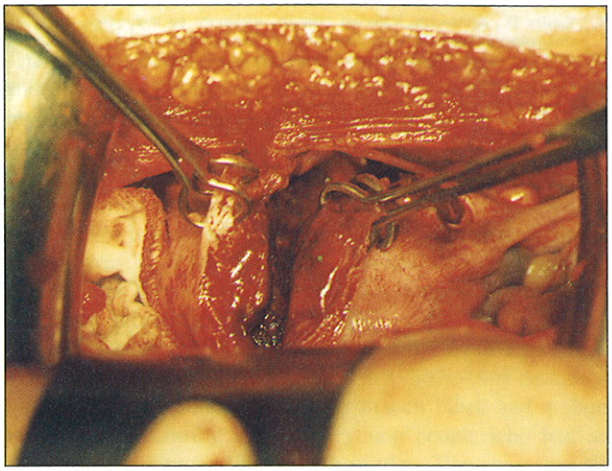
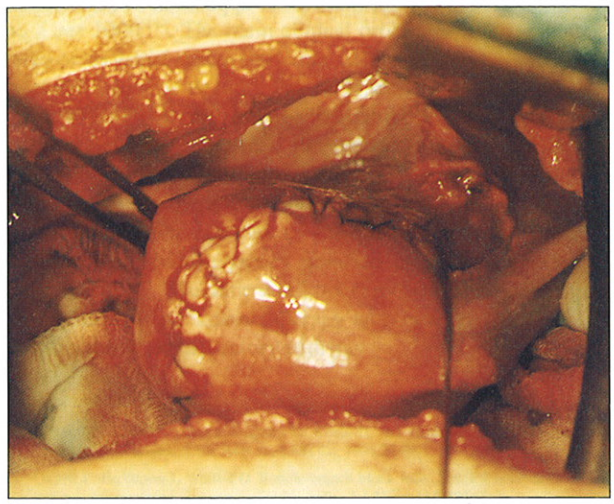


Fig 4 - Final result after repair



Despite a dose of intramuscular progesterone 150 mg on the first and second day of admission, she continued to bleed heavily. An examination under anaesthesia and a dilatation and curettage of the uterus was performed.

Operation

1. Examination under anaesthesia

Bimanual examination revealed a 4 cm diameter mass in the vagina. The cervix could not be felt beyond the mass. When the mass was pulled gently with a pair of volsellum forceps, it prolapsed out of the introitus. The diagnosis of a fundal fibroid with a uterine inversion became evident.

2.Vaginal operation to reduce uterine inversion

The patient remained in the lithotomy position and her abdomen and perineum was cleaned.

The fundal myoma was shelled out vaginally from the uterine wall via a 3 cm vertical incision as shown in Fig 1. Haemostasis was secured with 2'0' catgut sutures.

Laparoscopy was performed to confirm the diagnosis of uterine inversion and an attempt was made to reduce the uterus by pushing the fundus from below with a swab stick. This was augmented with hydrostatic pressure as described by O'Sullivan. Reduction failed even after transecting the cervix at 6 and 12 o'clock positions.

3. Laparotomy

A Pfannanstiel incision was made and the abdomen was entered in layers. The view from above is shown in Fig 2 where it shows the two round ligaments entering into a depression.

An incision was made on the anterior wall of the uterus extending from the cervix to the fundus as shown in Fig 3. This finally enabled the uterus to be inverted back onto itself and be replaced in the pelvis. The cervix was repaired with 2'0' chromic catgut sutures and the uterine incision repaired in two layers with 2'0' chromic catgut sutures. The uterine serosa was repaired with 3'0' prolene sutures. The final result is shown in Fig 4.

Recovery

Post-operative recovery was uneventful and she was discharged on the sixth post-operative day.

Histology reported a benign leiomyomata, measuring 6 x 4 x 3 cm with cut section showing areas of haemorrhage.

She was reviewed 4 weeks later. She was well except for some mild staining per vaginam. She did not have any urinary complaints. Her periods returned subsequently and her periods were lighter.

DISCUSSION

This was a rare and unusual case in that uterine inversion rarely occurs, especially so in a virgo intacta. Only 2 cases of non-puerperal chronic uterine inversion have been described in a computer based international medical literature search (Compact Cambridge Medline 1966-1991) over the last 25 years - Silveira PL & Galba AJ (1976)[1] and Vasilev D (1986)[2] - both of which were not published in the English language.

