Acute renal failure and hepatocellular damage as presenting symptoms of type II aortic dissection

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SUMMARY

Introduction Pericardial effusion can be a consequence of a number of pathological conditions, and as such it can cause impaired left ventricular filling followed by decreased cardiac output and blood pressure. This kind of hemodynamic compromise and its consequences are extremely uncommon unless pericardial effusion causes tamponade.

Case Outline We describe a very rare case of a 30-year old male patient, with an acute aortic dissection type II causing pericardial effusion without clinical nor echocardiographic signs of tamponade, while presenting with an acute renal and hepatic failure. After initial diagnostic uncertainties, and following final diagnosis of an acute aortic dissection, this patient underwent surgical aortic valve replacement with a satisfactory outcome.

Conclusion It is important to underscore the significance of clinical situation of simultaneously existing acute renal and hepatic failures in the setting of a "non-tamponade" pericardial effusion, following a type II aortic dissection. Although most commonly aortic dissection presents itself with typical clinical symptoms or patient history data, it is not that unusual for it to be hidden in an entirely atypical clinical milieu as the one described in this case.

Keywords: aortic dissection; intimal flap; renal failure; hepatic failure; pericardial effusion

INTRODUCTION

Pericardial effusion is a complication of a number of pathological conditions such as aortic dissection, trauma, malignancies, infections, metabolic disorders, and heart failure [1, 2]. As a consequence of impaired left ventricular diastolic filling, cardiac output and systemic blood pressure are decreased, which in turn leads to reduced hepatic and renal blood flow, thus causing ischemic injury to these organs [1, 3]. These changes are extremely rare if pericardial effusion doesn't cause tamponade.

We describe a very rare case of an acute aortic dissection type II causing pericardial effusion without clinical or echocardiographic signs of tamponade, while presenting with an acute renal failure (ARF) and hepatic insufficiency as well.

CASE REPORT

One day prior to the admission to our hospital, a 30-year-old male patient presented to his primary care clinic complaining of a chest pain spreading to the neck and jaw. He started having these symptoms a couple of days before, and during this period he also became oliguric.

Clinical examination revealed a blood pressure of 120/70 mmHg and 110/65 mmHg (right and left arm, respectively). On auscultation, there was normal breathing, heart sounds were not diminished, while subtle diastolic murmur was heard above the aorta. There was no pulsus paradoxus. Discrete jugular venous distension was visible. The liver wasn't palpable on the right costal margin. There were no signs of skin hemorrhaging.

Electrocardiogram (ECG) showed a sinus rhythm with heart rate of 60 beats per minute and negative T waves in D2, aVF, V5–V6. There were no changes in the ST segment. QRS complex voltage was normal, and electrical alternans was absent.

Initial laboratory test results disclosed raised levels of inflammatory markers, a fall in platelet count, and signs of hepatic and renal failure. Troponin values were also elevated (Table 1). Chest X-ray confirmed widened mediastinum.

There were history data about this patient having prior contact with pesticides, but subsequent toxicology report excluded poisoning as a possible etiology of the liver injury.

Abdominal ultrasound showed that there were no focal hepatic lesions. The kidneys were normal in size. There was no hydronephrosis, and the blood flow pattern, which was measured on the basis of Doppler examination, was also normal. Abdominal aorta dimension was normal and there were no signs of dissection or thrombotic masses in its lumen.

Focused echo examination revealed dilated aortic root and the ascending part of the aorta,

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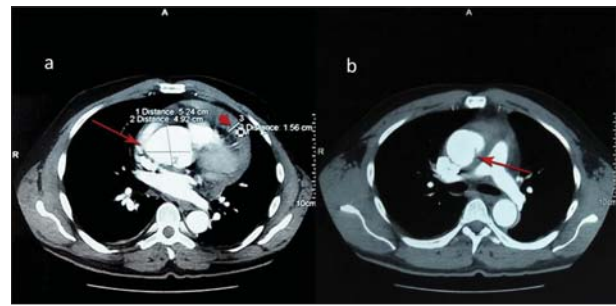Višegradska 26, 11000 Belgrade,
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Table 1. Complete blood cell count and plasma biochemical values on admission and during the critical phase of the illness

