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Aortic Dissection as a Complication in Patients on Maintenance Haemodialysis: Two Cases Report

Zhuo-Tao Xiao¹, Xin-Zhong Huang¹, Jian-Hua Wu¹, Xia Jiang^{2*}, Xi Huang^{3*} and Li Yuan^{1*}

¹Division of Nephrology, Affiliated Hospital of Nantong University, Nantong, Jiangsu, 226001, China

²Division of Nephrology, The Second Peoples Hospital of NanTong, Nantong, Jiangsu, 226001, China

³School of Mechanical Engineering, Nantong University, Nantong, Jiangsu, 226019, China

*Corresponding author: Xia Jiang, Division of Nephrology, The Second Peoples Hospital of NanTong, Nantong, Jiangsu Province, 226001, China, Tel: 86513- 85169100; Fax: 86513- 85169100; E-mail: 846991044@qq.com

Xi Huang, School of Mechanical Engineering, Nantong University, Nantong, Jiangsu, 226019, China, Tel: 86513- 85012673; Fax: 86513- 85012671; E-mail: 47293245@qq.com

Li Yuan, Division of Nephrology, Affiliated Hospital of Nantong University, 20 Xisi Road, Nantong, Jiangsu Province, 226001, China, Tel: 86513- 85050212; Fax: 86513- 85106566; E-mail: yuanlint@163.com

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Abstract

Aortic dissection (AD) is a rare disease that may result in a fatal outcome. Uremic patients are often associated with factors susceptible to AD. We herein report two cases of AD occurring in maintenance haemodialysis patients. The two male Chinese had history of smoking and poor uncontrolled hypertension for many years. One patient died of the aortic wall rupture with conservative treatment. The other patient had a history of myocardial infarction and long-term administration of anti-platelet drugs. The patient survives after prompt stenting and continuous maintenance haemodialysis. It is important to improve our understanding about aortic dissection (AD) in haemodialysis patients because of the high mortality and morbidity. Once a patient on maintenance haemodialysis is diagnosed with AD, surgery or endovascular graft exclusion should be considered according to the actual situation of individual patients, and the mode of dialysis should be converted in conservation treatment patients.

Keywords: Aortic dissection; Haemodialysis; Endovascular graft exclusion

Introduction

Aortic dissection (AD) refers to the intimal tear due to damage of the normal structure of the aortic wall from various reasons. Mechanically, it has been considered that circulating blood flows into the media of the aorta through the rupture of the intima forming true and false lumens. It can extend and dissect, causing a high mortality rate of 60-70% within a week, and 90% within three months [1]. Uremic patients with chronic renal dysfunction are often associated with refractory hypertension,

atherosclerosis and lipid metabolism disorders. These complications together with great fluctuations of blood volume before and after haemodialysis constitute risk factors of AD in patients on maintenance haemodialysis. This article reports two cases of AD occurring in patients on maintenance haemodialysis between 2000 and 2013 in the department of haemodialysis of the hospital.

Case 1

A 50-year-old Chinese male patient who had been on maintenance haemodialysis for a year at twice-a-week regular intervals came to the hospital because of sudden onset of persistent severe chest pain, when BP was 176/90 mmHg and HR was 88 bpm. He had a 20-year history of smoking and a 5-year history of hypertension without satisfied control. His usual BP was higher than 160/100 mmHg. Admission CT scan showed dilation of the aortic arch and thoracic aorta. Contrast-enhanced CT scan demonstrated the existence of a linear high-intensity shadow in the aortic arch, descending aorta, abdominal aorta, bilateral common iliac arteries and branches. A diagnosis of Debakey III AD was made. The break was located in the aortic arch (**Figures 1 and 2**). Electrocardiography (ECG) showed LV high voltage and ST-T change. Chest X-ray showed aortic sclerosis, enlargement of the heart profile and widening of the mediastinum. Intravenous nitroglycerin was administered to maintain BP between 131~118/81~68 mmHg. The patient was advised to take absolute bed-confinement and oxygen inhalation. At the same time heparin-free haemodialysis was prescribed. Surgical or intervention treatment was recommended but the patient refused and died of AD rupture in a week.

Figure 1: CT scan: aortic dissection (arrow).

Figure 2: CT 3D imaging: aortic dissection.

