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Testicular Necrosis Following Inguinal Herniorrhaphy: A Case Report

Takuya Yashima [1], Yoshihiro Nakagami [1], Kazuhiko Oshinomi [1], Masakazu Nagata [1], Takashi Fukagai [1]

1. Department of Urology, Showa Medical University, School of Medicine, Shinagawa, JPN

Corresponding author: Takuya Yashima, b18f-2332@med.showa-u.ac.jp

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Abstract

Testicular necrosis is a rare complication associated with inguinal herniorrhaphy. The main blood supply to the testicles comes from the testicular artery (internal spermatic artery), which branches off from the abdominal aorta; the external spermatic artery, which branches off from the inferior epigastric artery; and the arteries supplying the vas deferens. The testicles are extremely resistant to necrosis. Although early surgical intervention may reverse testicular ischemia, delayed diagnosis can necessitate orchiectomy. We report a case of testicular necrosis shortly after radical inguinal herniorrhaphy requiring orchiectomy following emergency exploration.

A 73-year-old man presented to our hospital 18 months after undergoing robot-assisted radical prostatectomy. He developed a left inguinal hernia following radical prostatectomy and subsequently underwent inguinal herniorrhaphy. On the fifth postoperative day, the patient presented to the hospital complaining of pain and swelling in the left testicle. Physical examination revealed swelling of the left scrotum, and scrotal ultrasonography showed no blood flow in the left testicle. Emergency scrotal exploration for suspected testicular necrosis revealed scrotal skin thickening and black discoloration of the testis and epididymis without evidence of strangulation. Owing to absent testicular blood flow on intraoperative ultrasonography, left orchiectomy was performed. Histopathological examination revealed hemorrhage and ischemic necrosis. When scrotal pain, erythema, or swelling develops after inguinal surgery, prompt evaluation with scrotal ultrasonography is warranted, and the potential need for emergency surgical exploration should be considered.

Categories: Urology
Keywords: doppler ultrasound, emergency exploratory laparotomy, orchiectomy, radical inguinal herniorrhaphy, testicular necrosis

Introduction

It is believed that the most common causes of testicular swelling, orchitis, and ischemic atrophy following inguinal hernia repair are surgical trauma to the testicular veins (i.e., venous stasis and subsequent thrombosis) [1]. The blood supply to the testis consists of the following arteries: (1) the testicular artery (internal spermatic artery), which branches off from the abdominal aorta; (2) the external spermatic artery, which branches off from the inferior epigastric artery; and (3) the arteries supplying the vas deferens. Furthermore, at the upper pole of the testis, there is an extensive collateral circulation between these vessels and those branching from the bladder artery, the prostate artery, and the pudendal artery. Consequently, ischemic changes in the testis are extremely rare [2]. Testicular ischemia may be reversible with prompt surgical intervention. However, after a period of golden time (within six hours), orchiectomy may be unavoidable.

Case Presentation

Case: a 73-year-old man

Present Illness

The patient presented 18 months after robot-assisted radical prostatectomy. During an outpatient visit, he reported a reducible swelling in his left groin and was referred to the Department of Gastrointestinal Surgery on suspicion of a left inguinal hernia. Preoperative computed tomography (CT) demonstrated a left inguinal hernia containing intra-abdominal fat, without definite evidence of incarceration (Figure *1*).

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FIGURE 1: Plain pelvic computed tomography

Left inguinal hernia present; intra-abdominal fat protrusion; about the size of a child's fist; can be reduced while the patient is lying down. No obvious signs of incarceration

Image taken with the patient in the prone position

Based on the new JHS classification, type L3 inguinal hernia was classified as an anterior left inguinal herniorrhaphy using a size L mesh plug.

