

Beyond the Usual Suspects (Aortic Dissection in Patients with Chronic Kidney Disease)

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Abstract

A comprehensive differential diagnosis is essential in the emergency department, even when patient presentations are atypical. Timely recognition of life-threatening conditions, such as aortic dissection, hinges on this critical diagnostic approach.

Keywords

Chest Discomfort, Atypical Presentation of Aortic Dissection, Differential Diagnosis, Acute Type A Aortic Dissection, Aortic Dissection Diagnosis

1. Introduction

This case underscores the critical importance of a comprehensive approach to patients with complex medical histories presenting to the emergency department. While the definitive diagnosis of aortic dissection emerged later in the patient's journey, subtle clues were present that, in hindsight, could have expedited recognition of this life-threatening condition. Aortic dissection should always be considered a differential diagnosis, even in atypical presentations, as early identification is paramount to optimal patient outcomes.

2. Case Presentation

A 54 years Filipino gentleman, presented to the ED with central chest pain for three days that limited his activity. The patient past medical history includes hypertension along with moderate aortic regurgitation due to dilated aortic root and paroxysmal atrial fibrillation on apixaban and carvedilol . The patient had radical nephrectomy due to metastatic renal cell carcinoma six years ago, complicated with cord compression which was treated with radiotherapy and chemotherapy.

On arrival to the ED his chest pain was dull in nature, not radiating and improved with paracetamol. His pain was associated with mild shortness of breath, palpitations, sweating and nausea. He had also decreased his usual oral intake and urine output. The patient recently did not use NSAIDs or was exposed to contrast. He has no recent history of infection nor antibiotic use. Initially he was alert, and oriented. His vitals were within normal reference, except for slight hypotension (**Table 1**).

On examination, the precordium was remarkable only for mild crackles in the base of the left lung. All other systems reviewed were intact. ECG was done in the ED Picked tall T wave with no ST elevation (**Figure 1**). All the following serial ECGs done at the beginning did not record any new changes. The chest radiograph shows cardiomegaly (**Figure 2**).

His laboratory investigations revealed high creatinine of 300 $\mu\text{mol/l}$ (his base line 199 - 200), hyponatremia, Na of 117 mmol/l and high Troponin T. The D-dimer was not ordered at that time (**Table 2**).

The patient was admitted and transferred to the floor. During his hospitalisation, the nephrologist was consulted and he advised for hypertonic saline due to hyponatremia. The CT/KUB ruled out obstructive uropathy but detected right minimal pleural effusion with atelectatic changes of the posterior-basal segments (**Figure 3**).

The differential diagnosis at that time was primarily AKI on top of CKD due to dehydration. The internal medicine was assessed. Later, the patient was admitted under AMU then transferred to the floor again after correction of his hyponatremia. The patient was mildly improved. However, he still has on and off chest pain with shortness of breath, for which the cardiologist was consulted again and diagnosed him with type 2 MI which was treated with supportive management and advised to stop all nephrotoxic medications. The Oncologist advised to hold everolimus and lenvatinib for now.

On the 4th day, the patient's chest pain exaggerated mainly in the left side. The pain increased with exertion. At that time, he was pale and moist, euvoletic, systolic murmur was heard without significant change in ECG (**Table 3**). However, T-trop were increased from 100 to 114 ng/l . He was treated with glyceryl trinitrate spray. Cardiology was consulted for the 3rd time, he advised to continue the same management and follow serial T-trop and serial ECG. Other differential diagnoses at this point are pleuritis and SIADH due to metastatic cancer to the lungs. The viral panel was ordered. Finally Echo was done and detected critical evidence of aortic dissection extending from the aortic root to descending thoracoabdominal aorta (**Figure 4**).

The cardiothoracic surgeon was consulted and he requested an urgent CT thorax with ECG gated. The gated CT thorax shows aortic dissection stanford type A with involvement of the brachiocephalic artery (**Figure 5**).