The aetiology of chronic inversion of the uterus are puerperal, inversion combined with the presence of a sessile submucous fundal myoma and inversion associated with an adenocarcinoma of the endometrium or a sarcoma. In this instance, the most probable aetiology was the fundal submucosal fibroid.

The history of this patient suggests that an acute uterine inversion precipitated the worsened symptoms. Unlike puerperal inversion where the timing of delivery is evident, the diagnosis of whether a non-puerperal uterine inversion was pre-existing before the diagnosis is made is a difficult one.

The ultrasound scan on this patient on admission did not allow the diagnosis to be made. As the uterine prolapse could not be reduced by the standard methods and an incision on the uterine wall was needed, the treatment was essentially that for a chronic uterine inversion. The two surgical methods of treatment of chronic inversion of the uterus are performed vaginally[3]. Kustner suggested that the inverted fundus is grasped with a single-bite volsellum and drawn forwards and upwards. A transverse incision is made, as long as possible, in the posterior vagina wall near the junction of the vagina and cervix. The pouch of Douglas is then opened. A finger or a Hegar dilator is now passed through the opening made into the pouch of Douglas and introduced along the hollow of the inverted uterus. The endometrium and then the muscle and peritoneal layers of the inverted uterus are incised along the whole length of the uterus. The edges of the cut cervix are now drawn apart with volsellum forceps. The inverted uterus is then turned inside out and the inversion corrected. Subsequently, the incised posterior wall of the uterus is repaired with interrupted catgut sutures, and lastly the wound in the posterior vaginal wall is closed. In Spinelli's operation, the uterovesical pouch is opened by an anterior colpotomy and the uterus split along its anterior wall. Otherwise the technique is similar to Kustner's operation.

The disadvantage of the vaginal approach is that access is limited and there may be difficulty in the repair of the uterine incision.

In the present case, the abdominal approach was adopted for easier access so that meticulous apposition of the incision could be achieved.

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ANSWER TO ELECTROCARDIOGRAPHIC CASE

Diagnosis: Wolff-Parkinson-White syndrome

DISCUSSION

The 12-lead electrocardiogram shows Q waves in leads II, III and aVF. The immediate differential diagnosis is an inferior myocardial infarction and possibly a posterior myocardial infarction as the R/S ratio in V2 was > 1. It may however occur as a normal variant but this is usually associated with small q waves with a duration < 0.04 second and the depth of the q waves is less than 25% of the R wave[1]. The presence of delta waves in V4-6, I and aVL however suggest that the diagnosis is due to preexcitation associated with the Wolff-Parkinson-White syndrome.

The electrocardiographic changes of the Wolff-Parkinson-White syndrome was first described in 1930[2]. They described the syndrome as functional bundle branch block and a short PR interval occurring in healthy young people who had an intermittent supraventricular tachycardia. The characteristic ECG changes are a short PR interval of less than 0.12 second duration, a widened QRS complex and a delta wave (slurred and thickened proximal limb of the QRS complex). They are also associated with secondary ST segment and T wave changes which are secondary to the abnormal ventricular excitation[3]. The Wolff-Parkinson-White syndrome may obscure or simulate a variety of disorders. It may simulate or obscure myocardial infarction. Left free wall accessory pathways may simulate a posterior myocardial infarction or a high lateral infarction because of tall R waves in V1 and q waves in I and aVL. The posteroseptal accessory pathways simulate inferior myocardial infarction, as in this patient. The estimated prevalence of the Wolff-Parkinson-White syndrome varies from 0.1 to 3 per 1000[4-6]. The main clinical problem is the association with paroxysmal supraventricular tachycardia and possible risk of sudden cardiac death[7]. The therapeutic options include intermittent drug therapy of the infrequent episodes of supraventricular tachycardia to prophylactic drug therapy for patients with recurrent tachycardia. In patients who do not respond to drug therapy or do not want life long drug therapy and those at increased risk of sudden cardiac death, curative therapy with radiofrequency ablation or surgical therapy is now possible[8]. The patient did not respond well to drug therapy and underwent curative therapy with radiofrequency catheter ablation of the accessory pathway.

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