RESIDENT'S CORNER

Testicular infarction as a sequela of inguinal hernia repair

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Background: Testicular atrophy and necrosis as a result of ischemic orchitis is a well-established complication after inguinal hernia repair. We reviewed four patients with a recent history of inguinal hernia repair and subsequently developed ischemic orchitis to evaluate management options to provide symptomatic relief and prevent testicular atrophy.

Case series: The first patient underwent loosening of inguinal hernia mesh for scrotal pain and decreased arterial testicular flow 8 hours after inguinal hernia repair. The second patient developed ischemic orchitis

Introduction

Ischemic orchitis and subsequent testicular atrophy are dreaded complications after open or laparoscopic inguinal hernia repair. The incidence of testicular atrophy after open inguinal herniorrphaphy is 0.03%-0.5%, regardless of surgeon experience.^{1,2} Recurrent inguinal hernia repair has a higher incidence of testicular atrophy and necrosis, with a rate of 0.8%-5%.³ Ischemic orchitis is more common after open rather than laparoscopic procedures especially after large indirect and recurrent inguinal hernias possibly due to greater manipulation of the spermatic cord.³⁻⁴ Either testicular atrophy or orchiectomy has been reported as a result of ischemic orchitis after inguinal hernia repair.⁵ To our knowledge, reversible ischemic orchitis after inguinal hernia repair has not been reported in literature. In this case series, we discuss four patients who developed ischemic orchitis after inguinal hernia repair and review their management to evaluate better treatment options to prevent testicular atrophy and provide symptomatic relief.

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Address correspondence to Dr. Lei Chu, Department of Urology, University of Pittsburgh School of Medicine, 3471 Fifth Avenue, Suite 700, Pittsburgh, PA 15213-3232 USA 20 hours after inguinal herniorrphaphy and required removal of mesh. Neither patient had testicular atrophy at their follow up visit. The third patient underwent orchiectomy for testicular pain and intraoperative findings of infarction. The fourth patient never experienced pain but showed testicular atrophy 6 months after inguinal hernia repair and was managed expectantly.

Conclusion: In our series, testicular ischemia was reversed in two of the four patients with mesh loosening or removal. Prompt evaluation and exploration of suspected ischemic orchitis after inguinal hernia repair may allow symptomatic relief and prevention of testicular atrophy.

Key Words: inguinal hernia, testicular infarction, ischemic orchitis

Case presentations

The first patient is a thirty-one-year old male with acute right testicular pain 8 hours after an open Lichtenstein direct inguinal hernia repair. Ultrasound demonstrated no arterial blood flow and only a small amount of venous flow. The patient was taken emergently to the operating room for scrotal exploration. Significant cord edema was present directly at the site of the repair. After the mesh around the cord was loosened, strong pulsatile testicular flow was demonstrated over the cord with an intraoperative Doppler ultrasound. Scrotal pain improved significantly postoperatively. At 1 month follow up, the patient had no recurrence of his inguinal hernia and no testicular atrophy.

The second patient is a twenty-one-year old male presenting to the emergency room with right testicular pain 20 hours after open Lichtenstein inguinal hernia repair with mesh. Doppler ultrasound documented decreased blood flow to the right testicle. The patient was taken emergently to the operating room for scrotal exploration; cord edema and thrombosed testicular veins were found. The mesh was removed and his scrotal pain improved significantly after surgery. The patient had immediate recurrence of his inguinal hernia postoperatively and hernia repair was planned at a later date. At 2 week postoperative follow up, he had no testicular atrophy.

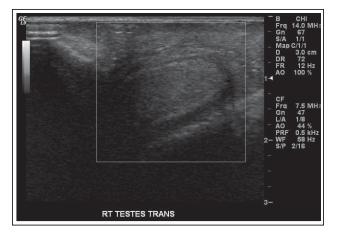


Figure 1. Atrophied testis with no discernible blood flow.

The third patient is a sixty-seven-year old male who presented to our office for benign prostatic hypertrophy management 6 months after open Lichtenstein mesh repair of right direct and indirect inguinal hernia. Physical exam revealed a new finding of an atrophied and firm right testicle. Doppler ultrasound of the scrotum showed a much smaller, 1.9*0.9 cm, and diffusely hypoechoic right testis with no discernible blood flow, compared to the left which measured 3.9 *2.6 cm; this is consistent with an old infarction of the right testis, Figure 1. As this patient had no history of testicular pain after inguinal hernia repair, he was managed expectantly.

The fourth patient is an eighty-three-year old male presenting to the emergency room with right scrotal pain 4 days after a Bassini repair of a right indirect inguinal hernia. Scrotal ultrasound demonstrated right testicular infarction and a small complex right hydrocele. The patient underwent emergent right scrotal exploration. An infarcted right testis was found intraoperatively and he underwent simple orchiectomy. His pain improved after orchiectomy.

Discussion

Testicular ischemia and necrosis have been reported after both open and laparoscopic inguinal hernia repair and usually present 2 to 3 days postoperatively.¹⁻⁴ Acute thrombosis of the pampiniform venous plexus rather than arterial injury is the classical etiology for testicular ischemia.⁵ Cautery excision of the hernia sac, extensive dissection of the spermamatic cord, mobilization of the testis from the scrotum, and inguinal hematoma are noted causes of venous injury and thrombosis.⁶⁻¹⁰ Symptoms of ischemic orchitis include pain, tenderness and swelling of the testis and spermatic cord; however, clinical findings can be unreliable.

Scrotal ultrasound with Doppler has been shown to have high sensitivity and specificity in evaluating testicular blood flow.¹¹ Evaluating intratesticular blood flow requires slow flow Doppler technique as the blood vessels in testis are small and blood flow is slow. The optimal Doppler settings should be set using the normal testis as a control. Color flow Doppler has an overall sensitivity of 86% and a specificity of 100%.¹¹ One of the intraoperative findings that can distinguish infarction from reversible ischemia is the lack of bleeding in a deep incision of the testis within 10 minutes. Lack of bleeding confirms infarction rather than reversible ischemia. Infarction is treated with orchiectomy for both pain control and to preserve fertility as leaving a necrotic testis in situ can lead to autoimmunization against spermatozoa.12

In our series, we were able to reverse ischemic orchitis in two of the four patients by loosening or removing mesh and neither patient showed testicular atrophy at follow up. Symptomatic relief and prevention of testicular atrophy may be possible with prompt evaluation and exploration of suspected ischemic orchitis.

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