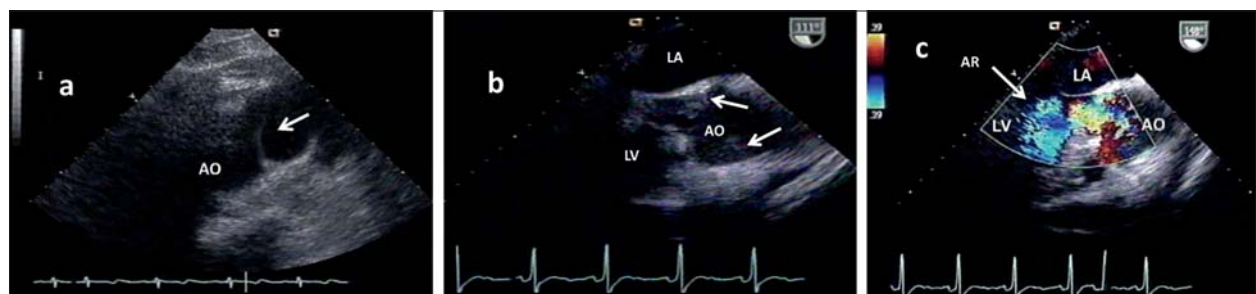| Variables	Time (hours)		Reference range
	8:00	20:00	
White blood cell count	18.5	18.1	3.4–9.7 ×10⁹/L
Red blood cell count	5.2	3.5	4.34–5.72 ×10¹²/L
Hemoglobin	154	107	138–175 g/L
Platelet count	81	37	158–424 ×10⁹/L
Hematocrit	/	0.311	0.415–0.53 L/L
Mean corpuscular volume	/	89.1	83–97.2 fL
Sedimentation rate	55	/	3–15 mm/hour
Aspartate aminotransferase	3,250	3,630	0–37 U/L
Alanine aminotransferase	3,630	5,195	0–41 U/L
Gamma glutamyltransferase	/	66	0–55 U/L
Total bilirubin	9	10	0–20.5 μmol/L
Lactic acid dehydrogenase	6,710	7,780	220–460 U/L
Potassium	3.9	4.4	3.5–5.1 mmol/L
Blood urea nitrogen	13.6	22.7	2.5–7.5 mmol/L
Creatinine	436	551	59–104 μmol/L
Glomerular filtration rate	15	11	>60 mL/min/1.73 m²
International normalized ratio	1.53	1.51	0.8–1.2
Prothrombin time	20	19	10–14
Activated partial thromboplastin time	36.5	43.2	22–32 seconds
C-reactive protein	85	/	0–3 mg/L
Creatine kinase	92	311	0–200 U/L
Creatine kinase MB	31	71	0–24 U/L
Troponin I	5.3	7	>0.30 μg/L
D-dimer	/	24.06	>0.5 mg/L
Fibrinogen	/	3.6	2.0–4.0 g/L

Figure 1. Contrast-enhanced computed tomography: (a) dilated ascending part of the aorta (arrow) with a moderate circular pericardial effusion (arrowhead); (b) poor visualization of an intimal flap (arrow) located in the same segment of the aorta

was referred to the Emergency Care Unit of our tertiary care hospital.

On the admission, physical examination was repeated and it wasn't significantly different from the previous one. Subsequent laboratory tests revealed a further deterioration of the liver and renal function, substantial fall in hemoglobin levels and red blood cell count, as well as additional drop in platelet count. D-dimer value was elevated (Table 1).

Because of the existing ARF, a nephrologist was consulted regarding the safety of repeating a CT scan with intravenous contrast application. Since the patient would most probably need hemodialysis later on, and as time was of the essence, the nephrologist's decision was against performing a CT scan again.

Based on a high clinical suspicion that this was a case of an acute aortic dissection, transthoracic echocardiogram (TTE) followed by a transesophageal echocardiogram (TOE) were performed instead. TTE revealed an intimal membrane located in the ascending part of the aorta. This was most clearly visible only after the patient was positioned in the right lateral decubitus (Figure 2a). Previously noted circular pericardial effusion was also evident. At this point, there were no echocardiographic criteria for tamponade. On TOE, a proximal extension of the dissection was revealed – a very thin intimal flap was identified just above the non-coronary cusp (Figure 2b). The dissection process was traceable up to the origin of the great vessels off the aortic arch. Moderate to severe aortic regurgitation was present (Figure 2c). Also, the aortic wall above the non-coronary cusp appeared to be thinned, so it was

accompanied by a moderate to severe aortic regurgitation. There was also evidence of a moderate circular pericardial effusion. Left ventricular walls were hypertrophic.

