



TESTICULAR INFARCTION IN A PATIENT WITH SPINAL CORD INJURY WITH EPIDIDYMITIS: A CASE REPORT

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Objective: To describe a case of epididymitis-related testicular infarction in a patient with spinal cord injury.

Case report: A 22-year-old man with a traumatic spinal cord injury (American Spinal Injury Association Impairment Scale A; neurological level, C4) developed epididymitis during hospitalization. He presented with spiking fever, autonomic dysreflexia, and increased spasticity. Physical examination revealed a rapidly progressive, firm swelling of the right hemiscrotum; however, the patient had no subjective complaint of pain owing to a loss of sensation. Ultrasound showed right testicular infarction. The condition was successfully managed with conservative antibiotic treatment. Follow-up ultrasound at 6 months revealed atrophy of the right testis.

Conclusion: Evaluating the progression of epididymitis is difficult in patients with a decreased or absent nociceptive perception, and depends on the patient's clinical course. We recommend close physical examination, including inspection and palpation, as well as greyscale and colour Doppler ultrasound scanning, to exclude the possibility of rare testicular infarction. Conservative treatment may be considered first.

Key words: testicular infarction; epididymitis; spinal cord injury; neurogenic bladder.

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In patients with spinal cord injury (SCI), sensation below the level of the SCI is diminished or lost, thus the evaluation of visceral pain is difficult. Neurogenic bladder dysfunction after SCI is an important issue, and the long-term goals of management of this condition are to adapt the patient to a socially acceptable bladder-emptying method and to avoid the occurrence of complications. Urinary tract infection (UTI) is one of the most common complications after neurogenic bladder dysfunction, and was estimated to have occurred in 50% of the SCI population in a study with a 5-year follow-up (1). UTI can be complicated further by the development of pyelonephritis, epididymitis, orchitis, prostatitis, and urosepsis. Epididymitis is not an uncommon complication after UTI, and may have

more serious consequences. We report here a case of an epididymitis-related testicular infarction in a patient with SCI.

CASE REPORT

A 22-year-old man was admitted to our ward for regular urinary tract surveillance and reconditioning for his physical capacity. He had an SCI due to a motor vehicle accident 2 years previously. Initial imaging showed multiple fractures on his right humeral shaft, tibia, and fibula, and a C5 burst fracture. According to the International Standards for Neurological Classification of Spinal Cord Injury, the patient had a complete lesion (American Spinal Injury Association Impairment Scale A) at C4 level. His upper extremity motor score was 23, and lower extremity motor score 0. His motor level was C4 at the left side and C5 at the right side, and his sensory loss was below T6.

The patient had voiding problems, and his urodynamic evaluation revealed a neurogenic detrusor overactivity problem. He was adapted to a bladder-emptying method involving intermittent catheterizations performed by a caregiver every 3–4 h. In addition, he had been taking anticholinergic medications for an overactive bladder for > 1 year. Throughout that period, he had several episodes of UTI without complications.

On the current admission, the patient was found to have asymptomatic pyuria (white blood cell (WBC) count: 36–50/high-power field (HPF)). His haemogram and vital signs were within the normal limits. However, on the sixth day after admission, he developed a fever (body temperature, 38.8°C) and showed symptoms of autonomic dysreflexia, increased spasticity of both lower limbs, and nausea with vomiting. Urine with heavy odour was noticed during intermittent urethra catheterization. His blood pressure was 158/108 mm Hg, and his heart rate was 117 beats/min. On physical examination, erythematous change, swelling, and bilateral warmth in the hemiscrotum were observed. Moreover, his right hemiscrotum was hard and firm. Laboratory examination revealed leukocytosis (white blood cell count (WBC): 20,400/mm³, segment: 95%) and elevated serum C-reactive protein level (6.82 mg/dl). Urinalysis revealed pyuria (36–50/high-power field (HPF)) with bacteriuria. An empirical antibiotic

(second-generation cephalosporin) was prescribed initially, and was switched to ciprofloxacin on the next day. Urine culture revealed *Escherichia coli*, with no resistance to any of the antibiotics in the sensitivity test. A Foley catheter was indwelled for the drainage of residual urine. The patient continued to have intermittent high fever, with a body temperature up to 39.5°C in the next 2 days.

