ACUTE HEART FAILURE: CASE ONE

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POLLING TECHNOLOGY

What do you prefer?
1. ICU
2. CCU
OUTLINE

- Two case presentations
- Pathophysiology
- Priorities in care
- Interventions
- Outcomes

CASE 1

Background info:
- 54 year old man (JG) presented to ED with
  - central chest pain (7/10), that has persisted for about 8 hours.
  - Was doing some heavy lifting yesterday- thought it was related.
  - Has started to feel short of breath over the last hour.
  - Decided to come to hospital to get checked out.
ED TRIAGE ASSESSMENT

- No known cardio-respiratory disease
- Father had a CABG 15 years ago
- Non-smoker and infrequent ETOH
- NKDA
- No prescription medications
- Vitals:
  - BP 100/80 (86)
  - HR 115 bpm
  - SpO₂ 92% on R/A
  - RR 26 bpm
- Based upon presentation: 12 lead ECG completed in triage area

12 LEAD ECG
INTERPRETATION

1. NSTEMI
2. STEMI
3. Pericarditis
4. A-OK

STEMI

- ST segment elevation in anterior and lateral leads.....
INITIAL INTERVENTIONS/ INVESTIGATIONS

- Moved to critical care area:
  - A/E R=L with faint crackles to lower/ mid lobes
  - Audible S1 & S2, no murmurs
  - PPP, but extremities cool
  - Peripheral IVs started and bloodwork drawn
  - Usual interventions started:
    - Aspirin 162mg chewed
    - O₂ not applied because SpO₂ >90%
    - Nitro s/l spray
    - Morphine IV
  - ERP reviewed ECG, history, assessment findings, fibrinolytic checklist and decides to offer reperfusion with fibrinolysis.

INTERVENTIONS

- Consent obtained
- Fibrinolytic protocol commenced
- Vitals:
  - BP 96/69 (78)
  - HR 117bpm
  - SpO₂ 90% on R/A (O₂ commenced)
  - RR 28 bpm
  - Neuro vitals remaining normal
  - Chest pain 2/10
- Transferred to ICU to await transfer to PCI centre (pharmacoinvasive strategy)
ON ARRIVAL TO ICU

Nursing Assessment:
- Vitals:
  - BP 90/62 (71)
  - HR 123bpm
  - SpO₂ 88% on 2L O₂ via np
  - RR 28 bpm
  - Neuro vitals remain normal
  - Chest pain reported at 3/10
- A/E R=L with coarse crackles to lower/ mid lobes
- Audible S1, S2, and S3 now
- Difficult to palpate peripheral pulses
- Extremities cool to knees/ mid-forearm

CARDIOGENIC SHOCK

- A condition of inadequate tissue perfusion caused by cardiac dysfunction
- The patho:
  - Loss of more than 40% of functioning myocardium
  - Impaired ability of the LV to pump blood forward
  - Decreasing SV
  - Decreased CO= reduced end organ perfusion
- Compensatory mechanisms to increase CO and restore tissue perfusion:
  - Vasoconstriction
  - Enhanced myocardial contractility
  - Increased heart rate
  - Increasing intravascular volume (renal and interstitial)
COMPENSATORY MECHANISMS

Counterproductive:
- Compensatory vasoconstriction and increased heart rate:
  - Increases afterload and myocardial O₂ demand
  - Worsens cardiac output for failing LV
- Increasing intravascular volume
  - Increases preload and end diastolic volume on failing LV
  - Increased myocardial O₂ demand
  - Overstretched LV = worsening SV (Starling’s Law)

End result = worsening tissue perfusion!

BACK TO THE CASE

Worsening status:
- BP 78/48 (58)
- HR 127bpm
- SpO₂ 91% on 100% NRB
- RR 38 bpm
- Obtunded, GCS 9 (E2,V2,M5)
- Anuric
- Mottling to peripheries
- ++crackles to lungs

Managing this in a centre that is not PCI capable and doesn’t have a balloon pump?
INTERVENTION IN ICU

- Revascularization
  - Failed fibrinolysis
  - PCI restores vessel patency, limits infarct size, and improves survival
- Stabilize and optimize the patient until he can be transferred to a PCI capable centre

PRIORITIES IN NURSING CARE

Prevent progression of cardiogenic shock and optimize tissue perfusion:

1. Limit myocardial O\textsubscript{2} demand
2. Enhance myocardial O\textsubscript{2} supply
3. Maximize cardiac output
4. Provide comfort and support
5. Monitor for complications
PRIORITIES IN NURSING CARE

1. Limit myocardial O₂ demand
   - Administer analgesics, sedatives
   - Administer medications to control afterload
   - Limit activities

2. Enhance myocardial O₂ supply
   - Intubate and ventilate
   - Monitor respiratory status
   - Administer prescribed medications
   - PCI

3. Maximize cardiac output
   - Prevent dysrhythmias
   - Administer antiarrhythmics
   - Administer inotropes & vasopressors
   - Optimize preload (cautiously)

4. Provide comfort and support
   - Quiet environment, family present

5. Monitor for complications
   - Further reduction in tissue perfusion
   - End-organ failure
PHARMACOLOGICAL SUPPORT IN CARDIOGENIC SHOCK

What pharmacological option would you anticipate the physician will order to maximize cardiac output and tissue perfusion?