Intraoperative findings: A 4 cm skin incision was made along the skin midline approximately 0.5 cm caudal to the internal inguinal ring, defined as the midpoint of the line connecting the pubic tubercle and the anterior superior iliac spine. The hernial sac was dissected from the spermatic cord. The hernial sac was dissected up to the level of the preperitoneal fat pad to expose the internal inguinal ring. A plug was inserted into the preperitoneal space through the internal inguinal ring and secured with 3-0 Vicryl from the medial pedicle of the plug to the transversus abdominis fascia. The vas deferens and testicular arteries and veins were passed through the slit in the onlay patch. The patch was secured to the pubic bone with 2-0 nylon and further secured to the transversus abdominis fascia with 2-0 nylon. The procedure was completed as planned without any significant intraoperative complications.

Postoperative course: On postoperative day 5, the patient presented with swelling, spontaneous pain, and erythema in the left scrotum. No fever or urinary symptoms were observed. The results of the blood chemistry tests are shown in Table *1*. No other notable findings were observed.

Blood tests	Value	Range
Hemoglobin	14.6 g/dL	14.0-18.0 g/dL
White blood cell count	10100 /μL	3500-9000 /μL
Platelet count	292000 /μL	150000-400000 /μL
C-reactive protein	3.46 mg/L	□0.30 mg/L
Lactate dehydrogenase	199 U/L	120-245 U/L
Creatine kinase	46 U/L	58-248 U/L

TABLE 1: Blood biochemistry tests

Ultrasonography of the scrotum revealed no blood flow to the left testis (Figure *2*). Emergency exploratory

orchiectomy was performed on the same day via a scrotal skin incision.

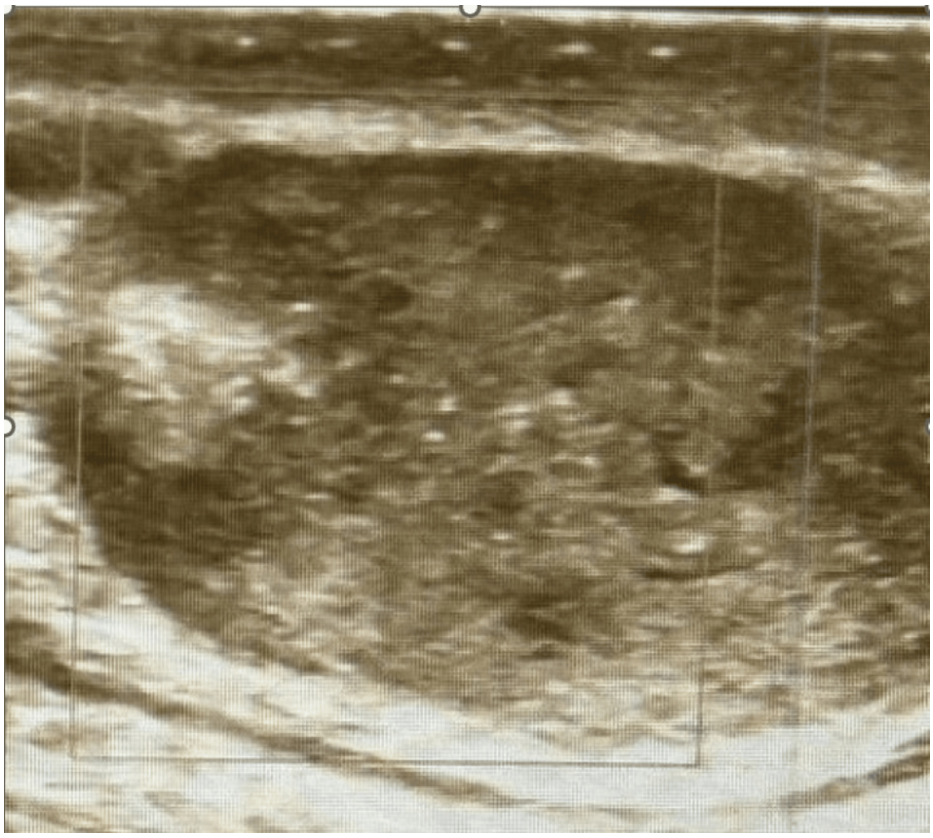


FIGURE 2: Scrotal Doppler ultrasound

A scrotal Doppler ultrasound revealed no blood flow in the left testicle

Intraoperative Findings

Gross intraoperative examination revealed scrotal skin thickening and discoloration of the testis and epididymis. However, the site of strangulation could not be identified through an incision (Figure *3*).

