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An Atypical Case of Silent Aortic Dissection in a Peritoneal Dialysis Patient: A Case Report and Review of Literature

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Patient: Male, 55
Final Diagnosis: Type-A aortic dissection
Symptoms: Exertional dyspnea • orthopnea
Medication: —
Clinical Procedure: Emergent surgical repair with mesh implant
Specialty: Cardiology

Objective: Unusual clinical course

Background: Aortic dissection presents with acute chest or back pain and is associated with high mortality. We present a case of aortic dissection with an atypical presentation in a peritoneal dialysis patient, and the challenges met with peritoneal dialysis.

Case Report: A 53-year-old African American male presented with progressively worsening exertional dyspnea and orthopnea for 3 days without any history of chest pain. His chest x-ray showed mild pulmonary edema. He was admitted with a diagnosis of heart failure. Bedside echocardiogram revealed severe aortic regurgitation and concern for possible aortic dissection. Computed tomography of chest with contrast showed Stanford type-A aortic dissection extending from the aortic valve to the level of the left subclavian artery. Emergent surgery was performed. Postoperatively, the patient was managed in surgical and trauma intensive care unit to keep the blood pressure in the desired range. Initially, he was started on continuous veno-venous hemodialysis and later on transitioned to intermittent hemodialysis. He was switched back to peritoneal dialysis after 6 weeks of surgery.

Conclusions: Atypical presentation of a silent aortic dissection without chest pain in the setting of renal failure and other comorbidities emphasizes that dialysis patients are different from the general population. Sometimes the management needs to be modified from the conventional ways to achieve the high level of success.

MeSH Keywords: Dissection • Hypertension • Peritoneal Dialysis • Uremia

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Background

Aortic dissection (AD) usually presents with sudden onset of severe "tearing" chest or back pain which may be accompanied by vomiting, sweating, or lightheadedness [1,2]. AD is a surgical emergency which can be catastrophic within minutes to hours [2]. It has a high associated mortality of 33%, 50%, and 75% at 24 hours, 48 hours, and 2 weeks respectively [3]. We present this rare case of an AD with atypical presentation, and the challenges met with peritoneal dialysis (PD). There is scarce literature on the incidence, etiology, and treatment options of AD in PD patients.

Case Report

A 53-year-old African American male with a history of human immunodeficiency virus infection (HIV) controlled on treatment, chronic kidney disease stage5d (CKD5d) on PD since 2016 and renal transplant waitlist, uncontrolled hypertension, obstructive sleep apnea, and non-ischemic cardiomyopathy (ejection fraction [EF] of 50–55% on transthoracic echocardiography and EF of 38% on nuclear stress test) presented to the Emergency Room with worsening orthopnea and exertional dyspnea for 3 days. His initial examination was significant for blood pressure (BP) 140/80 mm Hg, heart rate100 bpm, temperature 36.7°C (98.0°F), respiratory rate 18 breaths/minute and O_2 saturation 98% on room air. Lung auscultation revealed bilateral rales. The rest of his physical examination was within the reasonable limits. Electrocardiogram only showed sinus tachycardia and prolonged corrected QT interval of 530 milliseconds. Chest x-ray showed mild pulmonary edema with normal aortic diameter. He was admitted for heart failure management due to PD failure and was diuresed and dialyzed with a Dianeal 4.25% solution with net ultrafiltration of 1200 mL. The next morning, cardiac auscultation uncovered loud III/IV diastolic murmur at the left parasternal border. Urgent echocardiogram showed severe aortic regurgitation. Computed tomography (CT) chest with contrast confirmed Stanford type-A AD extending from the aortic valve to the level of the left subclavian artery (Figures 1–3). Emergent surgical repair of the ascending AD with mesh repair and intraoperative transesophageal echocardiography was performed. The patient was subsequently managed in the surgical and trauma intensive care unit, requiring epinephrine and dopamine infusions initially, and then nicardipine and nitroglycerin infusions to maintain BP. He initially received continuous veno-venous hemodialysis (CVVHD) for volume control which was later switched to intermittent hemodialysis (iHD) and subsequently discharged on iHD. Since the patient had a strong history of uncontrolled hypertension, his AD was attributed to it, and no additional workup for other causes of AD was performed. Six weeks after surgery, he was switched back to PD. It has been 10 months since his surgery; he continues to do well on PD without any complications.

