AN UNEXPECTED CAUSE OF CHEST PAIN WHILE SELF-PLEASURING: A RIPPING DOOM EXCITEMENT

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INTRODUCTION

- **Acute aortic dissection (AD) involves tearing of the aortic intima by shearing forces resulting in a false lumen, that depending on its location and extent may lead to hemodynamic compromise, malperfusion to vital organs or even rupture to the aorta.**
- **Classical presentation is of sudden chest or back pain described as sharp or ripping in quality.**
- **Up to 20% of the patients die before arriving to the hospital, and every hour without surgical treatment mortality increases by 1-3%, therefore, early recognition and treatment is of crucial importance.**

CASE PRESENTATION

- This is a 60-year-old male with history of hypertension, Liddle's syndrome, obstructive sleep apnea, and chronic cannabis use for insomnia. He arrived to another institution with complains of severe retrosternal chest pain of several hours in evolution that started during masturbation. The pain was ripping in character, starting retrosternal, and radiated to both sides of his neck and back. Initially managed with aspirin, ticagrelor and enoxaparin for a presumed diagnosis of a non-ST-segment elevation myocardial infarction (NSTEMI) and subsequently transferred to our institution.

- Upon arrival to our emergency department (ED) he had no chest pain and he was hemodynamically stable with a blood pressure of 174/68 mmHg and a heart rate of 59 beats per-minute. Physical exam was consistent with warm extremities, no focal neurological deficits, symmetrically strong peripheral arterial pulses, mild systolic ejection murmur at the LLSB, no crackles and no jugular venous distension yet bilateral carotid bruits were present.

- Serial high-sensitive troponins were abnormal and with increasing delta trend: 49, 56, 69, 136 ng/l at 0/1/3/6 hours respectively (abnormal cut-off >22 ng/l). Arrival ECG had sinus rhythm without ST-segment changes in favor of acute ischemia (figure 1). In view of chest pain associated with positive cardiac biomarkers, he was admitted to the Coronary Care Unit on intravenous nitrates.

- Waiting for his admission he suddenly had recurrent chest pain associated to marked hypotension (75/43 mmHg), for which intravenous nitrates were discontinued, fluid resuscitation was provided and started temporarily on intravenous norepinephrine. The on-call cardiology team was called for emergent evaluation. Subsequent electrocardiogram had subtle (1mm) ST-depression in leads V5-V6 and no electrical alternans (figure 1, red arrows). A bedside echocardiogram disclosed a hemodynamically significant moderate pericardial effusion (figure 2) with partial right ventricular diastolic collapse (figure 7) and an echogenicity overlying the pericardium (figure 2) highly concerning for pericardial clot. Additionally, a moderate aortic insufficiency (figure 6) was present, and an intimal flap was visualized on the ascending and descending aorta suggestive of aortic dissection (AD) (figure 2-4).

- A computer tomographic angiogram revealed a large pericardial effusion with an extensive AD extending from the level of the ascending aorta, including both main carotid arteries and inferiorly to the right iliac artery (figure 8-10).

- These findings were consistent with an extensive Stanford type-A AD with rupture into pericardium for which he was emergently consulted to the cardiothoracic surgeon. This surgery was performed emergently and included pericardial fluid drainage, followed by successful replacement of the ascending aorta and aortic valve repair with successful results.

ELECTROCARDIOGRAPHY

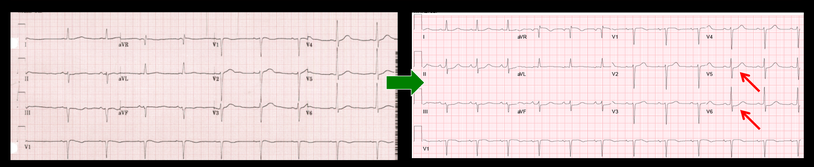

Figure 1: Initial ECG upon arrival with normal sinus rhythm and no ST-segment changes in favor of acute ischemia or electrical alternans. After developing hypotension, his chest pain returned and subsequent ECG done showing mild (~1mm) ST-depressions in leads V5-V6 consistent with myocardial ischemia possibly due to a supply/demand perfusion imbalance.

TRANSTHORACIC ECHOCARDIOGRAPHY

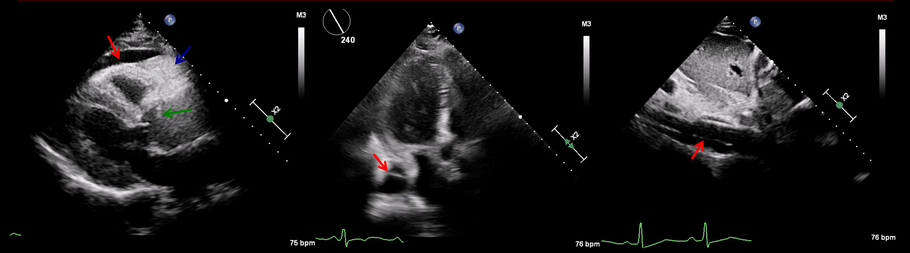

Figure 2: PLAX showing dilated ascending aorta with intimal flap (green). There is a moderate pericardial effusion (red) with associated pericardial coagulum (white).