This led to a decision to perform a contrast-enhanced computed tomography (CT) scan, which disclosed a dilated ascending part of the aorta (Figure 1a), as well as a structure which was highly suspicious of an intimal membrane located in the same part of the aorta (Figure 1b). This poor visualization of an intimal flap and its atypical configuration were interpreted to be a part of the dissection process, since a moderate pericardial effusion (up to 16 mm) of hemorrhagic density was also noted (Figure 1a).

Since these initial diagnostic findings were considered to be highly suggestive of an aortic dissection, this patient

Figure 2. (a) Transthoracic echocardiogram (right lateral decubitus); an intimal membrane located in the ascending part of the aorta (arrow); (b) transesophageal echocardiogram; proximal extension of the dissection – thin intimal flap (arrows) originating just above the non-coronary cusp with a thinned aortic wall in that segment; (c) aortic regurgitation (arrow)
AO – aorta; AR – aortic regurgitation; LA – left atrium; LV – left ventricle

considered to be a possible place of communication with the pericardial sac (Figure 2b).

Urgent aortic root replacement surgery using the Cabrol technique was performed [4]. Within the next couple of hours the patient's urine output started to increase. In the upcoming days, his renal function started to normalize, which was followed by his other laboratory results returning to normal as well.

Postoperative echo examination, performed four weeks after the procedure, showed a properly functional prosthetic aortic valve. Peak velocity and pressure gradient across the valve were 2.68 m/s and 28 mmHg, respectively, while mean pressure gradient was 17 mmHg. Mild transvalvular aortic regurgitation was present. Graft that was placed in the ascending part of the aorta was also visible. There was no pericardial effusion.

DISCUSSION

Current literature is very limited regarding case reports of a type II aortic dissection presenting both with symptoms of an ARF and hepatocellular damage occurring as a consequence of several pathologic mechanisms inducing malperfusion and ischemic injury to these organs.

Laboratory tests were reflective of the setting of an acute aortic dissection: elevated values of systemic inflammatory biomarkers (sedimentation rate, white blood cell count, C-reactive protein, D-dimer), as well as raised troponin levels [5, 6, 7]. Extremely low platelet count, prolonged prothrombin time, and elevated D-dimer value could be explained by an existing acute liver injury and beginning of a consumptive coagulopathy [8].

An aortic dissection process itself is characterized by elevated D-dimer values, independent of consumptive coagulopathy. For this reason, measuring D-dimer values is an integral part of laboratory testing and flowchart for diagnosis and decision-making in acute aortic syndrome [9]. This kind of biochemical testing is most suitable for ruling out an acute aortic dissection in patients with a low likelihood of the disease [9].

The acute hemorrhagic state that this patient was in was to some extent a result of blood loss both into the pericardium and a false lumen. Some reports describe the concept of platelet dysfunction (impaired platelet aggregation) as additional contributing factor for the state of altered hemostasis and bleeding tendency [8]. We think that endothelial and liver injury were both contributive to the impairment of hemostasis in this case.

Decreased glomerular filtration rate and elevated blood urea nitrogen and creatinine levels were a clear consequence of the existing ARF. His rising aspartate aminotransferase and alanine aminotransferase levels were indicative of a shock liver [10].

Malperfusion syndrome occurs in up to 30% of patients with acute aortic dissection [9]. It is known that pericardial effusion can cause impaired left ventricular diastolic filling, which in turn leads to decreased cardiac output and systemic blood pressure. These hemodynamic changes can ultimately result in a reduced renal and hepatic blood flow, thus causing ischemic injury to these organs [1, 3]. It is, however, important to emphasize that pericardial effusion is not the only pathologic factor contributing to the malperfusion of visceral organs. In this particular case, the other key mechanism of renal and hepatic damage was the dynamic compression of the true lumen due to high pressure blood accumulation in the false lumen, which is the result of a large proximal inflow into the false lumen and insufficient outflow into the distal aorta [9]. Moderate to severe aortic regurgitation furthermore lowered the effective stroke volume, thus also contributing to hypoperfusion of visceral organs.

