

## Vascular Abnormality in Hemodialysis-Associated Ischemic Priapism: A Case Report

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## 1. Abstract

**1.1. Background:** Ischemic priapism is a rare condition with fully penile rigidity and severe pain. Blood trapping in the penis is a crucial pathophysiology for ischemic priapism, but the reason causing the blood trapping varies. This report presents a case of a 48-year-old male experiencing a vascular abnormality-relevant ischemic priapism during hemodialysis treated with a caverno-glanular shunt.

**1.2. Case Report:** A 48-year-old male went to Outpatient Department to consult dizziness and hypodynamia. Laboratory examination found an increased blood creatinine (Crea) 695  $\mu\text{mol/L}$  (reference ranges [rr] 40-106  $\mu\text{mol/L}$ ), K 6.5  $\text{mmol/L}$  (rr 3.5-5.5  $\text{mmol/L}$ ), and an emergent hemodialysis was performed. Soon after the onset of hemodialysis, the patient developed an ischemic priapism. About 24 hours later, the patient was transferred to our Emergency Department, a swollen erected painful penis without affecting urination was detected. After examinations, a caverno-glanular shunt was performed, which effectively treated the refractory ischemic priapism. Following examinations revealed a reduced resistance of cavernous arteries and dorsal artery of the penis, as well as a mural thrombus of the right saphenofemoral junction, both of which may serve as a cause for ischemic priapism during hemodialysis in this patient.

**1.3. Conclusion:** Ischemic priapism is a rare complication of hemodialysis, and the cause is not well delineated. This case shows that vasculature abnormality may be involved in the pathogenesis of hemodialysis associated Ischemic priapism, and a regional ultrasound scan along with laboratory investigation before hemodialysis may identify vascular abnormality that pre-dispose to this complication.

## 2. Background

Priapism is a penile erection for more than 4 hours without sexual stimulation, which affects males of any age [1]. Based on the pathophysiology, priapism is divided into three subtypes: stuttering, non-ischemic and ischemic priapism [1, 2]. Stuttering priapism is a recurrent and intermittent painful erection, and is usually caused by sickle cell disease (SCD) [3]. Non-ischemic priapism, also known as high-flow priapism, is typically erected without pain and full rigidity, and is caused by extra blood flow into the corpora cavernosa as a result of penile, perineal or pelvic trauma [4]. Ischemic priapism with trapping blood in the penis is featured by severe pain and full rigidity, and needs an emergent treatment [5]. Although ischemic priapism is the most common type of priapism, its incidence rate is only 1.5- 5.34 per 100 000 person-years among the general population [6]. The reasons that cause ischemic priapism are various, and hemodialysis is a seldom reported reason,

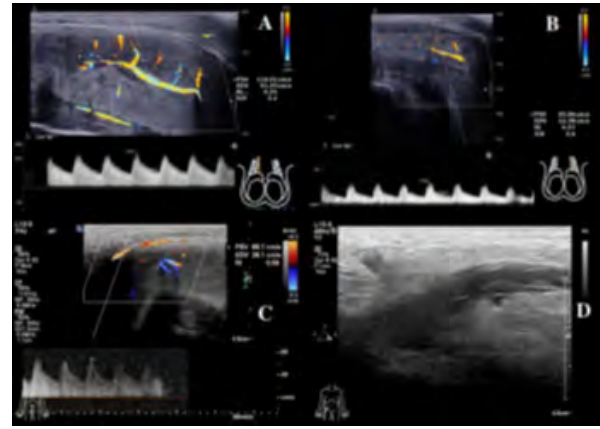
the mechanism of which is unclear [6, 7]. Here, the report presents a case of a 48-year-old hemodialysis male patient experiencing a vascular abnormality-associated ischemic priapism treated with a caverno-glandular shunt.

### 3. Case Report

A 48-year-old male patient went to Outpatient Department to consult dizziness and hypodynamia. Laboratory examination results are shown in table 1 with increased blood creatinine (Crea) 695  $\mu\text{mol/L}$  (rr 40-106  $\mu\text{mol/L}$ ), K 6.5  $\text{mmol/L}$  (rr 3.5-5.5  $\text{mmol/L}$ ), and decreased glomerular filtration rate (eGFR) 39.92  $\text{ml}/\text{mi}$  (rr  $\geq 82$ ). The patient has been diagnosed with chronic kidney disease and renal hypertension for 4 years, and is regularly taking irbesartan and nifedipine to control blood pressure.

An emergent hemodialysis was performed. Soon after the start of hemodialysis, the patient noticed an enlarged penis without any pain, which gradually developed into pain and fully rigidity. Finally, the severe penile pain forced the patient to terminate the hemodialysis in advance, and the termination did not relieve the pain effectively.