Figure 3: Apical 3-chamber view revealing descending thoracic aorta with intimal flap.

Figure 4: Subcostal view of the abdominal aorta showing the intimal flap.

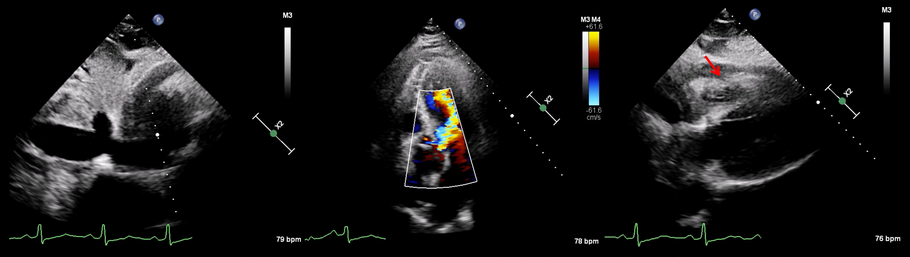

Figure 5: Subcostal view showing plethoric inferior vena cava without inspiratory collapse.

Figure 6: Apical 5-chamber view with color where one can appreciate a moderate-to-severe aortic valve insufficiency jet extending into the left ventricular apex. It is most likely secondary to dissection flap prolapsing into the valve and causing poor coaptation.

Figure 7: Subcostal view disclosing right ventricular diastolic collapse.

CT ANGIOGRAPHY

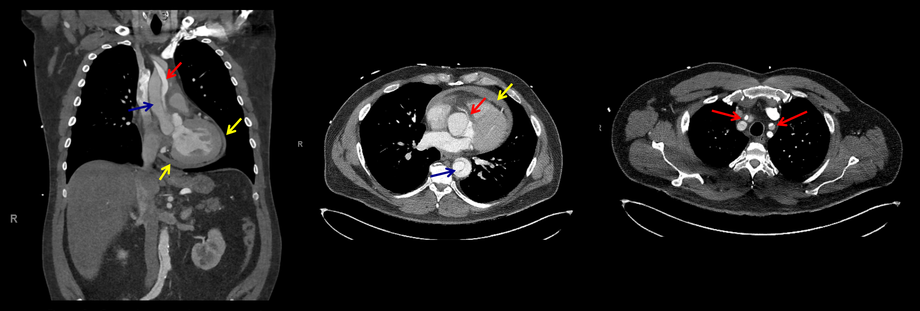

Figure 8: Coronal section showing extensive ascending thoracic aorta dissection (Stanford type-A AD) and a moderate pericardial effusion. The more darker lumen represents the false lumen (blue) whereas the brighter lumen represents the true lumen (red). Dissection extended to the right Iliac artery. Pericardial effusion is evident (yellow arrows).

Figure 9: Axial section showing ascending thoracic aorta dissection sparing the right coronary ostium (red) and extending into the descending thoracic aorta (blue). A moderate pericardial effusion is also evident anteriorly (yellow arrow).

Figure 10: Axial section dissection extending into the right and left common carotid arteries.

DISCUSSION (1/2)

Acute aortic syndromes (AAS) such as aortic dissection, intramural hematoma, penetrating ulcer or rupture are life-threatening cardiovascular emergencies affecting 3-6/100,000 individuals per year [1]. Clinical presentation can be quite unspecific therefore misdiagnosis rates are high reaching up to 39% [1] which in turn can have a detrimental impact over patient's outcomes if surgical treatment is delayed. Ascending aortic dissection is deadly by itself, however, when complications such as aortic regurgitation and cardiac tamponade arise, just as happened to our patient, mortality increase even further. Immediate surgical intervention for aortic root replacement plus aortic valve repair or replacement is recommended and can be lifesaving in cases were the ascending aorta is involved. Aggressive blood pressure (SBP <120mmHg) and heart rate control (HR=55-60) should be provided prior to surgery to avoid mitigating progression of the dissection.

DISCUSSION (2/2)

Aortic dissection in this patient could have been triggered by a myriad of factors. Firstly, he had Liddle's syndrome which imposes the aorta to chronic hypertension, one of the classical risk factors for dissection. *Abbass et al.* described a patient with Liddle's syndrome presenting with an AAS of the thoracic descending aorta [8]. Secondly, sexual activity has been linked to aortic dissection possibly due to a rise in catecholamine levels causing a significant elevation of blood pressure and thus sheer stress to the aorta [5]. In one retrospective study by *Gansera et al.* showed that the physical and emotional stress associated to sexual activity were a meaningful promoters of aortic dissection in 11% (p=0.03%) of younger males (<60 y/o) presenting with this diagnosis [6]. Finally, development of dissection with marijuana use has also been described although its pathophysiological mechanism leading to dissection remain elusive [7].

