Aortic Dissection

Section 1: Case Summary

Scenario Title:	Aortic dissection
Keywords:	Aortic Dissection, cardiovascular, chest pain
Brief Description of Case:	A 25 yr old NFA with recurrent visits for polysubstance use and OD's presents with decreased LOC. His girlfriend says he did upper and then downer. He has received bystander CPR Narcan 0.8mg IM from EHS. He arrives agitated, screaming, and complaining of chest pain

Goals and Objectives	
Educational Goal:	
Objectives: (Medical and CRM)	-recognize the different chest pain mimics in cocaine abusers -recognize the therapeutic dilemmas caused by polysubstance overdose -demonstrate the principles of management of suspected aortic dissection -communicate effectively with team and summarize patient presentation and changes to team
EPAs Assessed:	

Learners, Setting and Personnel				
Target Learners:	☐ Junior Learners	☒ Senior Learners	☐ Staff	
	☐ Physicians	☐ Nurses	☐ RTs	☐ Inter-professional
	☐ Other Learners:			
Location:	☐ Sim Lab	☐ In Situ	☐ Other:	
Recommended Number of Facilitators:	Instructors: 2			
	Confederates:			
	Sim Techs:			

Scenario Development	
Date of Development:	March 14, 2020
Scenario Developer(s):	Kaleena Patel
Affiliations/Institutions(s):	
Contact E-mail:	Kpatel5@shaw.ca
Last Revision Date:	
Revised By:	
Version Number:	1.0

Section 5: Scenario Progression

Scenario States, Modifiers and Triggers				
Patient State/Vitals	Patient Status	Learner Actions, Modifiers & Triggers to Move to Next State		Facilitator Notes
1. Baseline State Rhythm: Sinus tachy HR: 133 BP: 210/100 left arm (BP 240/100 right arm if asks for it) RR: 14 O_2SAT: 91 % T: 37.1^{o}C GCS: 15	*Screaming, agitated, complaining of chest pain, says he can't breath*	<u>Expected Learner Actions</u> ☐ IV, O2 via FM, recheck glucose, order ECG and CXR, tox screen, cardiac labs ☐complete secondary survey, note slight weakness on left limbs ☐ consideration of neuroimaging - activate hot stroke protocol ☐ treat pain (fentanyl boluses preferred but any opioid ok for now) and agitation (lorazepam) ☐interpret ECG	<u>Modifiers</u> -if pain not treated in first 3 minutes, RN to ask if she can give him anything for pain <u>Triggers</u> -5 minutes passes and CT head ordered >> stage 2	POCUS is normal at this stage
2. Improvement HR: 120 BP 195/90 RR 14 O2 91% RA	Patient calmer now that pain is controlled but concerned about his left arm weakness (left leg back to normal)	<u>Expected Learner Actions</u> ☐ interpret CXR and ECG > recognize possibility of aortic dissection (widened mediastinum, pleural effusion)>> order CT-aorta ☐insert 2 large bore IVs, consider insertion of arterial line if it won't delay care ☐ control BP and HR prior to sending to CT >> specify goals of therapy for titration. (goal HR 60, goal SBP 100-110). First give beta-blocker (labetalol or	<u>Modifiers</u> -learner to receive CXR and ECG at beginning of step 2 -After CT-aorta ordered, radiologist calls to ask which CT the ERP wants first if not specified <u>Triggers</u> - pt sent to CT without meds given>> cardiac arrest in CT>> end scenario	Learner does NOT need to know exact doses for medications but should know the general classes of meds to give and how to look up dosing (eg ask ED pharmacist or RN to get PDTM)

