ISSN 2379-4046

EMERGENCY MEDICINE
Open Journal

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Case Report

From Neck Pain to a Life-Threatening Condition: A Case Report

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Article information
Received: May 9th, 2024; **Revised:** May 16th, 2024; **Accepted:** May 16th, 2024; **Published:** May 17th, 2024

Cite this article
Vandewoude F, Verstraete S. From neck pain to a life-threatening condition: A case report. [In press]. *Emerg Med Open J*. 2024; 10(1): 4-10.
doi: 10.17140/EMOJ-10-174

ABSTRACT

A 30-year-old man presented with sudden neck pain radiating to his chest. He appeared anxious, pale, and clammy. Despite unremarkable history and clinical examination, elevated D-dimers led to a computed tomography pulmonary angiography (CTPA), which ruled out pulmonary embolism but revealed a widened ascending aorta. Further family history indicated significant vascular disease in the patient's father. Initial treatment included analgesia and control of heart rate and blood pressure. The patient was then transferred to a cardiothoracic center, where a detailed CT aortogram and transesophageal echocardiography confirmed an acute type A aortic dissection. This case underscores the diagnostic challenges of aortic dissection, highlighting the need for thorough clinical evaluation and suggesting future use of tools like the Aortic Dissection Detection-Risk Score (ADD-RS) with D-dimers for guidance.

Keywords
Emergency medicine; Case report; Aorta; Dissection.

INTRODUCTION

Neck pain is a frequent complaint among patients. The estimated prevalence is about 27 per 1,000.[1] Rarely, neck pain is a sign of acute severe pathology. There is a link between anxiety and the prevalence of neck pain.[2] Several guidelines have the principle of red flags,[3-5] but the majority of guidelines do not consider thoracic symptoms. This case report describes a case of a person with acute neck pain radiating to the thoracic and neck region.

CASE DESCRIPTION

A 30-year-old man presented to the emergency department because of a sudden onset of neck pain. He also felt very anxious. The complaints started during lunch at work. He first went to the infirmary at work. There, the complaints were attributed to hyperventilation. He was advised to go home and get some rest. Since he had never had these complaints before and felt very anxious, he decided to go to the emergency department.

On arrival at the emergency room, the symptoms have since improved. The man describes his pain as a pulling and straining sensation in his neck that radiates to the lower jaw and chest region. The young man repeatedly felt as though something was gravely wrong with him, but he was unable to articulate exactly what was going on. Though he hadn't had a fever, chills, or dyspnea in the previous few days, he still felt extremely weak and nervous. Due to his recent high levels of stress and his dual job responsibilities, he was getting very little sleep.

Vital observations were normal. There was normal blood pressure, pulse, and oxygen saturation. The clinical examination was also unremarkable. He had normal chest sounds. The only thing that stood out was how he looked. This young man with a blank medical history looked very pale and was very clammy and sweaty.

As the patient looked very pale clammy sweaty and there was also thoracic radiation, it was decided to perform further technical investigations. Blood sampling, an electrocardiogram (ECG), and a chest X-ray (RX) were started. Given the vague, atypical story and the notion of thoracic symptoms, D-dimers were determined with

to rule out any underlying pulmonary emboli.

The ECG and RX-thorax could both show no abnormalities. Blood collection results were all normal except elevated D-dimers of 1484 ng/mL. Based on these findings, a computed tomography pulmonary angiography (CTPA) of the thorax was then performed to rule out pulmonary emboli.

Differential Diagnosis

In a young man with complaints of neck pain with radiation to the neck and chest region, the differential diagnosis is very broad. The differential diagnosis in this case becomes even more complex as general symptoms were also present such as anxiety, pallor, and clammy sweating. Table 1 outlines the various possible diagnoses.