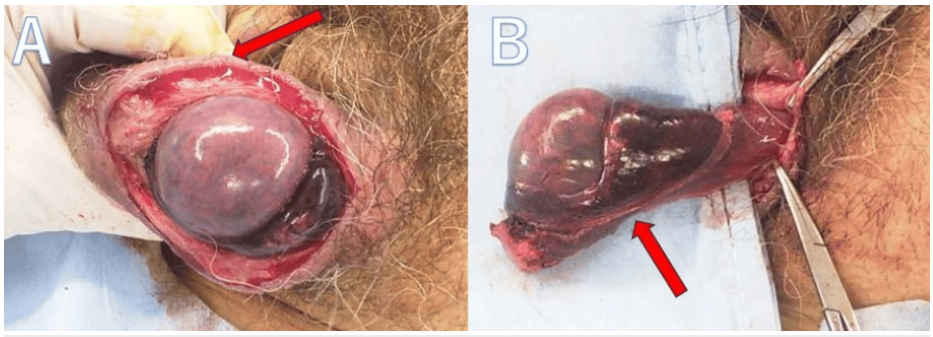


FIGURE 3: Intraoperative findings

Thickening of the scrotal skin (A) and darkening of the testicles and epididymis (B) were observed. No site of strangulation could be identified at the wound site. Intraoperative ultrasound was performed, and an interruption of blood flow was confirmed

Because no blood flow was detected on intraoperative testicular ultrasonography, an orchiectomy was performed.

Postoperative Histopathological Examination

It revealed diffuse congestion and hemorrhage, and extensive necrosis was noted in the testes and epididymis. The pathological diagnosis was hemorrhagic ischemic necrosis caused by testicular strangulation. There were no findings suggestive of malignancy (Figure 4).

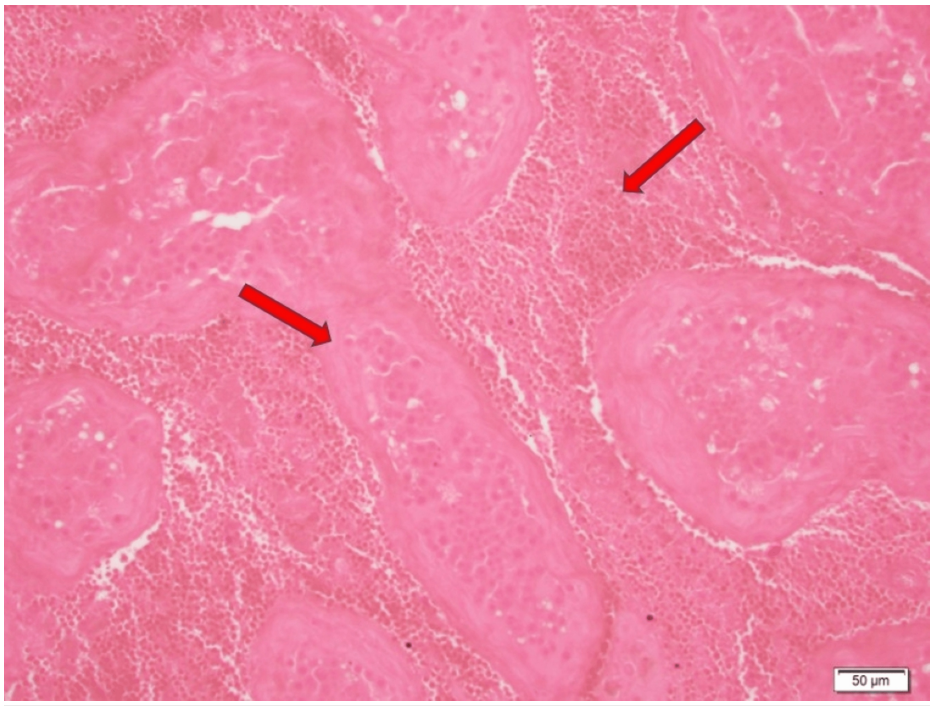


FIGURE 4: Histopathological findings

H&E: hematoxylin and eosin

Histopathological findings based on H&E staining show that the capillaries do not stain purple, suggesting necrosis. The presence of red blood cells between the capillaries suggests hemorrhage. Hemorrhage and ischemic necrosis were observed, but no malignant findings were noted

Postoperative Course

The postoperative course was uneventful with no complications, and the patient was discharged on postoperative day 7.

