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# VascularAbnormalityinHemodialysis-AssociatedIschemicPriapism:ACaseReport

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## 1. Abstract

**Background:** Ischemic priapism is a rare condition with full-lypenilerigidityandseverepain.Bloodtrappinginthepenisis a crucial pathophysiology for ischemic priapism, but the reason causing the blood trapping varies.This report presents a case of a 48-year-oldmaleexperiencingavascularabnormality-relevantis-chemicpriapismduringhemodialysistreatedwithacanerno-glan- dular shunt.

**Case Report:** A 48-year-old male went to Outpatient Department to consult dizziness and hypodynamia. Laboratory ex-aminationfoundanincreasedbloodcreatinine(Crea)695umol/L (reference ranges [rr] 40-106 umol/L), K 6.5 mmol/L (rr 3.5-5.5 mmol/L), and an emergent hemodialysis was performed. Soon after the onset of hemodialysis, the patient developed an ischem- ic priapism. About 24 hours later, the patient was transferred to ourEmergencyDepartment,aswollenerectedpainfulpeniswithout affecting urination was detected. After examinations, a cav- erno-glanulars huntwasperformed,whicheffectivelytotreatedthe 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,bothofwhichmayserveasacauseforischemicpriapism during hemodialysis in this patient.

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**Conclusion:** Ischemic priapism is a rare complication of hemodialysis,andthecauseisnotwellde-lineated.Thiscaseshows that vasculature abnormality may 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 sexu- al stimulation, which af-fects males of any age [1]. Based on the pathophysiology, priapism is divided into three subtypes: stutter- ing, non-ischemic and ischemic priapism [1, 2]. Stuttering priapismisarecurrentandintermittentpainfulerection,andisusually caused by sickle cell disease (SCD) [3]. Non-ischemic priapism, alsoknownashigh-flowpriapism,istypicallyerectedwithoutpain andfullrigidity,andiscausedbyextrabloodflowintothecorpo- ra cavernosa as a result of penile, perineal or pelvic trauma [4]. Ischemic priapism with trapping blood in the penis is featured by severepainandfullrigidity,andneedsanemergenttreatment[5]. Althoughischemicpriapismisthemostcommontypeofpriapism, itsincidencerateisonly1.5-5.34per100000person-yearsamong the general population [6]. The reason that cause ischemic priapismarevarious,andhemodialysisisaseldomreportedreason,

themechanismofwhichisunclear[6,7].Here,thereportpresents 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

A48-year-oldmalepatientwenttoOutpatientDepartmenttocon- sult dizziness and hypodynamia. Laboratory examination results are shown in table 1 with increased blood creatinine (Crea) 695 umol/L (rr 40-106 umol/L), K 6.5 mmol/L (rr 3.5-5.5 mmol/L), anddecreasedglomerularfiltrationrate(eGFR)39.92ml/mi(rr≥ 82 ). The patient has been diagnosed with chronic kidney disease andrenalhypertensionfor4years,andisregu-larlytakingirbesar- tan and nifedipine to control blood pressure.

An emergent hemodialysis was performed. Soon after the start of hemodialysis, the pa-tient noticed an enlarged penis without any pain,whichgraduallydevelopedintopainandfullyrigidity.Final-ly,theseverepenilepainforcedthepatienttoterminatethehemo- dialysis in advance, and the termination did not relieve the pain effectively.

