Acute testicular ischemia following manual reduction of inguinoscrotal hernia

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Abstract

Testicular ischemia caused by inguinal hernia repair, and even the presence of the hernia itself, has been recognized in the medical literature, with the latter more commonly in children, but such an event after manual reduction has never been reported before. We present the case of a 67-year-old man who presented to the emergency department with a painful left groin lump. A left inguinoscrotal hernia was diagnosed and reduced “en masse” with manual pressure at the bedside. The patient was discharged but developed acute-onset left scrotal pain as soon as he got home and then re-presented 2 days later with increasing severity of the pain and swelling ever since the hernia reduction. On examination, he was febrile, with a hard, tender, and swollen left testis. Serum inflammatory markers were elevated. Conservative management with intravenous antibiotics and analgesia was commenced. An ultrasound of the testes demonstrated lack of Doppler flow to the left testis, suggestive of acute ischemia. Three days later, there were persistent temperature spikes and significant pain; therefore, the patient underwent an acute left scrotal exploration where a necrotic, black left testis was discovered and excised. He was discharged on the 1st postoperative day; histological analysis confirmed testicular infarction.

Keywords: Inguinal hernia, manual reduction, testicular ischemia

INTRODUCTION

Inguinal hernias are common, with a lifetime risk of 27% in men and accounting for 75% of all abdominal wall hernias.[1] Patients often present in the emergency setting with painful or irreducible inguinal hernias, and often, clinicians will attempt reduction at the bedside, especially if it is preferable to avoid an operation. Such hernia reduction “en masse” has been reported to cause severe complications, with persistently incarcerated or strangulated bowel within the hernia postreduction widely reported in the medical literature.[2,3] The effects of this action on the testicular vasculature however are much rarer, and we describe an unusual case of manual reduction of an inguinoscrotal hernia, resulting in acute testicular ischemia.

CASE REPORT

A 67-year-old male with a background of hypertension presented to the emergency department with a painful left groin lump. A left inguinoscrotal hernia was diagnosed and reduced “en masse” with manual pressure at the bedside.
The patient was discharged but developed acute-onset left scrotal pain as soon as he got home and then re-presented 2 days later with increasing severity of the pain and swelling ever since the hernia reduction. On examination, there was a temperature of 38°C, with a hard, tender, and swollen left testis. Serum inflammatory markers were elevated with a white cell count of $21 \times 10^9$/$L$ and C-reactive protein of 157 mg/L.

The initial diagnosis at this point given the unusual history was of an acute left epididymo-orchitis, and conservative management with intravenous ciprofloxacin and analgesia was commenced. An ultrasound scan of the testes demonstrated lack of Doppler flow to the left testis, suggestive of testicular ischemia; the right testis was normal. Three days later, the patient continued to spike temperatures and was still complaining of significant pain. He therefore underwent an acute left scrotal exploration and left scrotal orchidectomy, where a necrotic, black, nontorted left testis was discovered and excised.

The patient was discharged on the 1st day postoperatively with little pain. Subsequent histological analysis confirmed acute testicular infarction.

**DISCUSSION**

Testicular atrophy, and even infarction, postinguinal hernia repair is a recognized complication, due to injury to the spermatic cord vessels at the time of surgery.[4] Testicular ischemia caused by an inguinal hernia itself causing vascular compression has been reported in an adult patient, with subsequent testicular salvage possible through operative intervention,[3] and one study looking at testicular ultrasonography in men with unilateral inguinal hernias found significantly higher testicular volumes on the ipsilateral side compared to the contralateral testis, with the mechanism attributed to intermittent mechanical compression on the spermatic cord within the inguinal canal.[5] This phenomenon has been more commonly described in infants and children with inguinal hernias with a reported incidence of 5%-34%,[6,7] which has been found to be higher in those with incarcerated hernias.[4]

As alluded to in the introduction, the potential effects on the bowel of en masse reduction of inguinal hernias are well recognized. This clinical maneuver has been described to have caused testicular infarction in a 2-month-old infant, resulting in orchidectomy.[10] However, to our knowledge, this is the first reported case in an adult patient and is likely to have resulted from manual compression to the testicular blood vessels in the spermatic cord as it ran through the inguinal canal. Unfortunately, testicular loss occurred in this case, possibly because the rarity of acute ischemia in this setting. Had the patient re-presented earlier, then their case may have been treated as an acute testicular torsion, even with the history of the hernia reduction, and undergone more urgent surgical intervention. Such a complication, albeit rare, should therefore be considered when severe testicular pain occurs following inguinal hernia reduction.

**CONCLUSION**

Testicular ischemia following en masse reduction of an inguinoscrotal hernia is a rare, but serious, event which clinicians should be wary of before attempting reduction of incarcerated inguinal hernias en masse, in addition to the recognized bowel complications. If patients present atypically through the development of severe testicular pain postreduction, then acute testicular ischemia secondary to the maneuver should be considered. One should have a low threshold for operative intervention, particularly in the presence of ongoing severe pain; where the symptoms have presented more than 48 h after their onset, imaging in the form of ultrasound scan may be considered to confirm such a diagnosis.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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