He was admitted to CT-ICU. An emergency consent for high risk of mortality and morbidity was taken for aortic dissection repair bentall surgery. In the

operating theatre he bled excessively despite the maximum efforts to stop his bleeding, the bleeding continued. A decision was made to keep the chest open and close it after his bleeding stops. He was shifted to CT-ICU, and suddenly he became bradycardic and hypotensive (**Table 4**). He was unresponsive to the maximal inotropic support. Moreover, he was not responding to the fluids and blood products. He developed Asystole. His chest was closed and ECG done to confirm asystole. Death was declared.

Table 1. Vital signs for the patient at the time of presentation.

	At the Frist ED Visit
Oral Temperature	37°C
Respiratory Rate	20 cycle/min
Oxygen saturation	98 on room air
Heart Rate	87 beats /min
Blood Pressure	94/39 mmHg

23-Jul-1969 (54 yr)	Vent rate	77	BPM	SINUS RHYTHM
Male Other	PR interval	208	ms	NORMAL EOG
Room:	QRS duration	102	ms	INLERPRETATION BASED ON A DEFAULT AGE OF 40 YEARS
Loc:15	QT/QTcB	376/426	ms	
	P-RT axes	-8 12 38		UNCONFIRMED REPORT

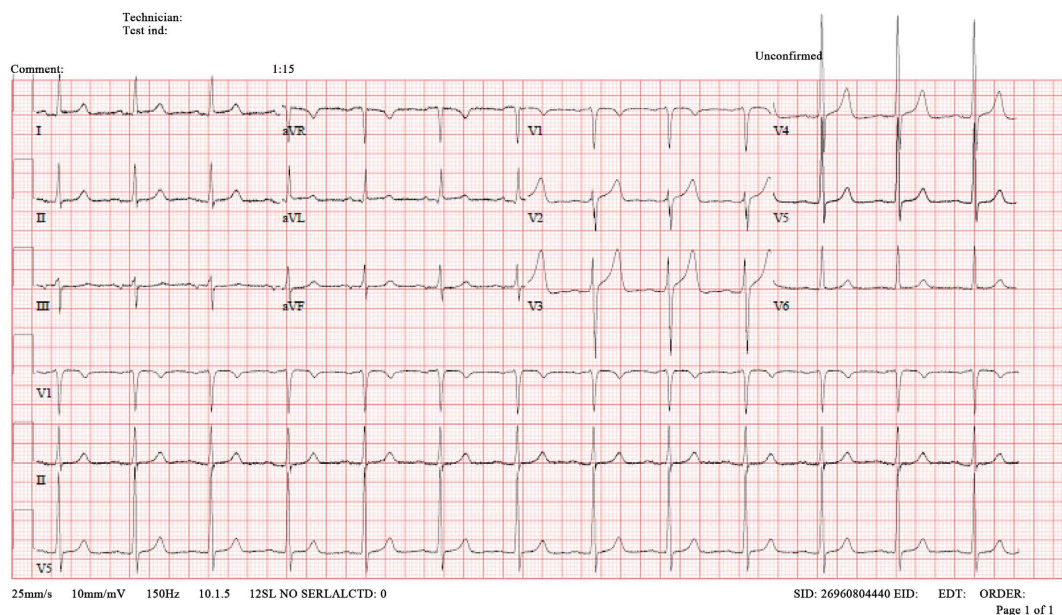


Figure 1. ECG at time in Emergency department, peaked T waves with hypertrophic cardiomegaly element noted in background of HTN.

3. Discussion

Aortic dissection (AAD) is a rare condition but potentially fatal one with a mortality rate reaching 23% (1). pathophysiology mainly occurs by the tearing of the inner layer of the aorta, which will create a false lumen at aorta layers, that will create with time false lumen or aneurysmal stacture, as a consequence, it may be ruptured and patient bleed to death, causing end-organ damage [1].



Figure 2. Chest X-ray at first day at Ed and before admission, showed possible early minimum left side pleural effusion with old dilated aortic root.

Table 2. Patient labs results.