About24hourslater,thepatientwastransferredtoourEmergency Department.Inphysicalexamination,apuncturewoodsattheleft femoralveinandahypertensionwith193/129mmHgofthepatient were found. Other physical examinations were unremark-able. Laboratory tests revealed elevated Crea 695 umol/L (rr 68-108 umol/L),urea 17.77 mmol/L(rr 3.1-8.0 mmol/L), leukocytes 13.9 ×109/L(rr3.5-9.5×109/L),neutrophils11.15×109/L(rr1.8-6.3×109/L),fibrinogen4.63g/L(rr2.0-4.0g/L),decreasedeGFR 7.47 ml/min (rr 56-122 ml/min) and hemoglobin 99 g/L (rr 130-175 g/L), other results are shown in table 2. Blood tests for liver function, blood cell morphology, cortisol, and adrenocorticotrop-ic 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 un-derwentanemergencysurgeryshuntbyinsertinga16-gaugenee- dle 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 re-lieved the penis pain and rigidity, and detumescence totally disappeared 48 hours latter, and an ultrasound scan of the penis and scrotum was taken out. Reduced resistance of both bilateral cavernous arteries (Figure 1Aand B) and dorsal artery of the penis (Figure 1C) was detected, and a mural thrombus at the right saphenofemoral junc-tion(Figure1D)wasrevealed.Noabnormalityinthescrotumwas found.

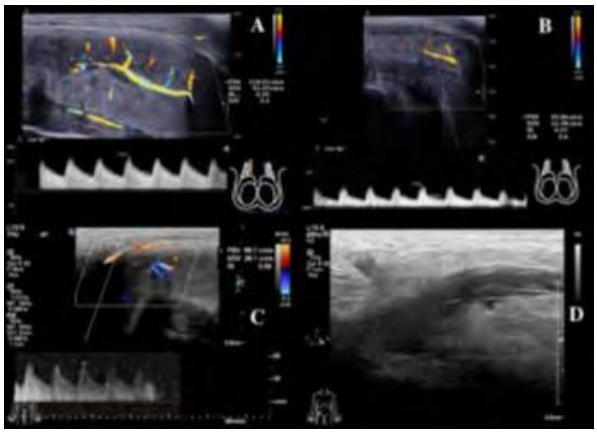


**Figure 1:** Color doppler ultrasound images of the penis. A.Blood flow spectrumoftherightpenilecavernousartery,resistanceindex(RI)=0.55. B.Blood flow spectrum of the left penile cavernous artery, RI=0.57. C. Bloodflowspectrumofpeniledorsalartery,RI=0.59.D.Themuralthrom- bus of the right saphenofemoral junction.

**Table1:**TheresultsoflabtestonOutpatient Department.

| Labtest | Result | Referencevalue |
|---|---|---|
| Leukocytes($\times10^9$/L) | 7.62 | 3.5-9.5 |
| Neutrophils($\times10^9$/L) | 4.58 | 2.0-7.7 |
| Hemoglobin (g/L) | 108 | 110-160 |
| Platelets($\times10^9$/L) | 210 | 100.0-300.0 |
| Creatinine (umol/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 | ≥ 82 |
| K(mmol/L) | 6.5 | 3.5-5.5 |
| Na (mmol/L) | 140.7 | 132-146 |

Source: Bazhong Central Hospital.

**Table2:**TheresultsoflabtestonEmergencyDepartment

| Labtest | Result | Referencevalue |
|---|---|---|
| Leukocytes($\times10^9$/L) | 13.9 | 3.5-9.5 |
| Neutrophils($\times10^9$/L) | 11.15 | 1.8-6.3 |
| Hemoglobin (g/L) | 99 | 130-175 |
| Platelets($\times10^9$/L) | 205 | 100-300 |
| Creatinine (umol/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$^2$) | 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:WestChinaHospital,SichuanUniversity.