Discussion

The main causes of acute scrotal pain include testicular torsion, orchiepididymitis, trauma, and hematomas; however, in the differential diagnosis, it is essential to distinguish between testicular torsion and inflammatory conditions [3]. A diagnosis of testicular ischemia is often reached by combining a thorough physical examination and medical history with color Doppler ultrasound findings [3]. Although testicular ischemia is rare, it can sometimes result from severe epididymitis, inguinal hernia repair, spontaneous thrombosis of the spermatic cord vessels, xanthogranulomatous spermatic cord inflammation, or filariasis [3]. Although color Doppler ultrasound has high diagnostic accuracy, surgical exploration is essential when testicular torsion is clinically suspected [3]. In this case, blood tests showed no elevated inflammatory markers, and ultrasound revealed no obvious hematoma. Scrotal ultrasound revealed no blood flow to the left testis; therefore, considering the possibility of testicular torsion or testicular necrosis, an emergency exploratory orchiectomy was performed.

The testis receives its primary arterial supply from the testicular artery, with additional contributions from small branches of the internal and external pudendal arteries to the testicular capsule. Furthermore, because collateral blood flow to the testes occurs via the inferior epigastric, bladder, prostatic, and scrotal arteries, the testes are extremely resistant to necrosis [1,4]. Moreover, some studies have reported that testicular ischemia does not occur in one-third of the cases, even when the spermatic cord is intentionally ligated [4]. Venous stasis following surgical trauma to the spermatic cord during radical inguinal hernia repair leads to thrombosis, which is believed to cause ischemic orchitis and testicular atrophy in 6500 patients [4]. Complications after inguinal herniorrhaphy occur in 1.7% to 8% of all cases. Hernia recurrence is the most common complication, occurring in 0.3%-3.8% of cases [1]. This is followed by vas deferens injury (1.6%) and vascular injury; in particular, injury to the spermatic cord vessels can cause ischemic orchitis, which

may lead to testicular atrophy (0.2%-1.1% of all inguinal herniorrhaphy cases) [1]. Trauma to the delicate veins of the pampiniform plexus is a major cause of testicular atrophy following inguinal herniorrhaphy [5]. Testicular injury ultimately leads to atrophy. While the incidence rate is estimated to be approximately 0.5% for primary hernia repair, it is reported to rise to 5%-10 times that rate for recurrent hernia repair [6].

Modern treatments for inguinal hernias involve repair using synthetic mesh [5]. However, direct contact between the mesh and blood vessels within the inguinal canal, as well as fibrosis around the mesh, may adversely affect testicular blood flow [5]. Although hernia repair surgery is routinely performed, localized testicular ischemia has been reported as a rare complication, likely resulting from postoperative edema and subsequent venous stasis [1]. Even a very small incision in the spermatic cord can damage the spermatic vessels [1].

Even when the risk of complications from radical inguinal hernia repair is low, testicular ischemia and atrophy can cause severe psychological distress and may affect quality of life (QOL), including the risk of infertility. Therefore, it is necessary to provide patients with a thorough explanation and ensure they understand the situation [7]. Other possible causes of focal and total testicular infarction not associated with testicular torsion include hemorrhagic infarction due to tumors or trauma; hematological factors such as sickle cell disease and polycythemia; infectious factors such as epididymitis and epididymo-orchitis; and vasculitis. In many cases, the cause cannot be identified [8].