Table 1. *Differential Diagnoses of Neck Pain with Radiation to the Thorax and Neck, with Additional General Symptoms of Anxiety, Pallor and Clamminess*

	PRO	CONTRA
Acute coronary syndrome	Radiation to the thorax and neck pale and clammy sweating.	No ECG abnormalities normal troponins No cardiac risk factors
Pericarditis	Radiation to the thorax.	No ECG abnormalities not posture-related No infectious symptoms
Esophageal spasm/Reflux disease	Arise during meals. stress	First episode pale and clammy sweating.
Cervical radiculopathy	Neck pain	Non-posture-related pain acute onset No specific dermatome, pale and clammy sweating.
Pulmonary emboli	Radiation to the thorax and neck pale and clammy sweating increased D-dimers.	No risk factors No breathing-related pain
Aortic pathologys	Thoracic pain increased D-dimers	No typical pain normotensive normal clinical examination
Anxiety or panic attack	Psychological stressors	First episode
Pneumonia	Thoracic pallor	No infectious symptoms

Case Progress

The CT angiography could not detect pulmonary emboli, but it could detect a pathologically widened Aorta. The Aorta ascendens had a width of 5.8 cm (Figure 1). The normal width of the Aorta ascendens is a maximum of 5 cm.[6]

The widened Aorta is suspicious for aortic pathology but does not yet mean acute aortic dissection or acute rupturing aneurysm. In the context of this patient with the atypical neck pain with thoracic radiation and on the other hand, the pale and clammy appearance does make acute aortic pathology more likely.

At the time of the initial presentation and first contact with the doctor, no family history had been performed. Clinically, the patient had no features that initially made the doctor think of Marfan syndrome. Which is frequently related to connective tissue and aortic pathology. Once the widened ascending aorta was identified, an additional family history was performed. The patient's father was diagnosed with 2 serious vascular disorders in the past, namely an Arteria carotid dissection and a ruptured aneurysm of the Arteria Lienalis. At the time there was a strong suspicion of aortic dissection, the patient was in a non-cardiosurgical hospital and initial therapy had not yet been started. Simultaneously, the initial medical treatments were started and the intensive care unit of a cardio-surgical center was contacted.

The patient was hemodynamically stable but was still clearly in pain. Pain relief was started by intravenous morphine. Labetalol and Nicardipine were then administered continuously *via* an infusion pump. The administration of the analgesia, beta-blocker, and a calcium antagonist was intended to keep heart rate and blood pressure on the relatively low side to prevent the expansion of the dissection zone. An urgent medically assisted transport to a cardiosurgical center was arranged. A CT aortogram was performed (Figure 2).

This could visualize the Aorta better than the initial CT pulmonary angiography. This examination showed, in addition to the widened Aorta ascendens, a dissection with an intimal flap/tear, which had an oblique anterior-posterior course. The tear ran from the Aorta Ascendens to the origin of the left arteria subclavia. Transesophageal echocardiography (TEE) (Figure 3) was then performed to evaluate the extent to which the aortic valve was