## 4. Discussion

Inthiscase,vascularabnormality,includingreducedresistanceof bilateralcavernousarteriesanddorsalarteryofthepenisaswellas amuralthrombusoftherightsaphenofemoraljunction,isinvolved 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,anditscausesarenotclear.Mostepisodesareidiopathicandseveralofthemmayberelevanttothefollowingreasons: (a) hematologic dyscrasia, (b) androgens, (c) anticoagulation, (d) adrenergicreceptorblockerdrugs[8-12].Patientswithhematologic 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 forerectiledysfunctionisimportantinmaintainingthefunctionof sensory nerve and smooth muscle cell in the penis [14,15]. Thus androgen supplement may increase the risk of ischemic priapism forhemodialysispatients.Heparinasananticoagulationiswidely used in hemodialysis and has been associated with priapism in a limitednumberofcases[16].Mechanismofheparin-inducedpriapismhasnotbeenwellelucidated.Someresearchersdeclaredthat heparin-mediated antiplatelet antibodies may cause the aggregationofthrombocyteswhichdecreasespenisbloodflowandleadto the priapism [17-19], while others suggested that heparin-related priapismisjustduetotheinadequatedosageofheparinforanticoagulation which leads to priapism by stimulating rebound thrombosis[17-19].Theadrenergicreceptorblockerassociatedpriapism 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].

Inadditiontothereasonsmentionedabove,abnormalvasculature is another possible reason for ischemic priapism in our patient. Reducedresistanceofbothbilateralcav-ernousarteriesanddorsal artery of penis was detected in our patient, and this abnor-mality mayleadtoahigh-irrigatedpenissimilartononischemicpriapism, which explains an enlarged penis without pain and rigidity at the beginning of hemodialysis for our patient. A mural thrombus at therightsaphenofemoraljunctionwasalsorevealedinourpatient, which was accompanied by a placement of venous catheter at the leftfemoralveinforhemodialysis.Bothsaphenofemoraljunction and femoral vein are important hubs for draining the superficial veinofthepenis[22],andtheirabnormalitymayimpedetheblood outflow of the corpora cavernosa. Static blood of corporal finally turned the nonischemic priapism into a pain and rigid ischemic priapisminourpatient.Inthiscondition,adifferentsiteforcentral venous catheter placement may help avoid the ischemic priapism in the patient.

Differentfrompreviouslyreportedpatientswhohadundergonehemodialysis for several months before priapism [8,17], our patient developed priapism almost at the begin of his first hemodialysis, whichmayfurtherverifytheinvolvementofabnormalvasculature in the pathogenesis. Besides, some patients as discussed before havehematologicdyscrasia[9,12],whileourpatientdidnotshow 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 remarkablerigidityofthepenis,lastmorethan4hours,soitseasy for us to diagnosis. He did not take any therapy before was transfertoourDepartment.Thus,anemergentcaverno-glandularshunt 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 etiologytreatmentisimportant[3,5].Asstutteringpriapismhasa riskofcavernosalfibrosis,andrepeatedepisodesofstutteringpriapism may lead to erectile dysfunction in 29% to 48% of patients [23].Aselective α1-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 bloodflowintothecorporacavernosa[1,2].Aselectiveα1-adrenergic receptor agonist such as phenylephrine is recommended to in-duce contraction of the cavernous smooth muscle, permitting venousoutflow[5].Asfirst-linetherapy,intracavernousphenylephrine injection is suggested at a concentration of 0.1–0.5 mg/ml of sterile saline, and repeated administration can be given at 3–5minintervalsuntilthepenisnotablydetumescence[24].With α1-adrenergicreceptoragonisttherapy,43%to81%ofpatientsget adetumescence[25].Iftherepeatedinjectionsfailedwithin1hof treatment, surgical intervention is needed [26]. Caverno-glanular shunt, as the first choice of surgical treatment for refractory ischemicpriapism,createdaconnectionbetweenoneorbothofthe corpora cavernosa and the glans is carried out[26]. In our case, like other reports [27], a shunt created by a large needle passing throughtheglansintothecorporarelievedthepainandrigidpenis effectively.

## 5. Conclusion

Hemodialysis associated ischemic priapism is a rare case, and its causeisnotwellun-derstood.Inourcase,abnormalvasculature

seems to be an important reasons, and a regional ultrasound scan before hemodialysis may help avoid the priapism. However, furtherstudiesarestillneededtobetterpreventthisurologicalemergency.

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