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# A Case Report of Aortic Dissection in A Uremic Hemodialysis Patient

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#### Abstract

Aortic dissection is a critical cardiovascular disease with extremely high mortality. It has an acute onset and poor prognosis. Patients with uremic hemodialysis have more risk factors for aortic dissection than ordinary patients. Early diagnosis and treatment have a positive impact on the prognosis of aortic dissection. This article reports a case of uremic hemodialysis complicated with aortic dissection, aiming to further explore the clinical diagnosis, treatment and distinguishing characteristics of uremic complicated with aortic dissection, and improve the early diagnosis rate of the disease.

Keywords: Aortic dissection • Uremic hemodialysis

# Introduction

A 46-year-old male patient. Two years before admission, there was no obvious cause for increased foam in the urine. One year ago, the patient began to experience decreased urine output, fatigue, loss of appetite, bleeding gums, tremor of hands and numbness of limbs, intermittent nausea, vomiting. The contents of the stomach were the contents of the stomach, and he went to a local hospital. A complete examination showed abnormal renal function.

## **Case Report**

The patient was hospitalized in our hospital for further diagnosis and treatment. After the complete examination, the patient was diagnosed as uremia.11 months ago, he underwent peritoneal dialysis catheterization and started peritoneal dialysis. However, the patient had a lot of fluid intake and poor self-management ability of peritoneal dialysis. The patient had repeated heart failure and severe scrotal edema and stopped peritoneal dialysis. Seven months ago, he underwent semi-permanent hemodialysis catheter placement in the internal jugular vein in our hospital and regular hemodialysis treatment in the dialysis center of our hospital. The condition is still stable, but the patient still has a lot of fluid intake during the dialysis interval, and he has selfmanagement ability. Poor, the average weight gain between each hemodialysis interval is about 5 Kg. The patient had a sudden chest pain at home 4 months ago, accompanied by chest tightness and pressure in the precordial area, accompanied by back pain, and the pain was tear-like. The pain was relieved after about 10 minutes, and there was still persistent pain, but the patient Didn't care. Two hours after the pain occurred, the patient came to our hospital's hemodialysis center for hemodialysis treatment. Monitoring of blood pressure during dialysis showed that the patient's blood pressure was significantly lower than usual. At the same time, the patient still had chest pain and chest tightness symptoms, so he was hospitalized. Improve auxiliary examination results: blood routine: red blood cells  $3.20 \times 10^{12}$ /L, hemoglobin concentration 92 g/L, hematocrit 28.2%, platelets 236 × 10<sup>9</sup>/L; blood biochemistry: potassium 4.81 mmol/L, sodium 143.5 mmol/L, chlorine 102 mmol/L, bicarbonate 21

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mmol/L, calcium 1.88 mmol/L, phosphorus 2.22 mmol/L; coagulation function: prothrombin time 15.0 s, prothrombin activity 65.5%, prothrombin ratio 1.29, international standardized ratio 1.32, partial thromboplastin time >170 s, thrombin time >160 s, D-dimer 8.61 mg/L, fibrinogen degradation product 19.80 ug/mL; ECG examination: sinus bradycardia, left ventricular high voltage, ST-T is abnormal and the QTc interval is prolonged.

The possibility of acute myocardial infarction cannot be ruled out due to the patient's chest pain. After a cardiology consultation, he was transferred to the cardiology department for continued diagnosis and treatment. The bedside cardiac color Doppler ultrasound and abdominal aorta color Doppler ultrasound examination were completed in the cardiology department. The results suggested: aortic sinus and ascending aorta Exfoliated intima-like echoes were seen in the cavities, showing ribbon-like movement, massive aortic valve regurgitation, massive regurgitation in the mitral valve, left ventricular enddiastolic diameter 71 mm, right atrium transverse diameter 51 mm, pulmonary artery pressure 60mmHg, LVEF 60%; abdominal aortic dissection. Due to the severe condition of the patient and the limited technical conditions in our hospital, the patient's aortic dissection is at risk of rupture at any time. It is recommended that the patient go to a higher-level hospital for further treatment. The patient completed the color Doppler ultrasound examination in the higherlevel hospital and clearly diagnosed the aortic dissection (DeBake type I). Due to the huge risks of the operation and the high cost of the operation, the patient finally refused the operation and was transferred to our hospital again. After the patient was transferred back to our hospital, he was given continuous bedside renal replacement therapy in the intensive care unit of our hospital. At the same time, he was given treatments such as blood pressure control, heart rhythm control, sedation, and pain relief. The condition gradually stabilized and was transferred to the nephrology department for continued treatment. Continue regular hemodialysis treatment. Two months ago, the patient started to have pain in both lower limbs, accompanied by difficulty in walking and standing, and underwent CTA examination of the aorta. The results showed that the aorta originated from the left ventricle and was able to walk. The aorta originated from the left common iliac artery and left kidney. The artery can be seen with a linear low-density internally shifted internal membrane.

