Spermatic cord torsion

Torsion of the spermatic cord was first described by Delasauve in 1840,¹ but only became more widely recognised at the beginning of the century after a classical paper by Rigby and Howard.² Since then there have been many reports on this now well described condition, and it is disappointing that so many testes are still lost. Reported rates of testicular salvage after spermatic cord torsion vary from 20 to 60%.³ These figures, however, are from centres with an interest and do not take into consideration those boys who never get admitted to hospital but sit at home taking antibiotics for erroneously diagnosed epididymitis. The true incidence of testicular infarction is probably considerably higher than most reports suggest. Is there any way of improving this rather sorry state of affairs? The testis begins to suffer irreversible damage after four hours of total ischaemia,⁴ and the only hope of improving testicular salvage rates is earlier diagnosis and treatment.

Incidence and age

In a series of cases seen over 15 years in Bristol the diagnostic rate doubled to give an annual incidence of 1 in 4000 males at risk.⁵ Although spermatic cord torsion can occur at almost any age, it is most common around puberty, with a second smaller peak in the first year of life. Spermatic cord torsion is rare over the age of 30.

Neonatal spermatic cord torsion

When the testis and its peritoneal coverings (tunica vaginalis) enter the scrotum in late gestation they are free to rotate within the loose adventitia. In neonatal torsion, epididymis, testis, and tunica all enveloped in the spermatic fascia twist together in a vertical axis to give the typical supravaginal spermatic cord torsion.

This usually occurs in utero or in the first few days of life, especially in the premature baby. The clinical presentation is usually of a swollen hard scrotal mass that is not tender and does not transilluminate. The baby usually seems surprisingly well. If torsion is borne in mind it is usually not difficult to distinguish from hydrocoele, incarcerated hernia, or scrotal haematoma. The presence of a scrotal mass that does not transmit light should be regarded as a torsion until proved otherwise and demands immediate surgical intervention if there is to be any hope of saving the testis. Investigations such as

radionuclide scanning or Doppler ultrasound are difficult to assess in the neonate and should not be used. Neonatal testicular torsion may occur bilaterally,⁶ and early exploration with fixation of the contralateral testis is mandatory to prevent the tragedy of bilateral testicular loss.

Torsion in the adolescent

Torsion of the spermatic cord in older boys almost invariably takes place within the tunica vaginalis, which is often capacious with a high attachment. The peak incidence at puberty probably coincides with loss of cremasteric support for the enlarging testis. Boys with torsion classically present with sudden onset of scrotal and lower abdominal pain often associated with vomiting. The pain sometimes occurs after vigorous exercise, but it is not uncommon to see boys who give a history of waking at night with groin pain and only notice the tender testis later. An appreciable number of patients will have had previous episodes of spontaneously resolving torsion causing intermittent short lived testicular pain.

Examination shows a very tender testis that is usually higher than the opposite testis, which may exhibit the typical horizontal lie. Initially, the affected testis is exquisitely tender, but as the ischaemia progresses the testis becomes slightly less tender and is accompanied by oedema and erythema of the overlying skin.

The commonest misdiagnosis is of acute epididymitis, although this is very rare in adolescent boys without urinary tract infection. Haemorrhage into a testicular tumour may present with a similar clinical picture. Idiopathic scrotal oedema is a rare condition of unknown aetiology affecting young boys who usually present with pink, oedematous scrotal skin and a palpably normal testis and cord.⁷ Torsion of testicular appendages presents with the same clinical features as spermatic cord torsion and is often only diagnosed at exploration. If the diagnosis of torsion is fairly certain there are no specific preoperative investigations that need to be carried out. The presence of pus cells in the urine raises the possibility of epididymitis and in this, and other circumstances where the diagnosis is difficult, it may be helpful to carry out a radionuclide scan, which can distinguish, with reasonable accuracy, the reduced flow associated with torsion from the increased blood flow of epididymo-orchitis.⁸

Although it may be possible to untwist the testis at an early stage by manipulation,⁹ this should not prevent or delay urgent surgical treatment. At exploration, the diagnosis will be apparent and if there is any doubt about viability the testis should be wrapped in a warm swab and reinspected in 10 minutes. After fixation of the affected testis it is essential to explore and fix the contralateral testis during the same operation.

Sequelae to spermatic cord torsion

There has been a surprising lack of long term follow up of boys who have suffered from this condition. Studies that have been carried out suggest that a considerable proportion of boys who have 'successful' explorations for torsion will still suffer a degree of testicular atrophy.^{10 11} Furthermore, analysis of semen from young men who have had torsion showed abnormalities in most cases, and the degree of diminished spermatogenesis could be correlated with the duration of the testicular ischaemia.

Endocrine function, assessed by measuring luteinising hormone, follicle stimulating hormone, prolactin, and testosterone is almost invariably unaffected. These findings suggest that there are abnormalities of the contralateral unaffected testis. Possible explanations for this include recurrent damage from subclinical torsion, congenital abnormalities of both testes associated with the predisposition to torsion, or sympathetic orchidopathy after autoimmunisation.¹²

Conclusion

It can be estimated that 0.5% of the male population

will have suffered spermatic cord torsion by the age of 25. Delayed diagnosis and referral remain the most common cause of testicular loss, and it is still disappointingly common to see late cases being treated for epididymitis. There is now increasing evidence that spermatic cord torsion can lead to later problems with fertility.

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