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Review

Mechanism, assessment, and incidence of male infertility after inguinal hernia surgery: a review of the preclinical and clinical literature

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Abstract

BACKGROUND: The treatment of inguinal hernia has changed considerably over the past 15 years. We reviewed the preclinical and clinical literature to find out the effect of inguinal hernia surgery on male fertility because it has been suggested that hernia surgery may impair testicular function and male fertility.

DATA SOURCES: A search on Embase, MEDLINE, and the Cochrane Library was performed to find related articles.

CONCLUSIONS: Animal models show substantial effects of hernia repair on the structures in the spermatic cord, which is more pronounced in mesh repairs. Although the number of studies and the included numbers of patients were limited, clinical studies indicate that these potential adverse effects do not seem to have a clinical impact on male fertility in humans with inguinal hernias. Future clinical studies, preferably with bilateral patients, are necessary to investigate the clinical relevance of the effects of inguinal hernia and hernia surgery on male fertility.

Inguinal herniorrhaphy is the most commonly performed general surgical procedure in the Western world. Worldwide, over 20 million inguinal hernia repairs are performed each year.[1] Treatment has changed considerably over the past 15 years. Surgical techniques have evolved from simple tissue-based repairs to the modern "tension-free" mesh repairs that are performed by a conventional open anterior or a laparoscopic/endoscopic approach.

Although recurrence rates have decreased with the introduction of mesh hernia repairs, more interest is now directed to other consequences of hernia surgery, such as persistent postoperative pain and the time needed to resume normal activities. Hernia surgery may impair testicular function too, and, ultimately, it may affect male fertility. Although it is presumed to be rare, in a retrospective study, the incidence of unilateral vas deferens obstruction in subfertile men with a history of pediatric inguinal hernia repair was reported to be as high as 27.8%.[2] This suggests a nonnegligible effect of pediatric hernia surgery on fertility. The objective of this review was to appraise the literature regarding the frequency, mechanisms, and assessment of impaired gonadal function and vas deferens patency after inguinal hernia and hernia surgery.

Methods

A literature search was performed in Embase, MEDLINE, and the Cochrane Library. Search terms were "inguinal

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hernia" OR "herniorrhaphy" OR "hernia surgery" OR "hernia repair" AND "perfusion" OR "volume" OR "infertility" OR "fertility" OR "atrophy" OR "ischemia" OR "ischemia" OR "semen analysis" OR "spermogram" OR "vas obstruction" OR "vas deferens" OR "vasal obstruction" OR "gonadotrophins" OR "gonadotropins" OR "azoospermia" OR "oligospermia" OR "testicular" OR "testis" OR "testes." All titles and abstracts of studies identified by the initial search were screened to select relevant studies in English. All cross-references were screened for potentially relevant studies not identified by the initial literature search.

Mechanisms of Injury to the Testis or Vas Deferens Associated With Hernia Surgery

Within the inguinal canal, the spermatic cord contains the vas deferens, the testicular vessels, the pampiniform plexus, the lymphatic vessels, and 3 nerves (ie, the genital branch of the genitofemoral nerve, the ilioinguinal nerve, and sympathic nerves of the testicular plexus).[3] Although vascular injury during surgery may result in ischemic orchitis, testicular atrophy, and hormonal dysfunction, iatrogenic damage or scarring may obstruct the vas deferens. Both vascular and mechanical injury to the spermatic cord may compromise fertility.

Vascular injury

The testicle has a rich blood supply. Its main arterial supply is the internal spermatic artery (also called the testicular artery) that arises from the aorta.[4] This artery branches at the posterosuperior aspect of the testis where capsular arteries perforate the tunica albuginea continuing as centripetal arteries that enter the testicular parenchyma.[5] A network of collateral arteries within the testes makes it so that disruption of the testicular artery does not always result in testicular ischemia. The veins draining the testicle form the pampiniform plexus. These veins coalesce and form the spermatic vein at the internal inguinal ring. The venous drainage has many collaterals such as the superficial and deep epigastric veins and scrotal veins.[4]

Damage to the vascular structures in the spermatic cord leads to ischemic orchitis. The reported frequency of ischemic orchitis and testicular atrophy is less than 1% after routine inguinal hernia repair, but this is increased after recurrent hernia repair.[6] Most patients with scrotal swelling and testicular pain after hernia repair will have a combination of both arterial and venous occlusion.[4] According to Wantz,[7] the principal cause of ischemic orchitis is venous congestion by thrombosis of the pampiniform plexus caused by surgical trauma.

Apart from the direct surgical trauma, the implantation of mesh causes an inflammatory reaction and the subsequent development of scar tissue surrounding the mesh. This may also result in the compression of the testicular arteries or obstruction of the veins. This is supposed to be another important cause of ischemic orchitis after tension-free mesh repair.[8]

Postoperative orchitis usually subsides completely without permanent damage to the testicle. Only rarely does the testicle become atrophic. The seminiferous tubules, which produce spermatozoa, are usually absent in atrophic testicles.[9] The testosterone-producing Leydig cells and the supporting Sertoli cells usually remain preserved. Atrophic testes measuring less than 2 cm in size may secrete quite normal amounts of testosterone. Only in very severe testicular atrophy is Leydig cell function lost. It has been suggested that unilateral herniorrhaphy could not produce androgen deficiency in cases of unilateral atrophy caused by a normal contralateral testis. Nevertheless, Yavetz et al[10] showed in infertile men with a history of unilateral herniorrhaphy that the highest follicle-stimulating hormone (FSH) levels were found in the group with postoperative atrophy of only 1 testis.

Mechanical injury to the vas deferens

The vas deferens may be damaged by intraoperative iatrogenic transection or compression. Twenty-five percent of iatrogenic vasal injuries are caused by transection-related injuries.[8] The compression-type injury can be caused by intraoperative handling of the vas, by encroachment in the postoperative scar tissue, and by tight placement of the mesh around the cord.[8] Both the transaction- and compression-type injuries can lead to oligo- or azoospermia. The reported incidence of injury to the vas deferens varies between .3% and 7.2%.[11] Furthermore, in subfertile men with a history of pediatric inguinal hernia repair, the incidence of unilateral vas deferens obstruction was as high as 27.8%, implying a substantial effect of hernia surgery on vas deferens patency.[2]

Assessing Testicular Function and Vas Deferens Patency

Testicular perfusion and volume analysis

Testicular volume, morphology, and perfusion can be assessed with ultrasound techniques. The vascular resistance or the resistive index (RI) represents testicular perfusion and can be measured by color Doppler, power Doppler, and spectral Doppler ultrasound. Using the pulsed wave Doppler ultrasound, the peak systolic velocity (PSV) and the end-diastolic velocity (EDV) can be measured, and the RI can be calculated as follows: $RI = PSV - EDV \div PSV$. Inguinal hernia and inguinal hernia repair may cause a mechanical obstruction of the blood vessels in the spermatic cord and may lead to an increased RI in the ipsilateral testicle. Moreover, Pinggera et al[5] found a relationship