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Right Ventricular Infarction

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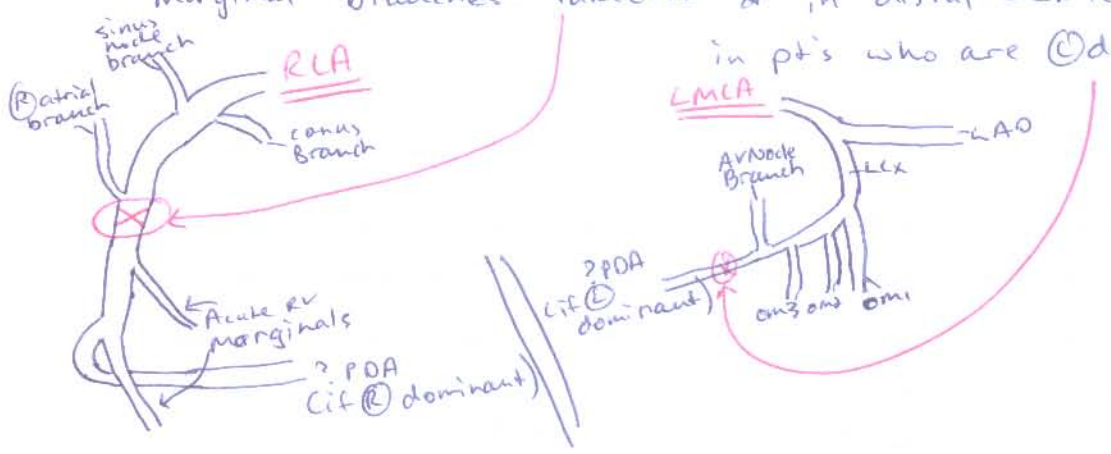
- occurs in ~½ of IW MI's

- Physiology:
  - normal RV → - same CO vs. LV
    - 1/6 muscle mass vs. LV
    - performs 1/4 stroke work → pulm. vascular resistance ~1/10 vs. systemic VR
  - coronary flow unique in that occurs in both systole + diastole (only diastole for LV) → if ϕ RVH
  - RCA main supplier
    - supplies lateral wall on Ⓡ → acute marginals
    - supplies posterior wall / posterior IV septum → PDA (if Ⓡ-dominant)
    - conus branch → ant. RV wall
    - LAD → also supports ant. RV wall

- Pathogenesis:
  - usually occurs w/ proximal RCA lesions → proximal to the marginal branches takeoffs or in distal LCx lesions in pt's who are Ⓒ dominant



  - may occur in absence of CAD → significant RVH → ↑'s ischemic demand on heart

- some RCA occlusions proximal to RV branches do not lead to RV infarcts?
  - Theories: ① RV has ↓'d O_2 demand vs. supply compared to the LV
    ② Biphasic RCA flow → both diastole + systole
    ③ ↑'d collaterals to RV vs. LV
    ④ ? Thebesian veins?
    - direct perfusion thru the RV cavity

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- Pathophysiology:
  - RV infarct → acute RV dilation + ↑'d intrapericardial pressure due to pericardial constraint
    - also ↓ RVSP's, ↓ LV EDV size, ↓ CO, ↓ aortic pressures
    - also equalization of (R) + (L) ventricular pressures (diastolic)
    - all above improve w/ pericardial incision
  - RV is very sensitive to Δ's in preload + afterload once ischemic
  - concomitant LV infarction (esp. IV septum) → lost compensatory mechanism for ↓ RV SF → LV septal contraction bulges into RV creating an RV systolic pressure wave → bulges into RV → generates systolic force for pulm. perfusion
  - RV becomes non-compliant → factors impairing RV filling → profound hemodynamic sequelae
    - Ex.) Volume Depletion
      Diuresis
      Nitrates
      ↓ atrial function / dyssynchrony (Ex.) AFib)