For accurate diagnosis, guidelines recommend establishing the preterm probability of AAS based on the aortic dissection detection risk score (ADD-RS), which consists of 12 risk markers classified into 3 categories: predisposing conditions, pain features, and physical exam findings [1]. In one multicenter and prospective observational study done by *Nazerian et al.* showed that patients with suspected AAS with a low or intermediate risk (ADD-RS=0-1) plus a d-dimer less than 500ng/mL, the sensitivity and negative predictive value were found to be very high in 98.8% and 99.7% respectively, thus helpful for effectively ruling out such diagnosis. Alternatively, in cases deemed as high risk (ADD-RS2-3), one should proceed directly to confirmatory testing with CT angiography or transesophageal echocardiogram irrespective of d-dimer levels. In the case of our patient, the chest pain with irradiation to his back as well as his state of hypotension conferred a high risk for AAS (ADD-RS=2) thus accordingly CT angiogram evaluation was performed to establish the diagnosis.

AAS can have the same presentation as acute coronary syndromes (ACS). Up to 40% of patients with AAS can have non-specific ST-segment changes raising the possibility of ischemia and ACS/NSTEMI [2]. In patients with AAS misdiagnosed as ACS, the employment of anticoagulation and antiplatelet agents can theoretically have lethal consequences [3] by worsening and fueling the dissection process. Therefore making the distinction early in the natural history of the disease is of the uttermost importance as in the case of our patient.

Echocardiography and point-of-care ultrasound (POCUS) is crucial in the evaluation of these patient. It is a powerful tool that certainly will aid the clinician make the distinction between AAS and ACS. In a retrospective study by *Pare et al.* the use of POCUS was associated with a faster diagnosis of AAS (median of 80 vs. 226 minutes, p=0.023), lower rate of misdiagnosis (0% vs. 43.8%, p=0.028) and a lower mortality (15.4% vs. 37.5%, p=0.27) [4].

Our case emphasizes the importance of early employment of echocardiography for making the distinction between AAS and ACS. An early distinction can guide the clinician as to determine whether providing antithrombotic agents is safe and will not hasten a possible AAS.

CONCLUSION

Acute AD is potentially a lethal medical emergency and may mimic or present with a concomitant acute myocardial infarction (AMI). A high level of clinical suspicion should lead to appropriate diagnostic imaging modality evaluation to r/o AD prior to the initiation of anticoagulation therapy for AMI, since this may worsen the dissection and thus translate into higher mortality.

REFERENCES

1. Nazerian P, Mueller C, Soeiro AM, et al. Diagnostic Accuracy of the Aortic Dissection Detection Risk Score Plus D-Dimer for Acute Aortic Syndromes: The ADvISED Prospective Multicenter Study. *Circulation*. 2018;137(3):250-258. doi:10.1161/CIRCULATIONAHA.117.029457
2. Tolefac PN, Dzudie A, Mouliom S, et al. Acute type A aortic dissection involving the iliac and left renal arteries, misdiagnosed as myocardial infarction. *Cardiovasc J Afr*. 2018;29(1):e9-e13. doi:10.5830/CVJA-2017-042
3. Zschaler S, Schmidt G, Kukucka M, Syrmas G, Zaschke L, Kurz SD. How to prevent inadvertent emergency anticoagulation in acute type A aortic dissection: when in doubt, don't. *Cardiovasc Diagn Ther*. 2018;8(6):805-810. doi:10.21037/cdt.2018.10.13
4. Pare, J. R., Liu, R., Moore, C. L., Sherban, T., Kelleher, M. S., Jr, Thomas, S., & Taylor, R. A. (2016). Emergency physician focused cardiac ultrasound improves diagnosis of ascending aortic dissection. The American journal of emergency medicine, 34(3), 486–492. https://doi.org/10.1016/j.ajem.2015.12.005
5. Ridha A, Safiullah S, Al-Abayechi S, Ghadai A and Nadeem AUR. A Rare Presentation of a Life- Threatening Condition Secondary to Masturbatory Activity: A Case Report. J Cardiovasc Disord. 2015;2(2): 1012. ISSN: 2379-7991
6. Gansera, L., Deutsch, O., Szameitat, L., Eichinger, W., & Gansera, B. (2016). Aortic Dissections Type A during Sexual Intercourse in Male Patients: Accident or Systematic Coincidence? Examination of 365 Patients with Acute Aortic Dissection within 20 Years. The Thoracic and cardiovascular surgeon, 64(2), 133–136. https://doi.org/10.1055/s-0035-1549111
7. Mason, E. K., Gak, A. E., Finno, J. G., Cannon, R. D., & Jacoby, J. L. (2019). Thoracic Aortic Dissection Associated with Marijuana Use. The Journal of emergency medicine, 57(2), 235–237. https://doi.org/10.1016/j.jemermed.2019.03.034
8. Abbass, A., D'Souza, J., Khalid, S., Asad-Ur-Rahman, F., Limback, J., Burt, J., & Shah, R. (2017). Liddle Syndrome in Association with Aortic Dissection. Cureus, 9(5), e1225. https://doi.org/10.7759/cureus.1225

AUTHOR DISCLOSURES: NONE