Since this patient developed symptoms a couple of days before being admitted to his primary care clinic, it is possible that fast accumulation of even a small amount of blood in pericardium initially led to substantial hemodynamic instability, which later entered into spontaneous resolution. This could explain the fact why this patient did not develop clinical signs of cardiac tamponade. Still, we hypothesize that even without echocardiographic or clinical signs of tamponade, this moderate pericardial effusion played an important role in creating a hemodynamic compromise of systemic circulation with the occurrence of both hepatic and renal failure.

ARF can additionally be explained with possible hormonal changes. Decreased left ventricular trans-mural pressure causes a decrease in atrial natriuretic peptide secretion [11]. Also, increased secretion of antidiuretic hormone in the setting of pericardial effusion has been noted in animal studies [12, 13]. Its physiologic effect implies to renal vasoconstriction and volume retention. Since this was probably a case of unrecognized primary hypertension, it is plausible that this patient suffered from a subclinical form of hypertensive renal impairment. This could have facilitated exacerbation of renal failure because of altered hemodynamics secondary to the pericardial effusion [1]. Liver damage can also be explained by the existing venous hypertension and congestion, which led to the mechanical stretch of hepatocytes.

Regarding the incomplete result of the CT scan, there are a number of pitfalls that can cause a diagnostic problem. Some of these are related to technical factors like the improper timing of contrast material administration relative to image acquisition, which in turn results in poor visualization of an intimal flap and a false negative diagnosis [14]. In this particular case this could have been one of the explanations, since manual injection of contrast material was used, rather than the automated intravenous contrast injection which has the ability of applying high flow rates. Conventional spiral CT angiography of the aortic root is also prone to cardiac and aortic wall motion artifacts [14]. Here, a non-ECG-gated recording of these structures was done, leaving it liable to mistakes regarding the dynamic changes of aortic root dimensions and its shape throughout the cardiac cycle [15]. It is also possible that the proximal extension of the dissection was overlooked since it was located immediately above the non-coronary cusp and thus disguised as part of the aortic valve apparatus.

In this case report we also wanted to emphasize the significance of TTE, in particular its right lateral decubitus as a mandatory part of the ascending aorta examination, since that was the position where the first clear image of an intimal flap was obtained from.

TOE was also a diagnostic method of choice in this case due to the state of ARF that this patient was in, since another contrast application would deteriorate renal function even more.

One of the key learning points of this case report is to underscore the importance and raise awareness about clin-ical situations of simultaneously existing ARF and hepatic failure, in the setting of a "non-tamponade" pericardial effusion, following a type II aortic dissection.

Although most commonly aortic dissection presents itself with typical clinical symptoms or patient history data, it is not that unusual for it to be hidden in entirely atypical clinical milieu as the one described in this case. Therefore, aortic dissection should be thought of as a "master of disguise" and should always cross the clinician's mind, especially in clinical settings that seem ambiguous.