		esmolol), then give afterload reducer (nicardipine or nitroprusside).	-pt sent to CT AFTER meds given >> stage 3	
3. Deterioration GCS 5 HR 90 BP 155/90 RR 6 O2 88% R/A	Patient returns from CT very drowsy, not moving left side at all, pupils pinpoint	<u>Expected Learner Actions</u> ☐Recognize change in mental status, and discuss possible reasons, repeat neuro exam >> pupils are pinpoint, not moving left side ☐ decision to intubate for airway protection, setup and choose meds (lidocaine and fentanyl, consider anesthetizing the subglottic structures to prevent sympathetic surge) ☐ post-intubation CXR, insert foley to assess renal function ☐ consult vascular surgery +/- stroke neuro	<u>Modifiers</u> --If gives more narcan – patient becomes much more agitated, not moving left body at all, BP increases to 200/110, HR 155, constantly moaning through clenched jaw but no change in GCS -if asks for BiPAP, RT to say patient too drowsy and doesn't meet criteria for safety -receive CT results after intubation <u>Triggers</u> - intubation done >> give CT results (acute aortic dissection involving the arch extending down to the level of the bifurcation with associated dissection and thrombus extending into both vertebral arteries)>> stage 4	

			-if learner gives tPa for suspected ischemic stroke, patient arrests and end of scenario	
4. Stabilization and transfer GCS – vented and paralyzed HR 60 BP 115/60 RR – vent O2 98% on 30%		<u>Expected Learner Actions</u> ☐ emergent vascular surgery consult – ask for BP guidelines repeat ECG, ☐ post-intubation care – adequate sedation and analgesia, recognize BP differential in arms and use limb with higher BP to guide therapy	<u>Modifiers</u> -RN to cycle BP at beginning of stage 4 and notes BP not obtainable on left arm <u>Triggers</u> -3 minutes >> end scenario	

Appendix C: Facilitator Cheat Sheet & Debriefing Tips

Include key errors to watch for and common challenges with the case. List issues expected to be part of the debriefing discussion. Supplemental information regarding any relevant pathophysiology, guidelines, or management information that may be reviewed during debriefing should be provided for facilitators to have as a reference.

Key errors:
-don't ignore chest pain in the setting of neuro deficits, and beware thrombolysis without further assessment! (same goes for MI)
-manage HR and BP early to avoid an arrest situation…this includes pain control, avoid too much narcan
-fluctuating symptoms (esp neuro) are common in this diagnosis as the dissection knocks off collateral flow or a pulsating thrombus intermittently occludes flow
-hypotension can be multifactorial – don't just assume it is rupture or your medications. You may still be able to stabilize the patient for transfer

Supplemental:
- IRAD study (International Registry of acute aortic dissection) in 2000 greatly improved our ability to diagnose (ie. Have a high clinical suspicion) aortic dissection.
- Ascending aortic dissections are almost twice as common as descending dissections

Stanford classification system:
- Type A = anything involving ascending aorta
- Type B = anything NOT involving ascending aorta
- In patients with an ascending aortic dissection, aortic arch involvement is seen in up to 30 percent-
- The indications for surgery in Type B dissection are: Malperfusion, ongoing progression, inability to control BP, and perforation.

Risk factors for Dissection
- Hypertension, age, male gender, family history, recent deceleration injury (i.e. MVC with airbag), prior cardiac surgery, known preexisting aortic aneurysm, recent cardiac cath.
- In younger patients (<40): cocaine or amphetamine use, pregnancy, connective tissue diseases (e.g. Marfan Syndrome), congenital heart disease, bicuspid aortic valve (9x risk), and weight lifting.

The Classic presentation
- Acute, "tearing" or "ripping" chest pain reaching maximal intensity at onset, radiating to back and/or between shoulder blades.
- Only 25% of patients have the triad of this pain, widened mediastinum, and pulse deficit.
- The most common descriptor of the pain is "sharp", ~5% presented painlessly (1), and 10% with syncope (IRAD(2).

3 Important Questions (think: SAH of the chest/abdomen):
- Quality of pain (most commonly "sharp" but highest LR for "tearing")
- Pain intensity at onset

- Radiation of pain (back and/or belly) 90% of the ED docs suspected dissection even before investigations were done if all 3 of these questions were asked.
- "Chest pain Plus…" CP + focal neuro deficit or pain below diaphragm or limb ischemia: think dissection!

