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

Study Design A EF Data Collection B EF Statistical Analysis C EF Data Interpretation D E Manuscript Preparation E DE 1, Literature Search F BF Funds Collection G BF		ABCDEF 1,2 EF 1 EF 3 E 1 DE 1,2 BF 4 EF 4	Mohammad Harisullah Khan Maryam Khan	 Department of Medicine, Drexel University College of Medicine, Philadelphia, PA, U.S.A. Department of Medicine, Hahnemann University Hospital, Philadelphia, PA, U.S.A. Aga Khan University Medical College, Karachi, Pakistan Department of Medicine, Dow University of Health Sciences, Karachi, Pakistan Department of Medicine, Division of Gastroenterology, Beth Israel Deaconess Medical Center, Harvard University, Boston, MA, U.S.A. 	
		E 5	Aysha Aslam		
		BCDF 1,2	Hasan Arif		
		ABCDF 1,2	Sandeep Aggarwal		
	Corresponding Author: Conflict of interest: Patient:		Waqas Javed Siddiqui, e-mail: dr.waqas20@gmail.com None declared Male, 55 Type-A aortic dissection		
Final Diagnosis:		iagnosis:			
	Sy	mptoms:	Exertional dyspnea • orthopnea		
	Medication:		— Emergent surgical repair with mesh implant		
Clinical Procedure:		ocedure:			
	S	pecialty:	Cardiology		
	C	bjective:	Unusual clinical course		
	Bac	kground:	•	pain and is associated with high mortality. We present a on in a peritoneal dialysis patient, and the challenges met	
		e Report:	A 53-year-old African American male presented with p for 3 days without any history of chest pain. His chest with a diagnosis of heart failure. Bedside echocardio possible aortic dissection. Computed tomography of tion extending from the aortic valve to the level of th Postoperatively, the patient was managed in surgical sure in the desired range. Initially, he was started on sitioned to intermittent hemodialysis. He was switch	progressively worsening exertional dyspnea and orthopnea at x-ray showed mild pulmonary edema. He was admitted gram revealed severe aortic regurgitation and concern for chest with contrast showed Stanford type-A aortic dissec- e left subclavian artery. Emergent surgery was performed. al and trauma intensive care unit to keep the blood pres- continuous veno-venous hemodialysis and later on tran- need back to peritoneal dialysis after 6 weeks of surgery.	
	Con	clusions:	Atypical presentation of a silent aortic dissection with	nout chest pain in the setting of renal failure and other co-	

morbidities emphasizes that dialysis patients are different from the general population. Sometimes the man-

agement needs to be modified from the conventional ways to achieve the high level of success.

MeSH Keywords: Dissection • Hypertension • Peritoneal Dialysis • Uremia

Full-text PDF:







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 O₂ 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).

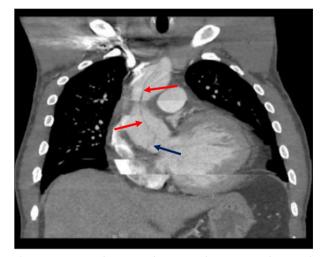


Figure 2. Computed tomography scan with contrast with coronal plane showing dissection of the ascending aorta and the aortic arch (red arrows) and dissection of the aortic valve (blue arrow).

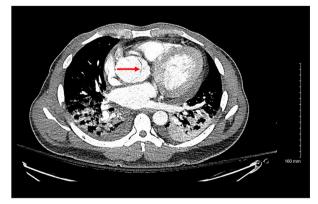


Figure 3. Computed tomography scan with contrast with transverse plane showing dissection across the aortic valve and the ascending aorta (red arrow).

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.

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

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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.

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