1. Norepinephrine
2. Dobutamine
3. Dobutamine + norepinephrine
4. Dopamine

- Remember the vitals:
  - BP 78/48 (58)
  - HR 127bpm
  - SpO₂ 91% on 100% NRB
  - RR 38 bpm

PHARMACOLOGICAL SUPPORT IN CARDIOGENIC SHOCK

- Dobutamine ($\beta_1$ & $\beta_2$) + Norepinephrine ($\alpha$)
  - Dobutamine ($\beta_2$) to increase contractility and CO
  - Norepinephrine ($\alpha$) to treat hypotension and counteract the vasodilation caused by dobutamine ($\beta_2$)

- Evidence suggests better outcomes with norepinephrine rather than dopamine.
  - No difference in treatment effect
  - Higher rate of death and arrhythmias with dopamine
RESULT OF INTERVENTIONS

- BP 91/50 (63)
- HR 115 bpm
- SpO₂ 94% on fiO₂ 80% PCV insp 30cmH₂O, PEEP 10cmH₂O, RR 15bpm
- Minimal spontaneous effort
- Sedated on midazolam 3mg/hr and fentanyl 50mcg/hr
- Anuric
- Mottling to limbs improved, but still cool from knees down
- Lactate improving
- +crackles to lungs
- Neuro vitals stable

TRANSFER

- The storm passes!
- Life Flight arrive and transport patient to PCI centre where he undergoes urgent PCI
  - Complete occlusion of proximal LAD and 90% occlusion of left circumflex
- PCI limits infarct size and interrupts the cycle of cardiogenic shock
- Over the next two days, the patient is weaned from the pharmacological support and extubated.
Questions?

References


REFERENCES


ACUTE HEART FAILURE: CASE TWO
CASE 2

Background info:
- 16 year old boy (DW) seen in the ED in a community hospital:
  - Went to Spain with older brother 6 weeks ago
  - Described a viral illness off and on since
  - Increasing fatigue over the last week
  - Mother noted today he looked more drowsy and a bit yellow now
  - Off to the ED

ED ASSESSMENT

- Alert and orientated, but drowsy with difficulty focusing
- BP 100/60 (73), HR 102 bpm, RR 18bpm, SpO₂ 95% on R/A, Temp 37.2°C
- Faint crackles auscultated to bases
- Tender abdomen (upper right quadrant)
- Jaundiced
- Denies any drug use
- Initial blood work sent
INITIAL BLOOD WORK

Hb- 138
Plt- 110
WBC- 11.6
INR- 2.4
Total bilirubin- 26µmol/L
AST- 1750 U/L
ALT- 1468 U/L
Alk Phos- 350 mU/mL

WHAT’S HAPPENING?

Based upon what you know so far, what do you think is going on?

1. Sepsis
2. Acute liver failure
3. Acute cholecystitis
4. Illicit drug ingestion
5. I thought this presentation was about heart failure?
INITIAL DIAGNOSIS

Acute Liver Failure
- Abrupt onset of:
  - Jaundice
  - Hepatic encephalopathy
  - Coagulopathy

TRANSFER

Because of abrupt onset of jaundice, neurological symptoms, abnormal LFTs and coagulation results, DW is transferred to a liver center for further assessment and treatment.
LIVER TRANSPLANT STEP DOWN UNIT

- Upon arrival, the transplant unit nurses confirm the previous findings and:
- Heart Sounds auscultated this time:
  - Expected to hear:
  - But they actually heard this:

WHAT DO YOU HEAR?

- Expected:
- Heard:

1. Systolic Murmur (aortic stenosis or mitral regurgitation)
2. Diastolic Murmur (aortic regurgitation or mitral stenosis)
3. Who cares? He has liver failure!
HEART SOUND

- Systolic Murmur
  - Mitral Regurgitation:

- Causes of MR:
  - ischemia, collagen disease, infection, calcification, ventricular dilation

FURTHER INVESTIGATIONS

- After the initial admission assessment, investigations are ordered for the liver failure workup:
  - CXR
  - Ultrasound doppler of the liver
  - Lab work for viral hepatitis, drug toxicity, Wilson’s disease, etc
What do you think of the CXR?
1. Looks good
2. Looks bad
ENLARGED HEART

NOW WHAT?
• More tests:
  • ECHO
    • Dilated cardiomyopathy
NEW DIAGNOSIS

- Dilated Cardiomyopathy
  - LV dilation and systolic dysfunction
  - Often involves the RV as well

Causes:
- idiopathic
- genetic
- myocarditis
- alcoholic

DIAGNOSIS

Diagnosis is determined to be:

- Idiopathic Dilated Cardiomyopathy
  - Most common cause of heart failure in the young
  - Unlikely to be genetic, alcoholic, toxin
  - Suspected to be a result of viral myocarditis

