Case Based Learning Series

"Student Led Adult Learning"

CPC CASE-BASED LEARNING SERIES
"Mastering clinical Reasoning in Emergency Medicine"

Seed GLOBAL HEALTH

Clinical Pathologic Case (CPC)

Emergency Medicine Case Based Series

My Body is Paining

Date: Friday 25th July 2025 | **Time**: 7:00pm - 8:00pm (EAT)

SCAN QR CODE TO REGISTER

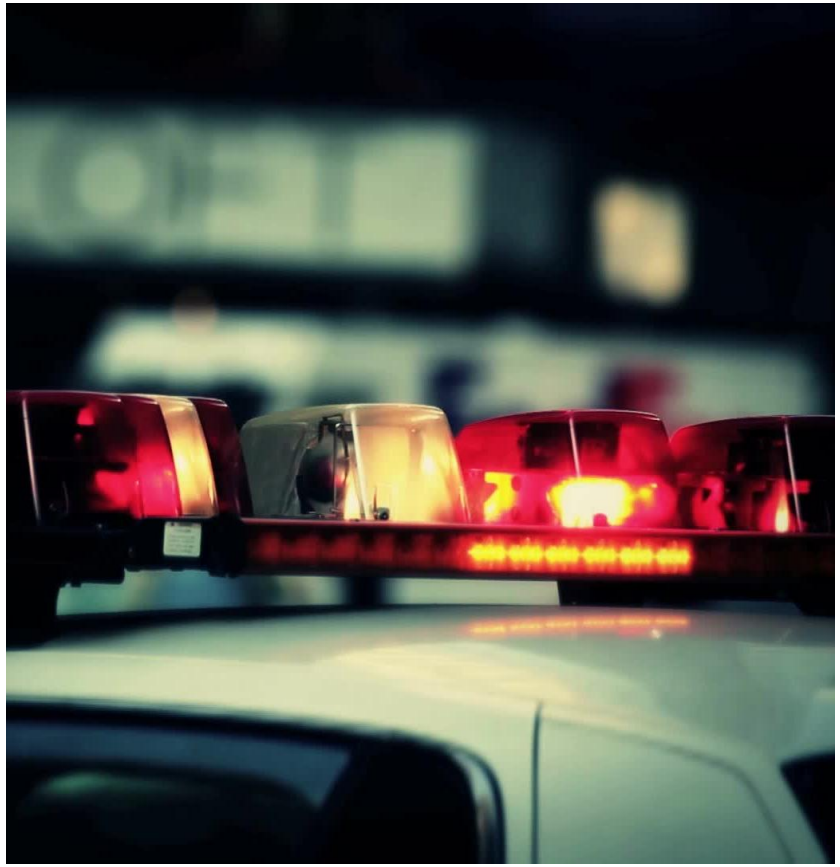
Expert	Mentor	Presenter	Presenter
			
Dr. Emuron Joseph EM Physician	**Dr. Banturaki Amon** MBChB, MMED, MPH	**Maria Natasha Namujja** 5th Year Student, KIU	**Bongozana Melvin** EMT Presenter

FUMSA

SCOME Medical Education

Presenting Complaint

35-year-old female presented with severe generalized body pain x 2/7.



Prehospital team Care

- **Staff:** 2 EMTs, Ambulance driver
- **Patient**: 1 patient who is currently non emergent
- **Equipment / Medications**: Communication devices, patient monitor, Normal saline, oxygen source, nasal prongs, syringes, non rebreather masks, giving sets, cannulae, penlight, simple face mask. Antibiotics, analgesics IV crystalloids
- **Mode of transport**: Type B ambulance
- **Documentation/Handover:** Dispatch note with vital signs

Prehospital team Care

Identify: I am Melvin an EMT called to pick up this patient

Background: I have a 35yr/F with h/o sudden worsening generalized body pain x2/7 and bilateral knee swelling.

Assessment: Mild R.D, with rapid pulses, bilateral swollen knee joint and fevers . Enroute, administered O_2, morphine, PCM

Recommendation: Continue IV fluids, O_2, morphine, PCM. Review by physician

Primary survey

PARAMETER	FINDINGS
Airway	Patent
Breathing	-RR -22bpm, SPO_2 -92% in room air - equal bilateral air entry and no added sounds,
Circulation	- PR -108bpm, rapid, regular and bounding - BP-123/88mmHg, CRT <2secs - Extremities were pale and warm
Disability	- GCS(15/15),PEARL , RBS- (not done)
Exposure	-Temperature-39.2^{o}C - No traumatic injuries

Poll 1

From the primary survey, what are the likely emergent conditions in this patient?

What are Emergent priorities?