A scrotal ultrasound was taken because of the progressive swelling of the right hemiscrotum with erythematous change. The greyscale ultrasound examination showed an increased right testicular volume with heterogeneous echogenicity. The colour Doppler ultrasound examination revealed no blood flow in

the right testis, and increased vascularity of the right epididymis (Fig 1A).

A urologist was consulted for the evaluation of the epididymitis-related testicular infarction. We discussed the case with the patient and his family, and agreed on continuing the conservative treatment with the use of intravenous antibiotics. We also informed the patient of a potential infection focus in the infarcted right testis, for which an orchiectomy might be indicated.

After a 2-week treatment with intravenous antibiotics, the laboratory profile suggested that the infection was under control. The patient was discharged and prescribed with oral ciprofloxacin treatment for a total of 4 weeks. Follow-up at the outpatient department was scheduled at 1 and 2 months after the initial hospitalization. Physical examination revealed a firm right testis with a regular size; however, there was no swelling of the right hemiscrotum. Testicular ultrasound at 1-month follow-up showed a marked decrease in volume and a heterogeneous echogenicity in the right testis, with a swollen soft tissue wall (Fig 1B). Increased blood flow in the circumferential region of the right testis was also observed. The 6-month follow-up testicular ultrasound showed atrophy of the right testis (size 26 × 12 × 9 mm) compared with the left testis (size 38 × 30 × 22 mm) (Fig. 1C).

DISCUSSION

Epididymitis-related testicular infarction is uncommon, and it has been mentioned in only a few case reports or case series (2–5). Patients with SCI-related neurogenic bladder dysfunction are at a higher risk of developing UTI, which could be accompanied by complications such as epididymitis. Epididymitis is mainly diagnosed based on the patient's clinical symptoms and the findings of physical examination. Previous reports on epididymitis-related testicular infarction often mentioned the presence of persistent testicular pain with scrotal swelling. However, in patients with a complete SCI, nociceptive visceral pain (below the SCI neurological level) caused by epididymitis is vague or even absent. Therefore, a more advanced epididymitis might be overlooked, and its detection can only rely on constant monitoring through physical examination.

There are 2 reasons that prevented us from performing immediate orchiectomy after the diagnosis of testicular infarction in this patient: (i) his systemic infection was not well controlled and an orchiectomy might induce further morbidities or a delay in wound healing; and (ii) an orchiectomy might cause hypogonadism by reducing the number of Leydig cells, which generally tolerate severe ischaemia (6, 7). The follow-up ultrasound at 1 and 2 months after hospitalization in this patient revealed heterogeneous echogenicity in