The Concept of CP +1 and 1+ CP in Aortic Dissection:

- The intimal tear in the aorta can devascularize any organ from head to toe including the brain, heart and kidneys, and so 5% of dissections present as strokes!! An objective focal neurologic deficit in the setting of acute unexplained chest pain has ***+LR = 33 for aortic dissection***.
- CP + CVA
- CP + paralysis
- CP + hoarseness (recurrent laryngeal nerve)
- CP + limb ischemia

In addition to thinking of CP +1 it may help to think *backwards* in time and ask the patient who presents with end organ damage if they had torso pain prior to their end organ damage symptoms. For example, for patients who present with stroke symptoms, ask them if they had chest pain, back pain or abdominal pain *before* the stroke symptoms.

Painless Aortic Dissection

While IRAD reported a painless aortic dissection rate of about 5%, a more recent study out of Japan reported that 17% of aortic dissection patients had no pain. These patients presented more frequently with a persistent disturbance of consciousness, syncope or a focal neurologic deficit. Cardiac tamponade was more frequent in the painless group as well.

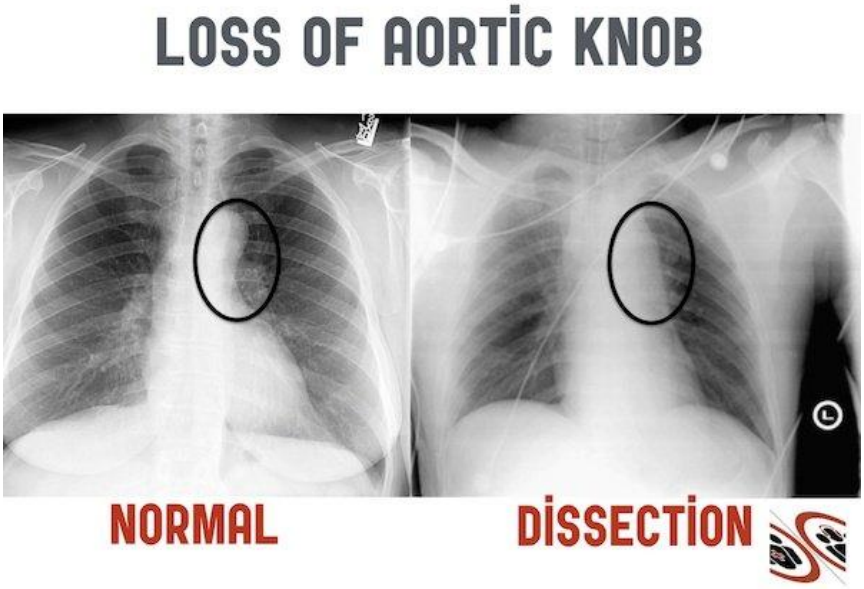
Hypotension

-is due to: MI, acute aortic rupture or acute aortic insufficiency, hemopericardium causing tamponade, hypotension in a limb

Diagnostic testing:

- Although D-dimer will usually be positive in nearly all patients with dissection, it cannot be used as a rule-out test as low levels have been found in younger patients, or patients with a thrombosed "false lumen". Both D-dimer and troponin could be normal in patients presenting early with dissection.
- CXR: The most common abnormality seen in aortic dissection is widening of the aortic silhouette, appearing in 60 to 90 percent of cases. **Calcium Sign:** separation of the outermost portion of the aorta from the calcified intima by >5mm
- other possible sings:loss of aortic knob, trachea displaced to right, left mainstream bronchus displaced downward, disparity of ascending and descending aorta caliber, apical capping, pleural effusion (usually left), localized bulge in the aorta.
- **<u>Remember to compare with previous CXR if available!</u>**

Aortic Dissection



https://www.grepmed.com/images/2377/aorticknob-dissection-diagnosis-radiology-chestxray-clinical-apwindow

- ECG – nonspecific changes and all types of ischemic patterns are possible…classic teaching is inferior STEMI due to back-dissection into RCA. This can infrequently lead to complete heart block.
- Ultimately, diagnostic test of choice is CT-aorta or TEE
- POCUS for rockstars– look for hemopericardium (can attempt drainage esp in cases of hypotension), can look for aneurysm or dissection flap in the abdominal aorta, measure aortic root dilatation in PSLA view.