	PRIORITY	*Findings*	*Immediate Action Taken*
B	Tachypnea & hypoxia	RR= 22 bpm SPO_2= 92%	Administer 2L/min of O2 by nasal prongs
C	Dehydration	PR -108bpm, rapid, regular and bounding	IV normal saline 1.5 litres
E	Hyperpyrexia	Temperature-39.2^{o}C	Tabs pcm 1gm STAT Encouraged tepid sponging Removed excess clothing

SAMPLE History

- **Signs & Symptoms:**

Severe generalized body pain, bilateral knee joint swelling

- **Allergies:**

No known allergies

- **Medications:**

Folic acid, penicillin V, ART (unknown regimen)

Continuation.

- **Past Medical Hx:**

Numerous admissions in her life due to similar episodes however this was the 2nd admission this year due to similar signs and symptoms.

8 previous blood transfusions

- **Last Meal-** lunch 6hours prior to admission

- **Events Leading to Presentation**: No clear trigger

Audience

- Any additional information?

Expert

What are your initial thoughts?

What is you preparation and approach to this patient?

Secondary survey

General: Alert, oriented, not in respiratory distress, Moderately dehydrated ,no cyanosis, no finger clubbing and no lymphadenopathy

Extremities: Bilateral lower limb pitting oedema (Grade 1)

Genitourinary: Oliguria , suprapubic tenderness, increased urgency and frequency of urination

Musculoskeletal: Bilateral knee joint swelling, warm and tender to touch; no deformities or erythema noted

Other Systems (CVS, Respiratory, Abdomen, CNS): Unremarkable.

Audience

- Any additional information?

Expert opinion?

Any additional thoughts at this point?

Any additional info you would want to get?

Investigations

Investigations	Results
CBC with differentials	WBC -28.64 x 10^9/L, (**5.00- 11.60**) Lymp. 7.25×10^9/L, (1.30 -4.00) Hb: 6.3 g/dL , (**11.5- 15.1**) PLT: 1027× 10^9/L, (**156 -342**)
LFTs	- D-Bilirubin -14.77mg/dL , (**0.1-1.2mg**/dL - I-Bilirubin- 33.32 mg/dL, (**0.68-1.12 mg/dL)** - T-bilirubin - >**7.00 mg**/dL. - AST-338 U/L (**0-33U/L)** - ALT- 67U/L. (**0-32U/L)**
RFTs	- Creatinine - 3.33 , (**0.58-1.12**)**mg/dl** - Urea- 109.8 mg/dl, (**15- 43)mg /dl**
Urinalysis	-Yellow and turbid -Nitrites++, Pus cells++,Blood _ trace
Blood slide	No malaria parasites

Poll 2

At this point, what are your possible

Differential Diagnoses

for this patient?

Differentials	Supportive findings	Contradicting findings
Sickle cell VOC	Known sickle cell anemia, generalized body pain for 2 days (severe), numerous admissions for similar episodes Bilateral knee joint swelling Anaemia (Hb<6.3g/dl) Fever	None
Septic arthritis	Joint swelling HIV infection Warm, tender knee joints Fever Leukocytosis (WBC -28.6)	Usually affects unilateral joints On joint Aspiration, there was no pus Concurrent UTI
Urinary tract infection	History of increased urgency and frequency of urination Suprapubic tenderness Urinalysis: Nitrites ++, Pus cells ++, turbid	None
Malaria	History of generalised body pain, Joint pain Fever anaemia	No malaria parasites seen on blood slide

ED course

Working diagnosis;

1) Urinary Tract infection

2) SCD Vaso-occlusive crisis complicated with acute kidney injury (AKI)

Poll 3

What are your management

Priorities

for this patient?

Management plan

- Continue oxygen therapy 2L -4L
- Oral morphine solution 10mg every 4hrs as needed
- Tabs paracetamol 1g tds *3/7
- Encourage tepid sponging
- Iv fluids NS 1.5L in 24hrs
- Transfuse with 460mls of packed red blood cells
- Iv ciprofloxacin 400mg b.d *3/7
- Tabs folic acid 5mg o.d *1/12

Expert

Pearls and pitfalls

Overall communication

1. Pathophysiology of AKI in SCD
2.The need for aggressive hydration, pain control and monitoring

1. Pathogenesis of AKI in SCD

- The major pathophysiological consequences of SCD that cause AKI are two among other multifactorial causes

a) Vaso-occlusive crisis

b) Haemolysis

a) vaso-occlusive crisis

In this crisis there is occlusion of microvasculature in the kidney leading to a shortage of oxygen and nutrients in the kidney tissues.

Prolonged ischaemia can cause kidney cells to die leading to acute kidney injury.