The lumen is divided into two cavities, the real cavity is small, and the density is high, and the false cavity is large, and the density is slightly lower. In the ascending aorta root, the internal membrane seems to be broken. The brachiocephalic trunk left common carotid artery, subclavian artery, and superior mesenteric artery originated from the true cavity, and the celiac trunk, inferior mesenteric artery, and bilateral common iliac artery originated from the false cavity. Diagnosis of aortic dissection (Stanford type A). As the disease progressed, the patient's lower limb pain gradually increased, accompanied by obvious bilateral hip and buttocks pain, and limited movement of the lower

limbs, so he was hospitalized again. Re-examination results after admission: blood routine: white blood cells  $12.58 \times 10^{9}$ /L, red blood cells  $3.55 \times 10^{12}$ /L, hemoglobin concentration 96 g/L, hematocrit 29.70%, platelets  $31 \times 10^{9}$ /L; coagulation function: prothrombin time 14.2 s, Prothrombin ratio 1.30, international standardization ratio 1.30, partial thromboplastin time 39.80 s, D-dimer 26.51 mg/L, fibrinogen degradation product 102.97 ug/mL. After admission, the patient gradually developed red ecchymosis and ecchymosis on both feet and lower extremities, and the skin of both feet and lower extremities was dark purple. Diffuse intravascular coagulation was considered. The patient's family refused further treatment. Three days after admission, the patient's heart rate and blood pressure dropped suddenly and eventually died.

## Discussion

Aortic dissection refers to a severe aorta in which the internal rupture and rupture of the aorta, the blood enters the aortic wall from the rupture of the intima, separates the arterial intima and media, and expands along the long axis of the aorta to form two channels of true lumen and false lumen. Disease, also known as aortic dissecting aneurysm. Its clinical features are rapid onset, sudden severe pain, high blood pressure, cardiovascular symptoms, and other vital organs or limb ischemia symptoms. The high incidence of aortic dissection is the middle-aged and elderly people aged 50-70. The annual incidence rate is 2.9 per 100,000. Men are more common than women. The disease is more dangerous. If not diagnosed and treated in time, the mortality rate will exceed 50% within 3 days of onset [1], the mortality rate can reach 60%-70% within 1 week. At present, there are few cases of hemodialysis patients with aortic dissection. Takeda retrospectively analyzed 896 hemodialysis patients in the past 10 years in 1997. Among the 113 deaths, 5 patients died of aortic dissection. The prevalence of hemodialysis patients with aortic dissection is much higher than that of the general population [2]. Compared with the general population, the mortality of blood patients caused by aortic dissection is significantly higher.

According to the location of the breach and the extent of the dissection, aortic dissection is divided into three types, namely Debakey classification. Debakey type I: The breach is located within 5 cm of the aortic valve, the proximal end involves the aortic valve, the distal end involves the aortic arch, descending aorta, abdominal aorta, and even the iliac artery. Debakey Type II: The location of the breach is the same as Type I, and the dissection is limited to the ascending aorta. Debakey type III: The breach is located 2 to 5 cm far from the opening of the left subclavian artery and involves the iliac artery distally. In clinical diagnosis and treatment, it is divided into two types: A and B (Stanford classification) according to the need for surgery: Stanford Type A: The breach is in the ascending aorta, suitable for emergency surgery. Stanford Type B: Dissection lesions are limited to the abdominal aorta or iliac artery, which can be treated with internal medicine first, followed by surgery or endovascular treatment. The traditional time classification defines the acute phase within 2 weeks of onset, the subacute phase from 2 weeks to 2 months, and the chronic phase more than 2 months. However, many current studies have shown that some patients with aortic dissection still have complications of various systems, such as rapid increase in the diameter of the aorta, after 2 weeks of onset [3]. This was consistent with the condition of this patient reported by us, and his condition worsened again several months after the onset of illness, and eventually died.

The specific pathogenesis of aortic dissection is still unclear. At present, many scholars believe that the related risk factors for aortic dissection include hypertension, atherosclerosis, infection, smoking, trauma, hyper free fatty acid, hyperlipidemia, pregnancy, latrogenic injury, autoimmune inflammatory disease, congenital heart disease and hereditary connective tissue disease (such as Marfan syndrome, etc.). Hypertension is the most important risk factor. About 70%-85% of patients with aortic dissection also suffer from hypertension [4]. The impact pressure on the blood flow of the arterial wall during hypertension, the morphology and proportion of elastic fibers and collagen fibers of the aortic wall are imbalanced, the stiffness of the aortic wall increases, and the intima of the blood vessel is easily torn and dissection occurs [5]. In recent years, studies have shown that immune-inflammatory