Physical exam:
Look. The patient doesn't always know they have Marfan's so you need to ***look*** for the signs:

1. **Arachnodactyly** (Elongated fingers): if they look long try to elicit the ***wrist sign*** (see this 15 second video **here**)
2. **Pectus excavatum:** sternal excavation (see image)
3. **Lanky limbs**

Listen. A new aortic regurgitation murmur (=retrograde dissection) has a surprisingly high +LR = 5. Acute aortic valve regurgitation occurs in ½ - 2/3 of ascending dissections
Feel. Feel for a pulse deficit which has a +LR = 2.7. (need 2 people to do this!)
-women are less likely to have a pulse deficit than men. Compared with younger patients, older adult patients (>70 years) were significantly less likely to have any pulse deficit

-Blood pressure: classic teaching is SBP difference of >20mmHg in upper limbs. Many patients have a difference in blood pressure between arms normally, so a BP difference does not rule in dissection; nor does a lack thereof rule out dissection. However, a BP difference may heighten your suspicion of dissection in the right clinical context
-Patients with dissection who have a ***wide pulse pressure*** should be considered pre-terminal, and usually require immediate surgery.

Management:
-Each hour that passes from the onset of symptoms portends a 1-2% increase in mortality
-transient neuro deficits (be careful about giving thrombolytics)
-goal is to decrease the Dp/Dt (delta-pressure/delta-time) = decrease heart rate, then decrease afterload
- put BP cuff on arm with the better pulse…same with arterial line
-use fentanyl boluses (25-50mcg at a time) to achieve analgesia
-goal HR 60-70 (or maybe lower!)
-goal SBP 100-110 (maybe higher if long-standing hypertension and worried about stroke)
-first use esmolol (250 to 500 mcg/kg IV loading dose, then infuse at 25 to 50 mcg/kg/minute; titrate to maximum dose of 300 mcg/kg/minute), then nicardipine (pure afterload reduction…2.5 to 5 mg/hour titrated to a maximum of 15 mg/hr)
-dissection can extend back into coronary vasculature to cause MI
-intubation – if you absolutely need to do this, Tx same as for a high-ICP intubation: pretreat with high-dose fentanyl and lidocaine, anesthetize the subglottic structures and cords to prevent sympathetic surge, use VL to get it on the first try and apply minimal force on the glottis

Incidentally:
-the vast majority of cocaine-induced CP is NOT due to MI
-cocaine is a sodium-channel blocker that can cause Brugada phenotype on ECG. This is transient and usually doesn't require treatment.

References

1. Scott Weingart. Podcast 91 – Treatment of Aortic Dissection. *EMCrit Blog*. Published on January 23, 2013. Accessed on April 17th 2020. Available at [https://emcrit.org/emcrit/aortic-dissection/].
2. Helman, A, Carr, D. Aortic Dissection Live from The EM Cases Course. Emergency Medicine Cases. February, 2017. https://emergencymedicinecases.com/aortic-dissection-em-cases-course/. Accessed [April 1, 2020].
3. Chopra, A, Carr, D, Helman, A. Aortic Dissection, Acute Limb Ischemia and Compartment Syndrome. Emergency Medicine Cases. November, 2012. https://emergencymedicinecases.com/episode-28-aortic-dissection-acute-limb-ischemia-compartment-syndrome/. Accessed [April 1, 2020].
4. Colla JSC et al. *Emergency Ultrasound: Identification of Aortic Dissection Using Limited Bedside Ultrasound.* Emergency Medicine*. 2017 March;49(3):135-137*