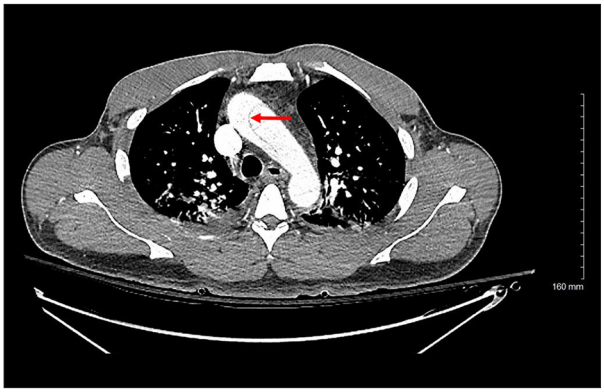

Figure 1. Computed tomography scan with contrast with transverse plane showing dissection across the aortic arch (red arrow).

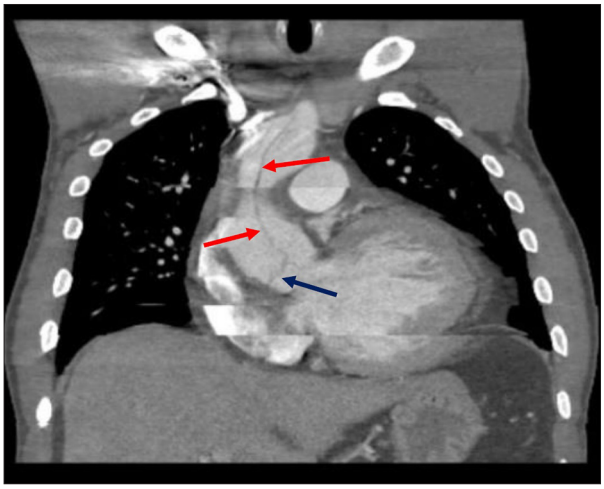

Figure 2. Computed tomography scan with contrast with coronal plane showing dissection of the ascending aorta and the aortic arch (red arrows) and dissection of the aortic valve (blue arrow).

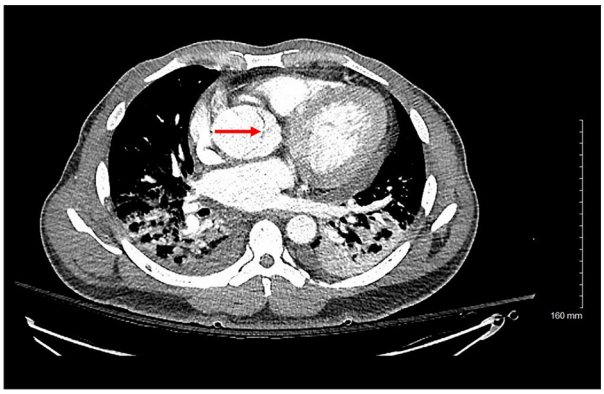

Figure 3. Computed tomography scan with contrast with transverse plane showing dissection across the aortic valve and the ascending aorta (red arrow).

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Table 1. Risk associated with an AD in PD patients leading to HD.

Potential complications	Why HD and not PD
Cardiopulmonary bypass surgeries are associated with substantial volume infusion including fluids and blood products	PD is not an effective method for ultrafiltration and fluid removal, therefore, CVVHD and then HD was employed [19–22]
Extension of the AD into abdominal aorta can lead to AAA formation	AAA can interfere with the PD catheter and prevent effective PD
Impaired wound healing	Due to increased abdominal pressure
Peri-operative hypertension in type-A AD is associated with worse outcomes	PD is not an effective method for ultrafiltration and fluid removal for blood pressure control

AD – aortic dissection; CVVHD – continuous veno-venous hemodialysis; HD – hemodialysis; PD – peritoneal dialysis; AAA – abdominal aortic aneurysm.