Our case involved testicular necrosis following the inguinal hernia mesh plug procedure; however, the surgery was completed according to the standard protocol, with no obvious signs of spermatic cord injury, signs of infection, direct contact between the mesh and vessels within the inguinal canal, or fibrosis around the mesh. Histopathological findings revealed no obvious thrombosis, and the site of spermatic cord constriction was unclear. However, although the surgery was completed according to the standard protocol, damage to the spermatic cord vessels cannot be ruled out because of the minimal incision made in the spermatic cord.

A previous case report documented worsening mesh infection occurring two months after mesh repair of an inguinal hernia. Although a mesh removal procedure was performed, ischemic necrosis of the left testis developed on the 10th postoperative day, leading to orchiectomy. This case raised the suspicion of vascular injury to the left spermatic cord following mesh removal [9]. This underscores the importance of preventing mesh infections.

If physical findings such as scrotal pain, redness, or swelling are observed following inguinal herniorrhaphy, it is necessary to carefully evaluate testicular ischemia immediately using methods such as scrotal ultrasound [4], and it is important to decide on an emergency exploratory scrotal incision and treatment plan.

However, while color Doppler ultrasound offers high diagnostic accuracy, it is important to be aware that false-negative results can be caused by incomplete testicular torsion (a condition in which systolic blood flow is recorded but diastolic blood flow is absent or diminished) or reactive congestion of the testicular tunica vaginalis (which may be mistaken for blood flow to the tunical arteries) [3].

The surgical approach varies depending on the condition of the testis during surgery and the duration of ischemia [4]. One intraoperative finding that can distinguish between infarction and reversible ischemia is the failure to achieve hemostasis within 10 min of a deep incision of the testis [10]. If hemostasis is not achieved, the condition is considered infarction rather than reversible ischemia. In cases of testicular infarction, orchiectomy is performed to relieve pain and preserve fertility, as retention of necrotic testicular tissue may provoke an autoimmune response against spermatozoa [10].

However, there are case reports indicating that hernia repair using mesh, while causing transient changes in testicular perfusion (blood flow), did not have any long-term adverse effects on microcirculation, did not affect the blood-testis barrier-which could potentially trigger severe immune reactions-and did not result in a significant increase in antisperm antibody (ASA) levels [6]. Testicular ischemia and necrosis have been reported following both open and laparoscopic inguinal herniorrhaphy, typically occurring two to three days postoperatively [10].

In a case series by Chu et al. [10], the first patient presented with scrotal pain and decreased testicular arterial blood flow eight hours after inguinal herniorrhaphy, and a procedure to loosen the inguinal hernia mesh was performed. The second patient developed ischemic orchitis 20 hours after inguinal herniorrhaphy, requiring mesh removal. In both cases, testicular atrophy was observed at the follow-up. In the third patient, new findings of atrophy and induration of the right testis were observed six months after inguinal hernia repair. A Doppler ultrasound examination of the scrotum revealed that the right testis was significantly smaller than the left, appeared hyperechoic throughout, and showed no apparent blood flow. These findings are consistent with a chronic infarction of the right testis. Since this patient had no history of testicular pain following inguinal hernia repair, he was placed under observation.

The fourth patient presented to the emergency department four days after inguinal hernia repair, complaining primarily of right scrotal pain. A scrotal ultrasound revealed infarction of the right testis and a small, complex hydrocele on the right side. The patient underwent emergency right scrotal exploration. During the procedure, the infarcted right testis was confirmed, and a simple orchiectomy was performed. The pain improved following the orchiectomy [10].

Areas of testicular ischemia and necrosis were excised and repaired during early surgery [10]. However, orchiectomy is considered unavoidable if ischemia is detected at a later stage. Furthermore, in cases of large inguinoscrotal or recurrent hernias, extensive dissection of the spermatic cord and displacement of the testis from the scrotum during surgery may increase the risk of ischemic orchitis and subsequent testicular atrophy. Therefore, some reports have argued that dissections medial and inferior to the pubic tubercle should be avoided [4]. There are also reports suggesting that excessive dissection of the distal hernial sac, displacement of the testis from the scrotum to the surgical site, and the associated scrotal surgery should be avoided [11].