- Diagnosis:
  - always suspect in IW MI's → get a (R)-sided EKG too
  - Exam Findings:
    - Clinical Triad → (A) Hypotension, (B) Clear Lungs, (C) ↑ JVP, (D) IW MI — these 3 w/
      (25% sensitive, 95% specific)
    - JVD alone → 88% sensitive / 69% specific
    - Kussmaul's sign:
      - JVD w/ inspiration → usually indicates constrictive pericarditis but also sensitive / specific for RV infarct
    - ? RV Gallops
    - ? TR
    - ? AV Dissociation

- Hemodynamic Measurements:
  - RAP > 10 and w/in 1-5 of the PCWP → 73% sensitive / 100% specific for RV infarction
  - severe non-compliance in RA waveform (y-descent > x-descent) → indicates RV infarct
    - (A) x-descent: Represents passive return atrial filling as blood settles back into the atria from the venous system after it was pushed back following the initial backflow w/ mitral/tricusp valve closure
    - (B) Y-descent: Represents the rapid inflow stage of diastole → blood flows rapidly from atria → ventricles w/ mitral/triaspid valve opening

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- RV pressure tracings:
  - broad → ↓ upstroke w/ delayed relaxation
  - bifid → septal contraction
  - dip/plateau in diastolic curve → ↓ compliance plus pericardial constraining

- EKG:
  - get ®-sided EKG's in inf. wall MI's
  - V_4R → most sensitive/specific lead for RV infarction (ST Elevations)
  - Assoc. conduction Abnorms:
    - (A) RBBB
    - (B) CHB

- Echocardiography:
  - RV dilation
  - ↑'d RVED Dimension : LVED Dimensions ratio
  - RV wall asynergy
  - Abnormal IV septal motion → reversal of transseptal pressure gradient due to ↑ RVEDP (paradoxical)
  - Short-axis view (ventricles in cross-section)
    - highest sensitivity/specificity views
  - Prognostic Markers:
    - ① ↑ RAP → interatrial septal bowing into LA
      - ↓ hypotension
      - ↑ chances of CHB
      - ↑ mortality
    - ② Tricuspid Regurg
    - ③ VSD
    - ④ Premature pulmonic valve opening → RV non-compliance

-Nuclear Imaging:

-gold standard to determine RV EF → radionuclide ventriculography

-normal RV EF ranges broadly (35-75%)

thus ↓RV EF not specific for RV infarction

-↓RV EF w/ wall-motion abnorms of RV →

92% sensitive / 82% specific

-Complications:

① Shock: -most serious complication

② CHB: -poor prognostic factor (~48% of RV infarctions)

③ AFib: -occurs in ~1/3 pt's (? due to RA dilation vs. atrial infarction)

④ VSD: -catastrophic (usually)

⑤ RV Thrombus: -may lead to PE

⑥ PE: -especially if ④ RV thrombus

⑦ Tricuspid Regurg

⑧ Pericarditis

⑨ (R)-to-(L) shunt: -Requires a PFO

-suspect in persistent hypoxemia not responsive to O_2

-Treatment:

Ⓐ Early: -Maintain RV preload

- ↓ RV afterload
- Inotropic support (Ex.) Dobutamine)
- Reperfusion

-fine line w/ fluid admin to ↑ RV preload →

- ↑ RV filling pressures w/ IVFs → further RV dilation → further restrains pericardium for LV function

- General practice:
  - Give IVF's x several L's → if CO not responding then add Dobutamine
- Avoid preload reducers:
  - (A) Diuretics
  - (B) Nitrates

(B) Maintain AV Synchrony:
- if AFib → consider prompt cardioversion
- if CHB → AV pacing superior to V-pacing alone
  - ↑'s CO

(C) Concomitant LV Infarction:
- assoc. w/ LV dysfunction → ↓ CO
  - ↑ RV afterload
- can consider afterload reducers
  - $Na^{\oplus}$ Nitroprusside
  - IABP
  - will unload LV and consequently RV too

(D) ? Role For Fibrinolytics:
- pt's w/ reperfusion → ↑ RV EF's w/ ↓ CHB
  - vs.
  - ϕ reperfusion

Prognosis:
- generally favorable
- IwMI mortality: 6%
- IwMI plus RV infarction Mortality: 31%
- most pt's will re-acquire their RV function so long-term prognosis often limited by concomitant LV infarction.