REFERENCES

1. Gluck N, Fried M, Porat R. Acute renal failure as the presenting symptom of pericardial effusion. Intern Med. 2011; 50(7):719–721. [DOI: 10.2169/internalmedicine.50.4426] [PMID: 21467704]
2. Nikolic S, Atanasejevic T, Popovic V. Tamponada srca usled rupture zida aorte na mestu zaceljenog rascepa – prikaz slučaja. Srp Arh Celok Lek. 2007; 135(3-4):212–215. [DOI: 10.2298/SARH0704212N] [PMID: 17642465]
3. Casale PN, Fifer MA, Graham RM, Palacios IF. Relation of atrial pressure and plasma level of atrial natriuretic factor in cardiac tamponade. Am J Cardiol. 1994; 73(8):610–613. [DOI: 10.1016/0002-9149(94)90346-8] [PMID: 8147312]
4. Kourliouros A, Soni M, Rasoli S, Grapsa J, Nihoyannopoulos P, O'Regan D, et al. Evolution and current applications of the Cabrol procedure and its modifications. Ann Thorac Surg. 2011; 91(5):1636–1641. [DOI: 10.1016/j.athoracsur.2011.01.061] [PMID: 21444065]
5. Eggebrecht H, Naber CK, Bruch C, Kroger K, von Birgelen C, Schmermund A, et al. Value of plasma fibrin D-dimers for detection of acute aortic dissection. J Am Coll Cardiol. 2004; 44(4):804–809. [DOI: 10.1016/j.jacc.2004.04.053] [PMID: 15312863]
6. Tsai TT, Nienaber CA, Eagle KA. Acute aortic syndromes. Circulation 2005; 112(24):3802–3813. [DOI: 10.1161/CIRCULATIONAHA.105.534198] [PMID: 16344407]
7. Braverman AC. Acute aortic dissection: clinician update. Circulation 2010; 122(2):184–188. [DOI: 10.1161/CIRCULATIONAHA.110.958975] [PMID: 20625143]
8. Tanaka M, Kawahito K, Adachi H, Ino T. Platelet dysfunction in acute type A aortic dissection evaluated by the laser light-scattering method. J Thorac Cardiovasc Surg. 2003; 126(3):837–841. [DOI: 10.1016/S0022-5223(03)00734-7] [PMID: 14502163]
9. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). Eur Heart J. 2014; 35(41):2873–2926.
10. Khan R, Gessert C, Bockhold S. Pericardial effusion presenting with anuric acute renal failure and hepatocellular damage. J Hosp Med. 2009; 4(1):68–70. [DOI: 10.1002/jhm.381] [PMID: 19140198]
11. Hynynen M, Salmenpera M, Harjula AL, Tikkanen I, Fyhrquist F, Heinonen J. Atrial pressure and hormonal and renal responses to acute cardiac tamponade. Ann Thorac Surg. 1990; 49(4):632–637. [DOI: 10.1016/0003-4975(90)90313-U] [PMID: 2138878]
12. Stokhof AA, Overduin LM, Mol JA, Rijnberk A. Effect of pericardiocentesis on circulating concentrations of atrial natriuretic hormone and arginine vasopressin in dogs with spontaneous pericardial effusion. Eur J Endocrinol. 1994;130(4):357–360. [DOI: 10.1530/eje.0.1300357] [PMID: 8162164]
13. Saklayen M, Anne VV, Lapuz M. Pericardial effusion leading to acute renal failure: two case reports and discussion of pathophysiology. Am J Kidney Dis. 2002; 40(4):837–841. [DOI: 10.1053/ajkd.2002.35697] [PMID: 12324921]
14. Batra P, Bigoni B, Manning J, Aberle DR, Brown K, Hart E, et al. Pitfalls in the diagnosis of thoracic aortic dissection at CT angiography. Radiographics. 2000; 20(2):309–320. [DOI: 10.1148/radiographics.20.2.g00mc04309] [PMID: 10715333]
15. de Heer LM, Budde RP, Mali WP, de Vos AM, van Herwerden LA, Kluin J. Aortic root dimension changes during systole and diastole: evaluation with ECG-gated multidetector row computed tomography. Int J Cardiovasc Imaging. 2011; 27(8):1195–1204. [DOI: 10.1007/s10554-011-9838-x] [PMID: 21359833]

Акутна бубрежна и хепатична инсуфицијенција као презентујући симптоми аортне дисекције типа II

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КРАТАК САДРЖАЈ

Увод Перикардни излив може настати као последица низа патолошких стања и довести до смањеног дијастолног пуњења леве коморе, што даље води смањењу минутног волумена и артеријске тензије. Овакав хемодинамски компромис циркулације и његове последице су узузетно ретки уколико перикардни излив не изазове тампонаду срца.

Приказ болесника Акутно настала аортна дисекција типа II код болесника старог 30 година довела је до појаве перикардног излива који није био компликован клиничким ни ехокардиографским знацима тампонаде срца, али се презентовала знацима акутне реналне и хепатичне инсуфицијенције. Након спроведене дијагностике, болесник је подвргнут хитној хируршкој интервенцији замене аортне валвуле са задовољавајућим исходом.

Закључак Важно је истаћи значај клиничке слике у којој истовремено постоји акутна бубрежна и хепатична инсуфицијенција на терену аортне дискеције типа II компликоване перикардним изливом без знакова тампонаде срца. Аортна дисекција се често презентује типичним клиничким симптомима и уобичајеним анамнестичким подацима. Клинички куриозитет представља њена појава и последице настале у околностима приказаним у овом случају.

Кључне речи: аортна дисекција; интимални флап; renална инсуфицијенција; хепатична инсуфицијенција; перикардни излив