Figure 1. *CTPA showing Pathologically Dilated Aorta Ascending to 5.8 cm*

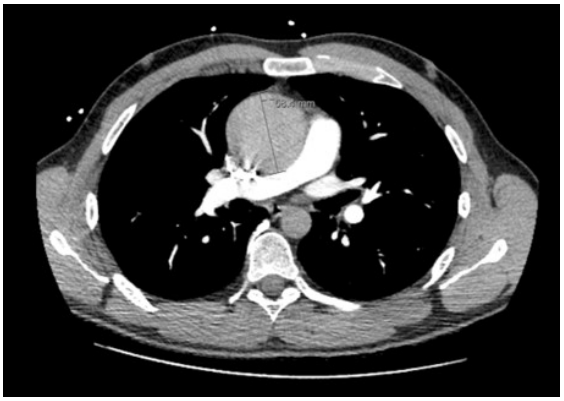
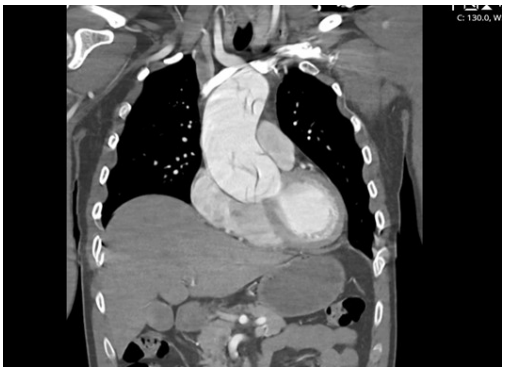
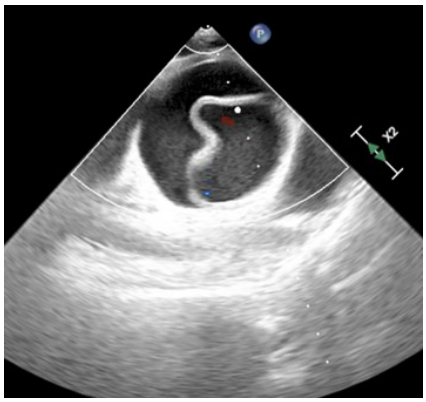


Figure 2. *CT Angiography of the Aortic Arch Showing Visualisation of Type A Aortic Dissection*



involved in the dissection. Fortunately, the aortic valve was not involved in the dissection, but it was noted that there was a bicuspid aortic valve.

Figure 3. *TTE with Visualisation of the Intimal Flap*



An acute type A aortic dissection was finally diagnosed. The patient was then transferred to the operating theatre for urgent aortic arch replacement.

Statistical Analysis

Considering we have data that doesn't conform to the traditional normal distribution, the statistical significance was confirmed using the Mann-Whitney U-test as appropriate. The resulting *p*-values less than 0.05 were considered statistically significant. All statistical analysis was performed using Microsoft Excel software.

Epidemiology

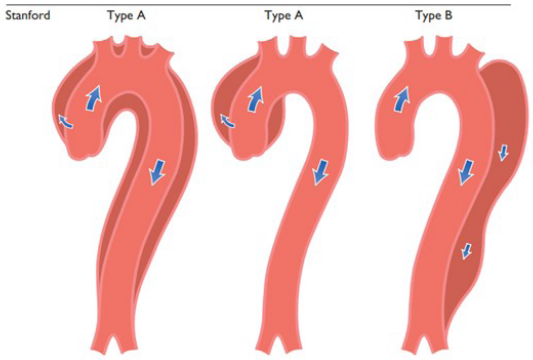
The overall incidence of aortic dissections in the general population is estimated to be about 4 per 100,000 persons per year.[7-9] This pathology has a bimodal distribution, yet most patients are between 60 and 80 years old. The main risk factors are chronic arterial hypertension and atherosclerosis.[8-10] Younger patients are mostly men with associated genetic connective tissue disorders such as Marfan's syndrome, Ehlor-Danos et al[11]. Other risk factors include past aortic instrumentation or surgery, coarctation aortae, bicuspid aortic valve, pregnancy, use of fluoroquinolones, and, finally, cocaine use.[12,13] Aortic dissections in women tend to occur in older age tend to be more complicated and have a higher mortality rate.[11]

Pathophysiology

An aortic dissection occurs after the intimal layer of the aorta ruptures. This leads to the formation of a false lumen where blood runs between the intimal and adventitial layers. As a result, both layers are torn apart (Figure 4).