About 24 hours later, the patient was transferred to our Emergency Department. In physical examination, a puncture wound at the left femoral vein and a hypertension with 193/129  $\text{mmHg}$  of the patient were found. Other physical examinations were unremarkable. Laboratory tests revealed elevated Crea 695  $\mu\text{mol/L}$  (rr 68-108  $\mu\text{mol/L}$ ), urea 17.77  $\text{mmol/L}$  (rr 3.1-8.0  $\text{mmol/L}$ ), leukocytes  $13.9 \times 10^9/\text{L}$  (rr  $3.5\text{-}9.5 \times 10^9/\text{L}$ ), neutrophils  $11.15 \times 10^9/\text{L}$  (rr  $1.8\text{-}6.3 \times 10^9/\text{L}$ ), fibrinogen 4.63  $\text{g/L}$  (rr 2.0-4.0  $\text{g/L}$ ), decreased eGFR 7.47  $\text{ml}/\text{min}$  (rr 56-122  $\text{ml}/\text{min}$ ) and hemoglobin 99  $\text{g/L}$  (rr 130-175  $\text{g/L}$ ), other results are shown in table 2. Blood tests for liver function, blood cell morphology, cortisol, and adrenocorticotropic hormone did not find obvious abnormality. Sexual stimulation, trauma, SCD, and illegal drug use were all excluded. After blood pressure was controlled by urapidil hydrochloride, the patient underwent an emergency surgery shunt by inserting a 16-gauge needle from the spongy glandular tissue into the corpora cavernosa. Then the penis was manually manipulated (milking maneuver) to drained out the blood of corpus cavernosa. After that, the penis was wrapped with a bandage. The shunt effectively relieved the penis pain and rigidity, and detumescence totally disappeared 48 hours later, and an ultrasound scan of the penis and scrotum was taken out. Reduced resistance of both bilateral cavernous arteries (Figure 1A and B) and dorsal artery of the penis (Figure 1C) was detected, and a mural thrombus at the right saphenofemoral junction (Figure 1D) was revealed. No abnormality in the scrotum was found.



**Figure 1:** Color doppler ultrasound images of the penis. A. Blood flow spectrum of the right penile cavernous artery, resistance index (RI)=0.55. B. Blood flow spectrum of the left penile cavernous artery, RI=0.57. C. Blood flow spectrum of penile dorsal artery, RI=0.59. D. The mural thrombus of the right saphenofemoral junction.

**Table 1:** The results of lab test on Outpatient Department.

Lab test	Result	Reference value
Leukocytes ( $\times 10^9/\text{L}$ )	7.62	3.5-9.5
Neutrophils ( $\times 10^9/\text{L}$ )	4.58	2.0-7.7
Hemoglobin (g/L)	108	110-160
Platelets ( $\times 10^9/\text{L}$ )	210	100.0-300.0
Creatinine ( $\mu\text{mol/L}$ )	695	40-106
Urea (mmol/L)	17.77	1.70-8.30
Cystatin C (mg/L)	3.89	0.00-1.16
Estimated Glomerular Filtration Rate (ml/min)	39.92	$\geq 82$
K (mmol/L)	6.5	3.5-5.5
Na (mmol/L)	140.7	132-146

Source: Bazhong Central Hospital.

**Table 2:** The results of lab test on Emergency Department

Lab test	Result	Reference value
Leukocytes ( $\times 10^9/\text{L}$ )	13.9	3.5-9.5
Neutrophils ( $\times 10^9/\text{L}$ )	11.15	1.8-6.3
Hemoglobin (g/L)	99	130-175
Platelets ( $\times 10^9/\text{L}$ )	205	100-300
Creatinine ( $\mu\text{mol/L}$ )	684	68-108
Urea (mmol/L)	14.6	3.1-8.0
Cystatin C (mg/L)	4.27	0.51-1.09
Estimated Glomerular Filtration Rate (ml/min/1.73m <sup>2</sup> )	7.47	56-122
K (mmol/L)	4.56	3.50-5.30
Na (mmol/L)	141	137.0-147.0
Prothrombin time (sec)	10.6	9.6-12.8
Activated partial thromboplastin time (sec)	27.4	24.8-33.8
Fibrinogen (g/L)	4.63	2.0-4.0

Source: West China Hospital, Sichuan University.

#### 4. Discussion

In this case, vascular abnormality, including reduced resistance of bilateral cavernous arteries and dorsal artery of the penis as well as a mural thrombus of the right saphenofemoral junction, is involved in the pathogenesis of ischemic priapism, which suggests that a regional ultrasound scan before hemodialysis may help avoid the priapism in this patient.