factors are involved in the occurrence and development of aortic dissection. Anzai found in animal experiments [6] that damage to the aortic wall caused by aortic dissection can increase the expression of neutrophil chemokines and granulocyte knockdown stimulating factors and promote the proliferation of neutrophils and accumulate in the blood vessels of the lesion. It releases matrix metalloproteinases and leukocyte mediator-6 to promote the inflammatory reaction process of the adventitia of the diseased aortic wall, leading to the progression and aggravation of aortic dissection. In uremic maintenance hemodialysis patients, the risk factors for aortic dissection are more diverse, mainly including renal hypertension, calcium and phosphorus metabolism disorders, lipid metabolism disorders, chronic microinflammatory conditions, and hemodynamics during intermittent hemodialysis treatment Unstable etc. The most important risk factors are hypertension and atherosclerosis. Studies have shown that atherosclerosis in uremic patients with long-term hemodialysis is different from atherosclerosis in general patients. Its outstanding feature is a significant increase in calcium deposits, accompanied by an increase in the thickness of the arterial media [7].

In the patient we reported, the initial manifestations of the patient were chest pain and hypotension, blood pressure returned to normal after the condition was stabilized, and cyanosis of the lower limbs appeared when the condition got worse again. The clinical manifestations of aortic dissection are diverse, of which pain is the most important and common clinical manifestation of the disease. About 96% of patients with aortic dissection have sudden and severe pain, which is tear-like or knife-cut [8]. The painful part is related to the location of the lesion, and it can radiate to the neck, shoulders, chest, abdomen and lower limbs. Sudden and severe pain is the clinical distinguishing point for early identification of aortic dissection. Most patients will also have shock manifestations such as anxiety, restlessness, clammy limbs, etc., but usually blood pressure is normal or elevated. This symptom affects aortic dissection. The diagnosis has a prompting effect [9]. In addition, most patients have a large difference in blood pressure between the upper and lower limbs at the onset of the disease. When the aortic dissection hematoma involves the opening of the coronary artery, myocardial ischemia or myocardial infarction will occur. When the hematoma involves the root of the aortic valve, heart failure or aortic regurgitation will occur. When the hematoma involves the brachial trunk, nerves will appear. Symptoms of systemic ischemia, such as syncope, ischemic stroke, paraplegia, etc., when bronchial artery, renal artery, and celiac artery are involved, pulmonary infarction, acute kidney injury, extensive gastrointestinal ischemia or necrosis may occur. Arterial dissection hematoma involving the abdominal aorta or iliac artery can cause acute lower limb ischemia, which can be clinically manifested as cold or cyanosis of the limbs and weakened or disappeared pulse or foot artery pulse [10-12].

In addition to the above-mentioned pain and other manifestations, the patient we reported also had diffuse intravascular coagulation. At present, the situation of aortic dissection with diffuse intravascular coagulation is relatively rare. Some studies believe that after the onset of aortic dissection, mechanical damage to the aortic endothelium causes the release of tissue factors, which leads to activation of coagulation factors in the blood, and then coagulation factors and platelets A large amount of consumption leads to the occurrence of diffuse intravascular coagulation [13]. Once diffuse intravascular coagulation occurs, the treatment of such patients is more difficult, and the mortality rate is higher. Many foreign studies have found that although surgical treatment of aortic dissection under cardiopulmonary bypass can effectively prevent the progression of the disease and reduce the mortality rate, it cannot reduce the occurrence of diffuse intravascular coagulation complications during the perioperative period. The treatment of aortic dissection mainly includes medical treatment and surgical treatment. Medical drug treatment mainly includes analgesia, sedative treatment, and treatment to control blood pressure and heart rate. It should be noted that while reducing blood pressure, it is necessary to maintain blood perfusion of the organs. Surgical treatment includes the development of surgical treatment and intravascular interventional treatment. Studies have shown that for patients with Stanford type A aortic dissection, it is still advocated to open surgical treatment as soon as possible, to perform aortic replacement during the operation, and to repair the aortic valve function at the same time [14]. However, due to the high application requirements, endovascular intervention Treatment has not yet become a routine way for Stanford an aortic dissection. For patients with complex Stanford B aortic dissection, early surgical intervention based on drug therapy is advocated. At present, endovascular interventional surgery has become the first choice for Stanford B aortic dissection. For patients with uncomplicated Stanford B aortic dissection, drug treatment can improve their prognosis, but there is still a risk of tumor-like expansion or even rupture of long-term diseased angiogenesis.

# Conclusion

Aortic dissection is a critical cardiovascular disease with extremely high mortality. Early diagnosis, early drug intervention, and surgical treatment can significantly improve the prognosis of the disease. Uremia patients have more risk factors than ordinary patients on hemodialysis. Therefore, when patients with uremia have sudden chest and abdomen pain, they should be alert to the possibility of aortic dissection. It needs to be related to acute myocardial infarction, pulmonary embolism and acute abdomen. Identify, further reduce the misdiagnosis rate and improve the prognosis of patients.

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