The sites where dissections occur are at the level of the sinotubular junction at the beginning of the ascending Aorta and just distal to the left arteria subclavia.[10,14] The dissection may extend towards the aortic valve or further distally. The proximal extension can cause various complications such as cardiac tamponade, aortic valve insufficiency, or myocardial ischemia. If the dissection ruptures further distally, malperfusion syndromes of various organs such as ischemia of the mesentery or spinal cord may occur. Sometimes a second tear occurs in the intimal layer, allowing blood flow to be restored and consequently the symptoms seem to improve spontaneously. [10,14] Sometimes the dissection breaks through the adventitia, causing a complete rupture through the Aorta. If this happens, it is quasi always immediately fatal.[10,14,15]

Figure 4. *Anatomical Classifications of Aortic Dissections According to Stanford Classification*[31]



Large arrows show the direction of flow of the true lumen. Small arrows represent the false lumen

Medical History

Patients with aortic dissection may present with a wide variety of symptoms. Initial symptoms depend on where the intima is first damaged. As the dissection ruptures further, symptoms may change over time. The diagnosis of an aortic dissection is missed in up to 38% of cases.[10,16] This highlights the importance of low-level clinical suspicion and the need for a comprehensive history and clinical examination.

The classic textbook presentation of an Aortic dissection is often described as the triad of acutely arising severe rupturing thoracic pain with radiation to the interscapular.[17] There is an increasing awareness that patients do not always present with these classic symptoms. In a lot of cases, there is not even any pain and only vague symptoms are present.[18-21]

In this case, the initial presenting complaint was neck pain. A retrospective study that analyzed Type A aortic dissections noted that in 25% of cases with a proven Type A dissection, neck pain and/or headache were the initial presenting complaints.[22] Some guidelines regarding the management of neck pain have noted associated thoracic pain as a red flag,[4] but most guidelines do not explicitly mention thoracic pain or radiation as a red flag.[3,5]

Syncope can also be a clinical manifestation of aortic dissection. This is often a sign of an associated major complication such as cardiac tamponade, obstruction of cerebral vessels, or stimulation of cerebral baroreceptors. Syncope occurs in about 7% of all dissections.[8,10]

As the dissection expands, symptoms of renal failure and mesenteric ischemia may also occur.[23]

Clinical Signs

Difference in blood pressure between the two arms: Dissection may cause an intimal flap or local compression of a haematoma resulting in reduced blood flow to the peripheral blood vessels. This can cause there to be a significant variation (>20 mmHg) in systolic blood pressure between the two arms. There is an association between a systolic blood pressure difference and aortic dissection, but this remains a very unreliable sign. The diagnostic relevance is thus rather limited, but this remains important to document because if a perfusion deficit is observed, one should take into account the highest blood pressure during blood pressure treatment.[24] In the case of this case, the blood pressure difference was not significant.

Heart murmur: Classically, it is taught that a newly arising diastolic heart murmur is linked to an aortic dissection. If the tear extends proximally, the aortic valve may become involved in a dissection. This can cause acute aortic insufficiency, which in turn translates into a new diastolic heart murmur. The diagnostic added value of this murmur is limited here, but in the context of aortic dissection, this again remains important to document. Note that in addition to a diastolic murmur, a systolic murmur may also be present in the context of aortic dissection. This is due to coarctation aortae, a bicuspid aortic valve, or a newly developed septum defect.[25] In the patient of this case study, despite the presence of a bicuspid aortic valve, no heart murmur was observed.

Neurological abnormalities: Neurological dysfunction may occur if the dissection passes over a region of the Aorta responsible for irrigating the spinal cord or if the dissection is adjacent to the carotid. This can lead to a focal neurological deficit due to an ischemic cerebrovascular accident or spinal ischemia.

In aortic dissection, the structural dilatation of the aorta may exert pressure on adjacent structures such as the esophagus, the laryngeal recurrent nerve, or the sympathetic ganglion in the neck. This compression can lead to dysphagia, hoarseness, or Horner syndrome. 20% of patients with type A aortic dissection show neurological impairment.[26] The patient in this case study showed no neurological symptoms.