- Post Myocarditis:
  - Well known cause of DCM
  - Viral infection is the most common cause of myocarditis
  - Culprit viruses...
  - The pathophysiology...
  - Prognosis
**But why the liver failure?**

- Pathology not well understood
- Hepatic congestion
- Reduced hepatic $O_2$ delivery from impaired cardiac output
- Threshold for hepatic $O_2$ delivery

**Back to the Case**

- How is DW doing now?
  - Diagnosis is DCM (viral cause) with acute liver failure
  - Goal of care: improve cardiac function!
  - Transferred to ICU
  - Currently self ventilating, central line is being inserted, and assessment findings are:
VITALS NOW:

- BP 86/54 (64), HR 108 bpm, RR 36bpm, SpO₂ 90% on 100% NRB, Temp 36.4°C
- Worsening crackles auscultated to lung fields
- Difficult to rouse
- Cool peripheries
- Urine output 10mL/hr
- ABG:

ARTERIAL BLOOD GAS

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1. Respiratory acidosis
2. Respiratory alkalosis
3. Metabolic Acidosis
4. Metabolic alkalosis
ARTERIAL BLOOD GAS

- Metabolic acidosis with partial compensation and hypoxemia

  - pH: 7.28
  - pCO₂: 23
  - pO₂: 57
  - HCO₃⁻: 10
  - BxE: -14.6
  - O₂Sat: 86.1%
  - Lactate: 9.5

CARDIOGENIC SHOCK

- End organ hypoperfusion:
  - Acute Liver Failure
  - Impending Respiratory Failure
  - Neurological Compromise (cardiac/ hepatic cause)
  - Renal Failure (cardiac/ hepatic cause)
Reduced SV

Reduced CO

Blood backing up (pulmonary edema)

Worsening gas exchange

Further worsening tissue perfusion

Further worsening acidosis

Myocardial depression from hypoperfusion and acidosis

Reduced effective pumping

Multi Organ Failure

INTERVENTIONS

- Supportive Care (busy Nurse!):
  - Assist with intubation,
  - Ventilate and sedate
  - +/- PA catheter
  - Assist with insertion camino bolt for ICP monitoring and treatment
- Monitor for progression of liver failure and treat
- Monitor for progression of renal failure and treat
INTERVENTIONS

Team’s Goal:
    Restore tissue perfusion with conventional heart failure therapy:
    - Monitor for brady/ tachyarrhythmias
    - Treat systolic dysfunction
      - Administer dobutamine gtt (positive inotrope)
        - $\beta_1$ receptor agonist
        - Mild $\beta_2$ agonist activity

INTERVENTIONS:

- This causes the BP to go down to 79/45 (56), and HR up to 124bpm.
- What next?

- Norepinephrine: vasopressor (α receptor agonist with some $\beta_1$ receptor activity)
Later in the shift...

- Despite initial improvement with the addition of norepinephrine, DW continues to deteriorate with ongoing progression of end-organ hypoperfusion:
  - Anuric
  - Significant pulmonary edema and unable to tolerate PEEP greater than 10cmH₂O
  - Mottled peripheries
  - Increasing lactates

Further steps to enhance tissue perfusion

- Commence CVVH
- Insert a balloon pump (IABP)
  - Counterpulsation causes:
    - Reduced afterload
    - Increased coronary and systemic perfusion
    - Increased CO
IABP

IABP clip
BUSY NURSE!

- Nursing care, monitoring, management of the patient with the IABP
  - Neuro assessment q1h
  - Assess perfusion to peripheries q1h
  - Assess urine output q1h
  - HOB at 30’ with cannulated leg straight
  - Check IABP timing q12h and prn
  - Check IABP insertion site q1h
- Running CVVH
- Monitoring ICPs
- Titrating inotropes and vasopressors
- Obtaining ABGs, interpreting and adjusting mechanical ventilation

CONTINUED DETERIORATION

- Despite initial improvement after insertion of the balloon pump, further deterioration of tissue perfusion
**CURRENT STATUS/ VITALS**

- BP- 78/45 (56)
- HR- 110bpm
- SpO₂ 90% fully vented on 100% fiO₂, with PEEP 10cmH₂O
- Blood work
  - Lactate- 11
  - ABGs: pH 7.11, pCO₂ 36, pO₂ 58, HCO₃ 8, BxS -17
- Family counseled that DW may not survive this admission

**FURTHER INTERVENTION**

- Insertion of percutaneous ventricular assist device (pVAD)
- Left atrium to femoral artery bypass
  - Unloads the left ventricle
  - Maintains perfusion to end-organs
- Bedside in-service for the liver transplant nurse!
**PERC LVAD**

**Now**
- MAP only
- 2 RNs caring for DW
- Still hoped for spontaneous recovery with supportive measures
- ABGs improved initially, then further deterioration
- Resident gave up hope for survival
- Patient prepared for transfer to Heart Transplant centre
**TRANSPLANT CENTRE**

- Assessed for candidacy for a heart transplant
- Listed
- Received a heart transplant

**OUTCOME**

- 6 weeks later, his Mom came to see us to tell us that he was recovering well at home with no residual complications from his acute illness.
QUESTIONS?

REFERENCES


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