Intramyocardial hematoma

Etiology:

- IDH is hemorrhagic dissection through the myocardium. Blood infiltration into the myocardial wall.
- Occurs as a complication of MI or during remodeling process.
- IDH is reported to have very low incidence and high mortality.
- Other causes: Severe thoracic injury, with application of stabilizer device for off-pump coronary revascularization.
- Can develop in LV free wall, RV, IVS.

Risk factors:

- Anterior wall infarct, large transmural infarction, Age >=60, HTN female sex, SVD, absence of previous cardiac events.

Pathogenesis:

- Rupture of intramyocardial vessels (esp. spiral vessels) into the interstitial space, decreased tensile strength of the infarcted area and acute increase of coronary capillary perfusion pressure. Hematoma stays entrapped within myocardium with an intact epi and endocardium.
- 90% cases rupture occurs after the first MI and has strong correlation with single vessel disease, reflecting lack of collateral circulation.
- It is most likely to appear within a few hours to a week after onset but reports of IHD are limited to case reports and case series.
- Post-infarction cardiac rupture usually develops within the first week, and very often within the first 24 h. This serious complication is more common in transmural and extensive infarcts. Most ruptures occurs during anterior wall MI, frequently near the IVS.
- According to the research conducted by Spinelli et al., 7 IDH is associated with coronary ‘no reflow’ (TIMI flow ≤ 2), as well as longer ‘pain-to-balloon’ time.

Diagnosis:

- Persistent S-T elevation more than 72 hours after the initial episode is an important clue in suggesting an intramyo-cardial hematoma.
- Echocardiographic diagnosis of septal and/or free wall IDH is based on presence of at least 3 of the following signs: 1) formation of one or more neo cavitations within the tissue with an echo-lucent center, 2) a thinned and mobile endomyocardial border surrounding the cavitary defect, 3) ventricular myocardium identified in the regions outside of the cystic areas, 4) changes in the echogenicity of the neo cavitations suggesting blood content, 5) partial or complete absorption os the cystic structure, 6) continuity between the dissection hematoma and one of the ventricular cavities, 7) communication between the 2 ventricular chambers through the myocardial dissection, 8) doppler recording of flow within the dissected myocardium.
- CMR can also reliably diagnose IDH. Contrast resolution allows for a clear distinction between the intramyocardial hematoma and myocardial tissue.

Differentials:

- Laminated thrombus: IDH there is a free space between the epicardial and endocardial layers with hypoechogenicity of blood during the initial stage. Pseudoaneurysm (complete rupture of myocardial wall contained by pericardium vs IDH where epicardium is intact), intracavitary thrombosis (distinguished from IDH by presence of endocardial layer surrounding the neoformation in IDH), prominent ventricular trabeculations (completely irregular shape of the ventricular wall with flow within it is hallmark of trabeculations).

Poor prognostic factors:

- Low LVEF, late presentation, cardiogenic shock, pericardial effusion and age >60 yrs.
- Overall mortality has been reported to be 47% with higher mortality reaching up to 78% in cases involving IVS.
- In a case series by Leitman et al (7) published in 2018, 42 cases of HDI have been diagnosed and published to date, in-hospital mortality was 23% with late presentation (>24 h after symptom onset) was also associated with increased mortality.

Treatment

- No guidelines for management.
- IDH limited to apex has a high probability of spontaneous resorption and an initial conservative approach is reasonable.

- Surgical management can be considered in the presence of hemodynamic instability or expanding hematomas with pericardial effusion.
- Multidisciplinary team approach involving cardiologist, cardiothoracic surgeons should be opted.

References:

1. http://dx.doi.org/10.1016/j.case.2017.05.004
2. https://doi.org/10.1016/j.jaccas.2022.07.025
3. https://doi.org/10.1016/j.jaccas.2020.07.038
4. https://doi.org/10.1186/s12872-022-02523-x
5. https://doi.org/10.1259/bjrcr.20200028