Technical Investigations

ECG: Several ECG abnormalities can be observed in aortic dissections. In 41% of cases, non-specific repolarization abnormalities are noted. In 31% of cases, there is a normal ECG. The remaining cases mainly have signs of cardiac ischemia. This is a sign of extensive dissection that also involves the coronaries.[10] This patient had no ECG abnormalities but eventually had a dissection of the left anterior descending coronary artery (LAD) during the perioperative period.

D-dimer: This is a degradation product of fibrin networks and reflects the activation of the extrinsic pathway of the coagulation cascade. This pathway is also activated by the tearing of the intima. D-dimers lower than 500 ng/mL therefore have a strong negative predictive value in dissections. D-dimers could consequently be a good screening tool in ruling out aortic dissection.[27]

Chest X-Ray: Sometimes a widened mediastinum or abnormal aortic silhouette is seen on the Chest X-ray. These findings occur in up to 63% of patients with type A dissection, BUT in 17% of cases, no abnormalities can be seen. Consequently, a normal RX-thorax is not a good excluder for the diagnosis of aortic dissection.[10]

CT-aortogram: The entire Aorta is visualized and therefore you can identify the true and false lumen. This examination is the preferred choice because it visualizes the Aorta in great detail, but on the other hand, it is also a very fast investigation.[28,29] In this case, a CT angiography of the pulmonary vessels was first performed. This was to diagnose pulmonary emboli. Pulmonary emboli and aortic dissection are often part of the same differential diagnosis. This frequently creates a diagnostic dilemma. CT pulmonary angiography can give hints of a possible dissection but often leads to many false negatives, making this not a good diagnostic tool.[29,30] In the case of this case, CT pulmonary angiography and pathologically widened ascending aorta could show what prompted further diagnostics.

Transesophageal echocardiography ultrasound: Transesophageal echocardiography (TEE) in the hands of an experienced operator, can be as specific and sensitive as a CT angiography of the Aorta. You can visualize the moving heart better than on a CT scan. This may also allow you to detect additional regional contractility disturbances, which is a sign of myocardial ischemia. A bicuspid aortic valve may also be noticed.

The disadvantage of TEE is that there is no complete overview of the extent of the dissection and the patient needs to be sedated to perform this examination.[28] In this case, an additional

TEE was performed because one was uncertain to what extent the aortic valve was involved in the dissection. A bicuspid aortic valve was seen, but it appeared to be spared from dissection.

Treatment

The principle of therapy is that you try to prevent aortic dissection from rupturing further. You try to do this by controlling heart rhythm and wall tension (blood pressure (BP)). This principle is called anti-impulse therapy. The targets are a heart rate below 60 beats per minute and a systolic blood pressure between 100 and 120 mmHg.[31,32]

Achieving these targets is done in three steps. In the first step, you provide adequate pain relief. This is usually with opiates and can sometimes be enough to control blood pressure and pulse. In the second step, you try to control the heart rate using beta-blockers such as Labetalol or Esmolol. In a third step, you may start vasodilator therapy using calcium antagonists such as Nicardipine. This last step is usually not started without the association of beta-blockade because you want to avoid secondary reflex tachycardia.[31]

The definitive treatment is cardiothoracic surgery, with surgical techniques varying depending on the type of dissection.[31]

DISCUSSION

In this case, pulmonary emboli were initially thought to be the cause of these atypical neck pain symptoms. Distinguishing between an aortic dissection and pulmonary emboli is an important diagnostic challenge for clinicians. Both conditions can present with overlapping clinical symptoms. The lack of specific guidelines for simultaneous evaluation of these two conditions further complicates the matter.