Group	Detail	Value w/unit	Flags	Normal Range
General Haematology	Hgp	12.2 g/dl	Low	11.3 - 17
Blood Chemistry	Creatinine	300 umol/l	High	62 - 106
Blood Chemistry	Sodium	117 mmol/l	Critical Low	133 - 164
Blood Chemistry	Troponin-T HS	81 ng/l	High	3 - 15

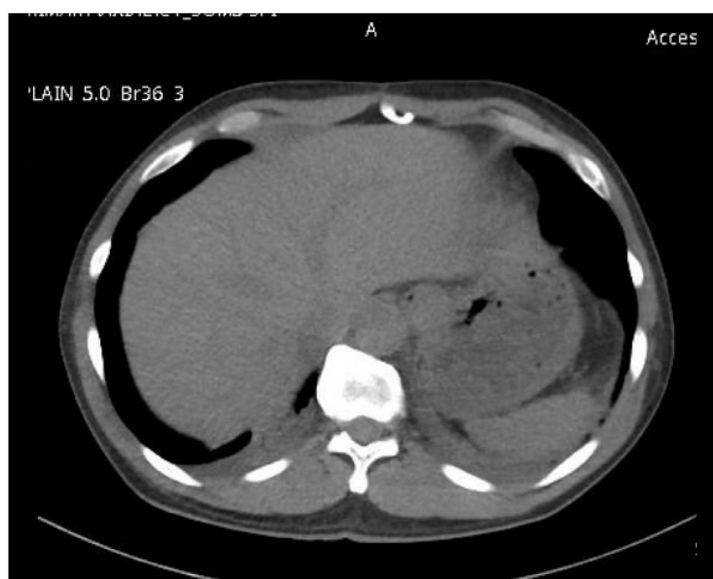


Figure 3. CT-KUB only finding was mild pleural effusion noted mainly at posterior segment.

Table 3. Vital signs for the patient on the 4th day.

Vital Signs	The 4th Day
Oral Temperature	36.7°C
Respiratory Rate	18 cycle/min
Oxygen Saturation	94 simple mask
Heart Rate	85 beats/min
Blood Pressure	129/49 mmHg

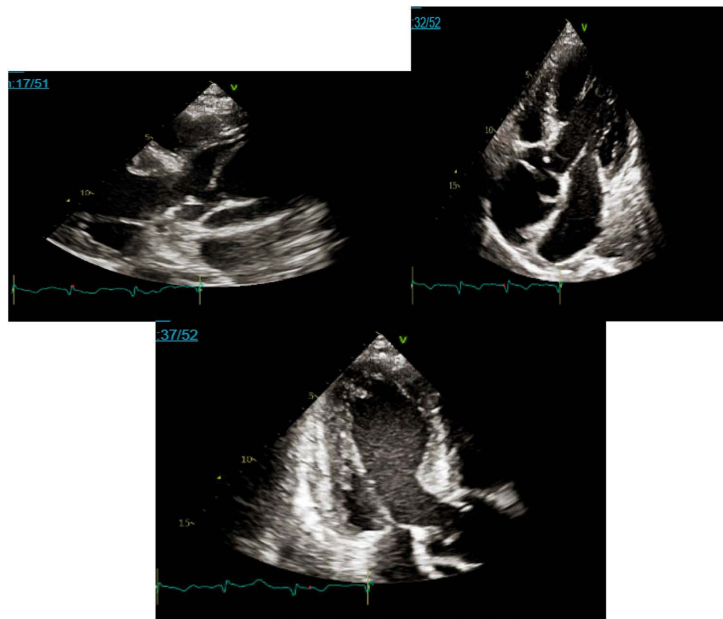


Figure 4. Two views from ECHO showed clearly flap started from aortic root with clear dilation at area.