Discussion

The incidence of an acute AD is between 2.6 to 3.5 per 100 000 person-years, while in-hospital mortality is 27.4% [4,5]. The clinical presentation includes aortic regurgitation, cardiac tamponade, and end-organ ischemia, but sudden severe chest pain is the most common symptom [6]. AD is a dynamic process and presentation can differ significantly depending on the severity, type of vessel involved, and extension into the false lumen, and it can be confused with other cardiac conditions.

The pathophysiology of the AD in CKD5d patients depends on several factors leading to arterial wall weakening [7]. These patients generally have intrinsic arterial wall weakness which causes its remodeling, leading to hypertrophy, stiffness, diffuse dilatation, and aneurysmal formation which contributes to the acute AD [8]. Stanford type-A dissection involves ascending or ascending and descending aorta (Figure 2) and requires urgent surgical intervention because of the high associated mortality: approximately 1% per hour for the first 48 hours [9]. Stanford type-B involves descending aorta and is mostly managed with BP control. Preoperative antihypertensive treatment is associated with better survival, whereas patients with preoperative normotension or hypotension who were not candidates for antihypertensive treatment have worst survival. Beta blockers are traditionally used in patients with an AD, but nitrates are believed to be the most protective drugs in reducing the left ventricular contractility and in turn reducing the aortic tension [10,11].

Type-A AD predominantly presents with anterior chest pain and atypical symptoms such as orthopnea or exertional dyspnea. Back pain is usually the presenting complaint in a type-B AD. Major risk factors include increasing age, hypertension, diabetes mellitus, atherosclerosis, and renal failure [12,13]. The strong association of elevated blood urea nitrogen with an aortic aneurysm has led to its rising prevalence in patients with renal failure awaiting renal transplant [14,15]. Patients who are dialysis dependent present atypically and require a high index of suspicion to make a diagnosis [16]. Our patient is a typical case of atypical presentation of an acute AD and was at high-risk based on the aforementioned risk factors, and presented without any chest pain.

CT scan with contrast, magnetic resonance imaging (MRI), transesophageal echocardiography (TEE), or angiography can help in diagnosis. The CT scan with contrast is extensively used due to its high sensitivity and specificity and its easy availability[17], but it is challenging to give contrast to patients with substantial residual renal function. MRI has the highest sensitivity and specificity but is less readily available. Bedside TEE is preferred in hemodynamically unstable patients [18].

Management is directed at hemodynamic stability via analgesia and BP control using beta-blockers or nitrates. In our patient, PD was another intervention which required consideration for accompanying risks after thoracotomy for aortic repair (Table 1). Initial management with CVVHD, which is an efficient way of removing fluid [19–22], and later on iHD for 6 weeks post-surgery, was an effective bridge before reinstituting PD.

Mortality risk is highest in the first 2 years after an acute event, and individuals should be followed carefully during this period [23]; 29% of late deaths are due to rupture of either a dissecting aneurysm or another aneurysm. The management guidelines for prevention of long-term complications include BP control (<120/80 mm Hg), serial imaging at 3, 6, and 12 months and annually to identify re-dissection or aneurysm formation, and evaluation of high-risk conditions [18,24]. It is important to remember that all these recommendations are for the general population who develop AD. Currently there is very limited literature available and there are no guidelines addressing how to deal with patients who are dialysis dependent and develop AD.

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Conclusions

Our case was of a silent AD in a renal failure patient where diagnosis could have been confused with heart failure or peritoneal dialysis failure due to the atypical presentation. This case was challenging with regards to diagnosis and management of PD in the absence of extensive literature and guidelines. In similar cases, the conventional management should be modified to achieve the highest level of success, as our patient who was successfully managed with iHD.