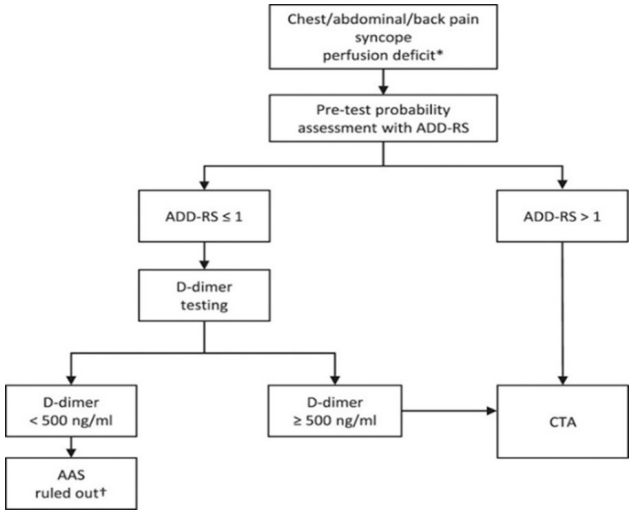
Addressing this diagnostic dilemma begins with a thorough history and clinical examination, focusing on detecting risk factors. For example, a history of deep vein thrombosis, immobilization, or recent surgery may skew the diagnosis of pulmonary emboli. Connective tissue disease or a family history of vascular disease may point more toward aortic dissection. In this case, there were no risk factors present for pulmonary emboli, but there was a positive family history of vascular pathology. This highlights the importance of family anamnesis.

The Aortic Dissection Detection Risk Score (ADD-RS) is a valuable tool to estimate the risk of aortic dissection and then determine your diagnostic steps. This score uses 12 clinical features carefully selected based on their association with aortic dissections. These features include specific predisposing conditions, pain characteristics, and typical findings on clinical examination (Table 2).[33]

Each Feature Present is Worth 1 Point

A proposed diagnostic algorithm was tested in the Aortic Dissection Detection-Risk Score Plus D-Dimer in Suspected Acute Aortic Dissection (ADvISED) trial. This pathway combines the an ADD-RS score with D-dimers. The use of this pathway could effectively rule out aortic dissection in patients at low to intermediate risk according to the ADD-RS and negative D-dimer (< 500 mg/dL) in a safe manner (Figure 5).[34]

Figure 5. *ADD-RS Pathway*[34]



Despite the potential of this diagnostic pathway, the ADvISED trial lacks important external validity. This study only took place in large centers so the results cannot be generalized to smaller hospitals and general practices.[35]

Given the absence of risk factors of pulmonary embolism

Table 2. *Aortic Dissection Detection Risk Score (ADD-RS)*[34]

High-risk conditions	**High-risk pain features:** thoracic, abdominal or back pain with	High-risk characteristics noticed during clinical examination
Marfan syndrome	acute onset	**perfusion deficit:** pulse deficit or difference in systolic blood pressure or focal neurological deficit
family history of aortic disease	intense intensity	diastolic murmur
known aortic valve disease	tearing or pulling sensation	hypotension or shock
recent aortic surgery		
known thoracic aortic aneurysm		

and the presence of risk factors of aortic dissection, in this case, the decision to do a CT aortogram immediately could in principle be made based on history and based on the ADD-RS tool.

CONCLUSION

This article highlights the diagnostic complexity of an aortic dissection, especially when accompanied by atypical symptoms such as neck pain in this young patient. Neck pain, although not often associated with severe pathology, can be an important presentation in Type A aortic dissections. This highlights the importance of having a broad differential diagnosis. The importance of a comprehensive family history is emphasized in this case, where predisposition to certain vascular diseases can be revealed and guide the diagnostic process. Despite the often absent classic clinical signs, documenting them remains crucial not only for diagnosis but also for guiding the treatment process and estimating the extensiveness of dissection. The overlapping symptoms of pulmonary emboli and aortic dissection present a diagnostic challenge, but the difference in risk factors for the two conditions can help in making informed decisions about which imaging modality should be used. Although tools such as the ADD-RS combined with D-dimers could be a promising tool, their current lack of external validation necessitates a cautious approach to their application in clinical practice.

FINANCIAL SUPPORT

No financial support was received for this manuscript.

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CONSENT

The authors have received written informed consent from the patient.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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