There are limited cases reporting hemodialysis relevant ischemic priapism, and its causes are not clear. Most episodes are idiopathic and several of them may be relevant to the following reasons: (a) hematologic dyscrasia, (b) androgens, (c) anticoagulation, (d) adrenergic receptor blocker drugs [8-12]. Patients with hematologic dyscrasia such as SCD have increased cells in circulation, and this high blood viscosity may block blood flow out of the penis, resulting in deposited hypoxia blood that leads to penis rigidity and pain [12]. Similar to SCD, androgen has been verified to increase erythrocytosis and blood viscosity, and affects the blood flow of the penis [13]. Besides, androgen as replacement therapy for erectile dysfunction is important in maintaining the function of sensory nerve and smooth muscle cell in the penis [14,15]. Thus androgen supplement may increase the risk of ischemic priapism for hemodialysis patients. Heparin as an anticoagulation is widely used in hemodialysis and has been associated with priapism in a limited number of cases [16]. Mechanism of heparin-induced priapism has not been well elucidated. Some researchers declared that heparin-mediated antiplatelet antibodies may cause the aggregation of thrombocytes which decreases penis blood flow and lead to the priapism [17-19], while others suggested that heparin-related priapism is just due to the inadequate dosage of heparin for anticoagulation which leads to priapism by stimulating rebound thrombosis [17-19]. The adrenergic receptor blocker associated priapism is due to the non-selective adrenergic receptor blocking effect on smooth muscle of cavernosal arteries and trabeculae, and this effect causes a consistent relaxation of the smooth muscle which is an important mechanism of priapism [20, 21]. Of the adrenergic receptor blocker induced cases of priapism, most of the blockers are antipsychotics or antihypertensive drugs with a non-selective antagonism of adrenergic receptor [20, 21].

In addition to the reasons mentioned above, abnormal vasculature is another possible reason for ischemic priapism in our patient. Reduced resistance of both bilateral cavernous arteries and dorsal artery of penis was detected in our patient, and this abnormality may lead to a high-irrigated penis similar to nonischemic priapism, which explains an enlarged penis without pain and rigidity at the beginning of hemodialysis for our patient. A mural thrombus at the right saphenofemoral junction was also revealed in our patient, which was accompanied by a placement of venous catheter at the left femoral vein for hemodialysis. Both saphenofemoral junction and femoral vein are important hubs for draining the superficial vein of the penis [22], and their abnormality may impede the blood

outflow of the corpora cavernosa. Static blood of corporal finally turned the nonischemic priapism into a pain and rigid ischemic priapism in our patient. In this condition, a different site for central venous catheter placement may help avoid the ischemic priapism in the patient.

Different from previously reported patients who had undergone hemodialysis for several months before priapism [8,17], our patient developed priapism almost at the begin of his first hemodialysis, which may further verify the involvement of abnormal vasculature in the pathogenesis. Besides, some patients as discussed before have hematologic dyscrasia [9,12], while our patient did not show any clue of hematologic dyscrasia (all the blood test including complete blood cell count did not show any positive result, thus we did not display the associated results in this article). While, Our patient had very typical symptoms like painful erection with remarkable rigidity of the penis, last more than 4 hours, so its easy for us to diagnosis. He did not take any therapy before was transfer to our Department. Thus, an emergent caverno-glandular shunt was taken out, which re-leased the pain and rigidity effectively.

Like other kinds of priapism, treatment for hemodialysis relevant priapism depends on what type of priapism. For stuttering priapism, the goal of treatment is the prevention of episodes, so the etiology treatment is important [3, 5]. As stuttering priapism has a risk of cavernosal fibrosis, and repeated episodes of stuttering priapism may lead to erectile dysfunction in 29% to 48% of patients [23]. A selective  $\alpha 1$ -adrenergic receptor agonist such as phenylephrine injection is recommended after 1 to 2 hours of stuttering priapism [5]. For non-ischemic, priapism often goes away on its own, and immediate treatment may not be necessary [23].

Ischemic priapism as the only type of emergent priapism requires prompt diagnosis and immediate treatment to re-establish the blood flow into the corpora cavernosa [1,2]. A selective  $\alpha 1$ -adrenergic receptor agonist such as phenylephrine is recommended to induce contraction of the cavernous smooth muscle, permitting venous outflow [5]. As first-line therapy, intracavernous phenylephrine injection is suggested at a concentration of 0.1–0.5 mg/ml of sterile saline, and repeated administration can be given at 3–5 min intervals until the penis notably detumescence [24]. With  $\alpha 1$ -adrenergic receptor agonist therapy, 43% to 81% of patients get a detumescence [25]. If the repeated injections failed within 1h of treatment, surgical intervention is needed [26]. Caverno-glandular shunt, as the first choice of surgical treatment for refractory ischemic priapism, created a connection between one or both of the corpora cavernosa and the glans is carried out [26]. In our case, like other reports [27], a shunt created by a large needle passing through the glans into the corpora relieved the pain and rigid penis effectively.

#### 5. Conclusion

Hemodialysis associated ischemic priapism is a rare case, and its cause is not well understood. In our case, abnormal vasculature

seems to be an important reasons, and a regional ultrasound scan before hemodialysis may help avoid the priapism. However, further studies are still needed to better prevent this urological emergency.

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