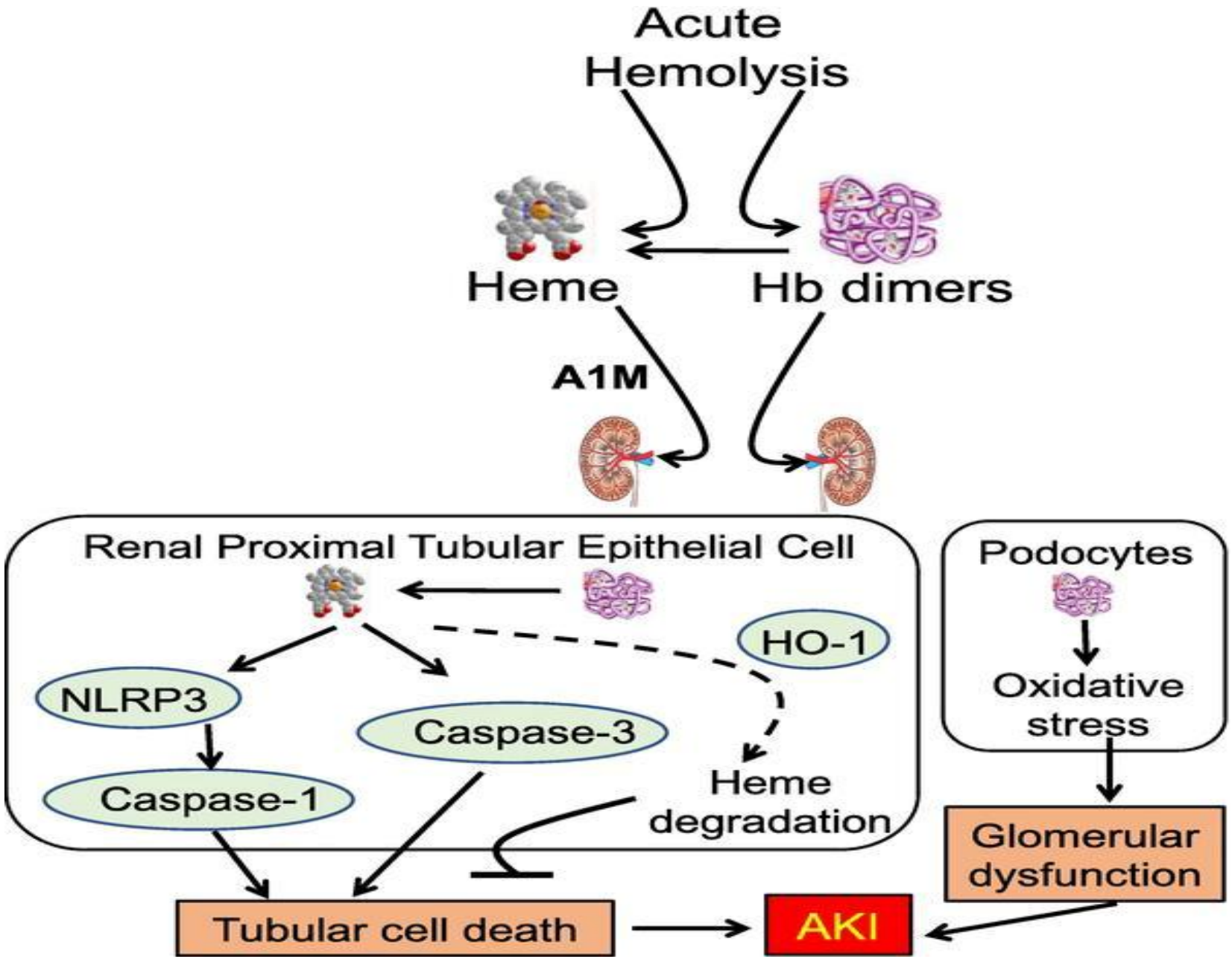
Continuation

b) **Hemolysis**

- Sickle red blood cells have a shorter life span and break down easily leading to production of free haemoglobin and heme .

- Free haemoglobin is directly toxic to the kidney cells and may cause podocyte injury leading to glomerular dysfunction.

- Excess heme is binded to alpha-1 microglobulin which is transported to renal tubular epithelial cells and it’s excess deposition causes proximal tubular epithelial cell death and promoting AKI


Acute
Hemolysis
Heme
Hb dimers
A1M
Renal Proximal Tubular Epithelial Cell
HO-1
NLRP3
Caspase-3
Caspase-1
Heme
degradation
Podocytes
Oxidative
stress
Glomerular
dysfunction
Tubular cell death
AKI

2. pain control, hydration and monitoring

- **Pain control**

Uncontrolled pain stimulates the sympathetic Nervous system leading to release of catecholamined that cause vasoconstriction, worsening sickling and causing more pain.

How.....? Opiods are the first line (morphine, hydromorphine) , adjuvants (NSAIDs, acetaminophen, gabapentin)

- **Hydration**

Adequate Hydration improves blood flow to the body tissues and prevents further sickling. Hydration improves renal blood flow and tubular function thus preventing AKI progression.

How.....? IV fluids(0.9%saline or LR) at 1.5 × maintenance

Goal urine output >0.5ml/kg/hr

Contn.

- **Close monitoring**

For early detection of life-threatening complications such as ACS, stroke, bacteremia, sepsis

How....? Monitoring vitals e.g hourly pulse oximetry , labs like CBC , creatinine, LDH, frequent clinical examination

EDUCATION:

- Highlights
- QR code with resources

References

1.Kidney Injuries in Sickle Cell Disease | IntechOpen
https://www.intechopen.com/chapters/80581