References:

1. Nienaber CA, Clough RE: Management of acute aortic dissection. Lancet. 2015; 385(9970): 800–11
2. White A, Broder J, Mando-Vandrick J et al: Acute aortic emergencies – part 2: Aortic dissections. Adv Emerg Nurs J, 2013; 35(1): 28–52
3. Wiesenfarth JM: Acute aortic dissection. Medscape. 2016 (Updated: Dec 28, 2017) Available from: URL: *https://emedicine.medscape.com/article/756835-overview*
4. Clouse WD, Hallett JW, Schaff HV et al. (eds.), Acute aortic dissection: population-based incidence compared with degenerative aortic aneurysm rupture. Mayo Clinic Proceedings, Elsevier, 2004
5. Meszaros I, Morocz J, Szlavi J et al: Epidemiology and clinicopathology of aortic dissection: A population-based longitudinal study over 27 years. Chest, 2000; 117(5): 1271–78
6. Hagan PG, Nienaber CA, Isselbacher EM et al: The International Registry of Acute Aortic Dissection (IRAD): New insights into an old disease. JAMA 2000; 283(7): 897–903
7. London GM, Guerin AP, Marchais SJ et al: Cardiac and arterial interactions in end-stage renal disease. Kidney Int, 1996; 50(2): 600–8
8. London GM, Drüeke TB: Atherosclerosis and arteriosclerosis in chronic renal failure. Kidney Int, 1997; 51(6): 1678–95
9. Nienaber CA, Eagle KA: Aortic dissection: New frontiers in diagnosis and management. Circulation, 2003; 108(5): 628–35
10. Brennan KB, Aggarwal S, Szeto WY et al: Blood pressure and antihypertensive therapy predict outcomes in acute aortic dissection. Anesthesiology annual meeting: American Society of Anesthesiologists; Anesthesiology annual meeting, 2009; Available from: *http://www.asaabstracts.com/strands/asaabstracts/abstract.htm?absnum=1595&index=4&year=2009*
11. Cheung AT, Aggarwal S, Augoustides JG et al: Perioperative renal failure predicts outcome in stanford type A aortic dissection. Risk, 1992; 12(13): 5
12. Hagan PG, Nienaber CA, Isselbacher EM et al: The International Registry of Acute Aortic Dissection (IRAD): New insights into an old disease. JAMA, 2000; 283(7): 897–903
13. Mehta RH, O'Gara PT, Bossone E et al: Acute type A aortic dissection in the elderly: Clinical characteristics, management, and outcomes in the current era. J Am Coll Cardiol, 2002; 40(4): 685–92
14. Merion RM, Pelletier SJ, Goodrich N et al: Donation after cardiac death as a strategy to increase deceased donor liver availability. Ann Surg, 2006; 244(4): 555–62
15. Port FK, Merion RM, Roys EC, Wolfe RA: Trends in organ donation and transplantation in the United States, 1997–2006. Am J Transplant, 2008; 8(4 Pt 2): 911–21
16. Tong YQ, Wang Q, Hou ZH: Aortic dissection and hemodialysis: A case report. Dialysis & Transplantation, 2007; 36(10): 543–51
17. Nienaber CA, von Kodolitsch Y, Nicolas V et al: The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. N Engl J Med, 1993; 328(1): 1–9
18. Tsai TT, Fattori R, Trimarchi S et al: Long-term survival in patients presenting with type B acute aortic dissection. Circulation, 2006; 114(21): 2226–31
19. Siddiqui W, Kohut A, Hasni S et al: Readmission rates after acute decompensated heart failure. J Mol Cel Cardiol, 2017; 112: 155
20. Siddiqui WJ, Kohut AR, Hasni SF et al: 90 days readmission in acute decompensated heart failure – a systematic review and meta-analysis. J Card Fail, 2017; 23(8): S85
21. Siddiqui WJ, Kohut AR, Hasni SF et al: Readmission rate after ultrafiltration in acute decompensated heart failure: A systematic review and meta-analysis. Heart Fail Rev, 2017; 22(6): 685–98
22. Siddiqui WJ, Vela-Ortiz M, Goldman J, Aggarwal S: Ultrafiltration vs. conventional diuretics in acute decompensated heart failure – a meta analysis. Am J Kidney Dis, 2017; 69(4): A91
23. Pansini S, Gagliardotto PV, Pompei E et al: Early and late risk factors in surgical treatment of acute type A aortic dissection. Ann Thorac Surg, 1998; 66(3): 779–84
24. Erbel R, Alfonso F, Boileau C et al: Diagnosis and management of aortic dissection. Eur Heart J, 2001; 22(18): 1642–81

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