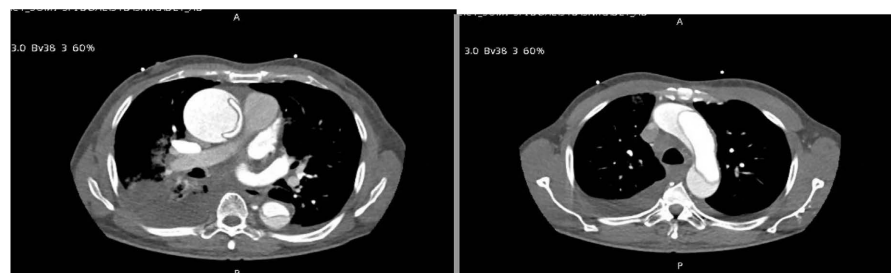


Figure 5. CT gated ECG two views showed the aortic dissection clearly and classified it as A type A standard extended up to abdominal aorta.

Table 4. Vital Signs for the Patient on the 4th day in the AMU.

	CT-ICU
Oral Temperature	36.4°C
Respiratory Rate	34 cycle/min
Oxygen Saturation	95 on room air
Heart Rate	74 beats /min

ED: Emergency Department, AMU: Acute Medical Unit, CT-ICU: Cardiothoracic-Intensive Care Unit.

Despite advancements in surgical and diagnostic Technology, early diagnosis remains challenging due to unclear clinical picture in some patients, atypical presentation, also other comorbidities may present coincidentally at same time of presentation [2], may mask aortic dissection symptoms and make it less likely as a differential diagnosis same what happen in our case.

Type A is a life-threatening one, more serious and mainly originates from ascending aorta in most cases [2], less likely Type B, that may be treated medically and it started from other aortic areas such as descending one [3].

The main symptoms and clinical picture presented with typical chest pain radiating to back, sudden in nature usually, sharp, sever in nature not respond well to analgesia, also with time patient will show signs of hemodynamic instability, hypo or hypertension, tachycardia, tachypnea with neurological manifestation as syncope or stroke symptoms in some cases [4].

In compression to this case with atypical presentation of non-radiating chest pain for more than three days, not severe in nature and relieved with simple analgesia, in hemodynamic stable patient, make the diagnosis of aortic dissection challenged, especially with background medical history of CKD and other comorbidities.

The patient was presented with atypical symptoms at the Emergency department, which raised multiple other differential diagnoses. Mainly common differential diagnoses in this case were ACS, acute coronary syndrome (ACS), pulmonary embolism (PE), fluid overload, particularly in the setting of cAKI on top of CKD that patient had or AKI due to dehydration. Initially, patient laboratory findings of severe hyponatremia with elevated RFTs can further obscure the diagnosis of AAD. Additionally, an initial elevation in troponin T, which did not change significantly over time, even after admission, contributed to diagnostic uncertainty [5].

Diagnostic tools such as (CT) or (MRI), used to aid diagnosis and treatment, cause time really crucial in this condition, any delay can cost patient life, imaging modality mainly will visualise the flap or false aneurysmal strature by following aorta from main origin until last branch and see contrast flow [6], surgical intervention focused mainly in repair aortic layers and to provide good blood perfusion to all organs to avoid any end organ damage [7].

This case highlights the importance of considering AAD as the differential diagnosis of patients with atypical presentations with a background of a lot of comorbidities, even in the absence of classic symptoms or electrocardiographic changes.

Learning points

- AAD should be considered one of the differential diagnoses with atypical presentation.
- The symptoms of AAD have a high variation related to the injured part of the aorta.
- AKI on the top of chronic can be result of extensive AAD.

- Special attention for correct diagnosed should be mix high risk condition with physical examination finding with proper investigation.
- D-dimer may improve the diagnosed in this case but not all high d-dimer considered as MI.
- Cardiac troponin level is a difficult interpret in the CKD, the absolute value change and changes in serial troponin measurement it has high sensitivity.
- Fixed errors day after day of admission can lead to catastrophic results.
- Echo should be one of the ED investigations when ever AAD is suspected.

4. Conclusion

This case underscores the importance of maintaining a high level of suspicion for aortic dissection as a differential diagnosis in patients presenting with atypical chest pain, even when there is a compelling alternative diagnosis, such as myocardial infarction, if the patient had clear risk factors. Early recognition and prompt intervention are crucial for improving patient outcomes.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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