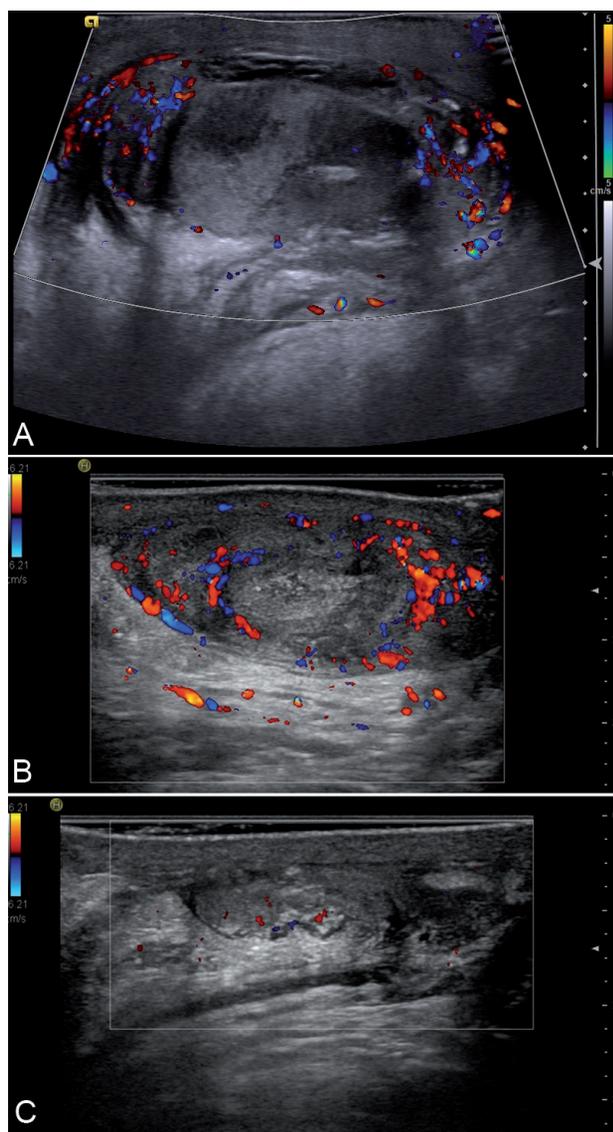


Fig. 1. (A) Colour Doppler image of the sagittal view of right testis showing absence of a colour signal within the entire testis. (B) Colour Doppler image at 1-month follow-up, the image showed decreased testicular volume and heterogeneous echogenicity in right testis, with swelling of soft tissue wall. (C) Six months later the image demonstrated atrophy of the right testis.

his right testis, and there was evidence of the presence of peripheral blood flow. These findings suggest that some testicular tissues may have survived the infarction episode. Thus, the function of some Leydig cells may be preserved (6, 7).

The outcome in the case reported here suggests that conservative medical treatment may be considered in treating an epididymitis complicated with testicular infarction that is detected with a colour Doppler ultrasound. However, the risk of progression to an ischaemic testis should be considered. A history of testicular infarction may cause subfertility or infertility in men (8). Furthermore, the possibility of the production of autoantibodies to the normal testicle tissue after an epididymitis should also be taken into consideration.

The mechanism of epididymitis-induced testicular infarction is not fully understood. The possible mechanisms include: (i) acute onset of oedema of epididymitis, which compromises the venous drainage of the testes and leads to thrombosis formation (1), or (ii) bacterial toxins that cause endothelial damage of vessels (2), which results in thrombus formation. A dysfunction of venous drainage may lead to interruption of arterial flow and subsequently cause testicular infarction.

Since epididymitis is not uncommon in SCI patients, physicians should consider the possibility of an epididymitis-related testicular infarction in patients with an unresolved or rapidly progressive epididymitis, especially in those with persistent febrile episodes after being prescribed with appropriate antibiotics, non-resolving thickening, and/or enlargement of the testicle or spermatic cord (5). We also suggest performing an imaging examination when there is a progression of epididymitis under these circumstances. Greyscale and colour Doppler ultrasound scanning are the most convenient, accessible, and useful tools for evaluating epididymitis and its possible complications, such as testicular infarction. Contrast-enhanced ultrasound may also be another option for evaluating testicular infarction (4).

Since complications of UTI, such as epididymitis or orchitis, could lead to subfertility and infertility if they

progress to testicular infarction, appropriate bladder-emptying strategies and skills should be emphasized in men with SCI, in order to prevent the development of UTI.

In conclusion, although epididymitis-related testicular infarction in patients with complete SCI is rare, it is an important issue because it may affect fertility. Physical examination and imaging studies are important in evaluating the progression of epididymitis in patients with complete SCI, who have no subjective perception of nociceptive visceral pain. Conservative treatment with antibiotics may be a sensible choice of treatment.

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