Furthermore, laparoscopic surgery minimizes dissection of the spermatic cord, thereby reducing the risk of inguinal and testicular complications caused by damage to spermatic cord structures or adjacent nerves [5]. Consequently, it is appropriate to actively consider laparoscopic surgery. It is necessary to bear in mind that complications such as testicular necrosis may occur even in surgeries performed near the spermatic cord [4].

Conclusions

We report a case of early-onset testicular necrosis following inguinal herniorrhaphy that ultimately required orchiectomy after emergency surgical exploration. Because the testis is resistant to ischemic changes, testicular necrosis following inguinal herniorrhaphy is an extremely rare complication that is not readily considered in the differential diagnosis of postoperative complications. When symptoms such as scrotal pain, erythema, or swelling appear postoperatively, it is important to immediately consider early emergency scrotal exploration following the evaluation of testicular blood flow, such as Doppler ultrasound. Furthermore, testicular necrosis must always be considered as a potential postoperative complication in other surgeries performed near the spermatic cord.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Takuya Yashima, Yoshihiro Nakagami, Takashi Fukagai

Acquisition, analysis, or interpretation of data: Takuya Yashima, Yoshihiro Nakagami, Kazuhiko Oshinomi, Masakazu Nagata, Takashi Fukagai

Drafting of the manuscript: Takuya Yashima

Critical review of the manuscript for important intellectual content: Takuya Yashima, Yoshihiro Nakagami, Kazuhiko Oshinomi, Masakazu Nagata, Takashi Fukagai

Supervision: Takashi Fukagai

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. The Ethics Committee of Showa Medical University Hospital issued approval CR2026025. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Mincheff T, Bannister B, Zubel P: Focal testicular infarction from laparoscopic inguinal hernia repair . JSLS. 2002, 6:211-3.
2. Gaines RD: Complications of groin hernia repair: their prevention and management . J Natl Med Assoc. 1978, 70:195-8.
3. Pietro P, Francesco A: Testicular ischemia following mesh hernia repair and acute prostatitis . Indian J Urol.

2007, 23:323-5. 10.4103/0970-1591.33736

4. Ates E, Kazici HG, Amasyali AS: A rare complication of inguinal hernia repair: total testicular ischemia and necrosis. Arch Ital Urol Androl. 2019, 91:46-8. 10.4081/aiua.2019.1.46
5. Ramadan SU, Gokharman D, Tuncbilek I, et al.: Does the presence of a mesh have an effect on the testicular blood flow after surgical repair of indirect inguinal hernia?. J Clin Ultrasound. 2009, 37:78-81. 10.1002/jcu.20516
6. Stula I, Družijanić N, Sršen D, et al.: Influence of inguinal hernia mesh repair on testicular flow and sperm autoimmunity. Hernia. 2012, 16:417-24. 10.1007/s10029-012-0918-1
7. Cabral R, Milne DM, Dos Santos K, Lovell R, Sookhai S, Cawich SO: Traumatic testicular dislocation complicated by torsion post hernioplasty: a case report. Cureus. 2025, 17:e86057. 10.7759/cureus.86057
8. Wenn J, Murray BP, Kynast RW, Zhen J: An uncommon cause of testicular infarct: a case report and review of the literature of testicular infarcts from laparoscopic inguinal hernia repair. J Emerg Med. 2024, 67:e375-8. 10.1016/j.jemermed.2024.05.006
9. Tchangai BK, Kpatcha TM, Adabra K, Dosseh DE: Testicular necrosis and subsequent orchiectomy as a complication of inguinal mesh infection surgery: a case report. J Surg Case Rep. 2023, 2023:rjad074. 10.1093/jscr/rjad074
10. Chu L, Timothy DA, Stephen VJ: Testicular infarction as a sequela of inguinal hernia repair . Can J Urol. 2009, 16:4953-4.
11. Calcagno C, Gastaldi F: Segmental testicular infarction following herniorrhaphy and varicocelectomy . Urol Int. 2007, 79:273-5. 10.1159/000107962