Case 2

A 70-year-old Chinese male who had been on haemodialysis for 8 months at thrice-a-week regular intervals came to the hospital because of sudden onset of severe tearing chest pain. He had a 20-year history of smoking and a 15-year history of hypertension. Hypertension was not well-controlled with the maximum systolic pressure reaching 200 mmHg. He also had history of myocardial infarction 2 years ago and used aspirin routinely. Chest X-ray suggested widening of the mediastinum. Contrast-enhanced CT scan suggested thickening of the aortic arch and thoracic aorta, scattered calcifications on the vascular wall, and an intimal flap moving medially in the lumen forming a true and a false double cavity. Partial contrast filling was observed in the false cavity. The diagnosis of DeBakey III AD was made (**Figure 3**).

Figure 3: CT 3D imaging: aortic dissection.

After admission, the patient was administered Dolantin for analgesia and intravenous nitroglycerin to maintain BP. Heparin-free haemodialysis was performed. Endovascular graft exclusion was performed, and covered stents were implanted in the aortic arch and the thoracic aorta (**Figure 4**). Postoperative treatment included intensified BP control, maintenance haemodialysis using low-molecular-weight heparin for anti-coagulation, and administration of anti-platelet drugs. The patient has been followed up for three years, showing a stable general condition.

Figure 4: CT 3D imaging: post stent implantation.

Discussion

Aortic dissection is a rare but serious and life-threatening clinical condition in which blood flows into the damaged aortic wall. It is reported that tearing of the aortic wall does not stop until the eventual development of a fatal rupture in more than 60% acute AD cases [2]. Patients on maintenance haemodialysis are often associated with hypertension, lipid and calcium phosphorus metabolic disorders, arteriosclerosis, and hemodynamic change, all of which may contribute to the occurrence of AD [3-5]. Takeda et al. [6] investigated the cause of death in 113 chronic dialysis patients who died during a 10-year follow-up period. Their autopsy study revealed that 5

(5.4%) of these patients died of AD, which is high than 0.59% in general populations.

Once the diagnosis of AD is confirmed, appropriate conservative treatment is the first consideration before further treatment is instituted. Conservative treatment mainly includes lowering the LV systolic pressure to 110 mmHg, reducing HR and analgesia [7]. In De Bakey I or II AD, surgical resection of the ascending aorta is standard management. For De Bakey III AD, medical conservative treatment or intervention therapy is an alternative option [2]. Bleeding from the ruptured AD is the severe complication of AD. Zheng et al. [8] reported an AD patients presenting with myocardial ischemia was misdiagnosed as myocardial infarction and died of AD rupture after administration of anti-coagulants and thrombolytic therapy. Jing et al. [9] observed 13 AD patients with co-existence of coronary artery diseases who underwent endovascular graft exclusion followed by use of anticoagulants during percutaneous transluminal intervention (PCI) and subsequent long-term administration of anti-platelet therapy after surgery. Their 11-month follow-up study showed that anticoagulants and thrombolytic therapy were safe in patients who underwent endovascular stent graft exclusion. There is no guideline about how to manage in HD patients diagnosed with AD. Ouniss et al. [10] reported conservative treatment and conversion from haemodialysis to peritoneal dialysis in three haemodialysis patients when they developed AD, thus avoiding the use of anti-coagulants during haemodialysis and reducing the impact of haemodialysis-related hemodynamic change on the aorta. No AD rupture occurred during the surviving period of the patient after conversion to peritoneal dialysis. Both patients reported herein had a long history of hypertension, poor BP control, arteriosclerosis, vascular calcification, and smoking, all of which are susceptible factors to AD. The patient in Case 1 refused to receive surgical or intervention treatment after confirmation of the diagnosis and died of AD rupture in a week. The patient in Case 2 had a history of myocardial infarction and long-term aspirin administration. Stenting was performed after confirmation of the diagnosis. He is still maintained on haemodialysis and receives 100 mg daily aspirin. His general condition has been good so far.

In summary, haemodialysis patients are often associated with various susceptible factors of AD, in whom the incidence of AD is higher than that in general populations. If a haemodialysis patient suddenly complains of severe and persistent chest pain, AD should be highly suspected. For haemodialysis patients with confirmed diagnosis of AD, surgery or endovascular stent graft exclusion should be considered, especially for the patients who didn't want to change the dialysis mode. As in maintained

haemodialysis, hemodynamic changes and use of anticoagulants are likely to lead to deterioration and poor prognosis of AD. For those who choose conservative treatment, conversion to peritoneal dialysis should be considered.

Conflict of Interest

No conflict of interest was declared by the authors.

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