ECG

HYPERKALEMIA'S FOOTPRINT ON THE ECG : A VISUAL GUIDE FOR CLINICIANS

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OUTLINE

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Hyperkalemia's Footprint on the ECG : A Visual Guide for Clinicians

A Narrative Review

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It is wisdom to envision the footprint of time and to plan the further steps accordingly. One may thus protect oneself from the catastrophic events of adversity , which seem to approach through the rustling wind of threat.

Cracking the code of hyperkalemia on the ECG allows its signs of impending adversity to be recognized earlier. Though it is an alarming puzzle , understanding its electrocardiographic dynamics may pave the way to saving the patient's life. ECG changes in hyperkalemia are markers of urgency and severity.

- ☐ **Hyperkalemia slows the race of life , resembling a tortoise's journey—not one of triumph, but of a terminal end.**
- ☐ **It drives a double-edged progression—on one side, the slowing of the conduction pathways; on the other, the decay of myocardial responsiveness. These paths runs in parallel until they collapse into a preterminal warning : the sine wave.**

The ECG is a rapid and invaluable bedside diagnostic tool in hyperkalemia

1. Introduction (Keypoints)

- The ECG is the first whisper of hyperkalemia before the heart cries out – the heart does not wait for lab result. Recognizing such changes in time is not just diagnostic skill – it is a clinical foresight as well..
- The status of hyperkalemia is strengthened in the presence of symptoms such as nausea , vomiting , paraesthesia , muscle weakness , palpitation , unability to standing up ± drowsy status in association with the predisposing entities as enumerated below :
  - Skipped haemodialysis with pre-existing renal disease
  - Acute kidney injury
  - History of diabetes mellitus / hypertension as a predisposing cause of renal insufficiency
  - Drugs
    Potassium-Sparing Diuretics, including Spironolactone ,
    ACE inhibitors , ARBs.
  - Excessive consumptions of potassium rich diet (fruits , leafy vegetables , potatoes , etc.) by a patient with predisposed conditions.
  - History of trauma resulting in 'Rhabdomyolysis'
- If the wave pattern on ECG is suggestive of hyperkalemia , it has a high specificity for its diagnosis and it would be worth to initiate the concerned emergent treatment as per need.

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Here to mention that **Venous blood-analysis** provides an immediate result of the patient's potassium level.

- The waves suggestive of hyperkalemia on ECG are more dramatic in its behaviour with faster evolution toward its lethal journey (sine wave , cardiac asystole / ventricular fibrillation). There should be no delay in the institution of its treatment , as needed. **One suggested regimen :**
    - 10 ml of 10% of calcium gluconate mixed with 100 ml D5W or NS to be infused over 5-10 minutes (it may be repeated as per need to achieve QRS < 100 ms or till the appearance of P-wave on ECG)
    - 2 Amps of D50W followed by 10 units rapid acting insulin IV
    - Salbutamol 8 puffs by aerochamber or 20 mg nebulized , but after IV insulin

  Immediately refer the case to the expertise centre for further treatment and haemodialysis , as per need.

2. Electrophysiological Sequence Under Normal Condition

The Cardiac Action Potential is a series of brief changes in voltage across the cardiac cell membrane, brought about by fluxes of ions through ion channels.

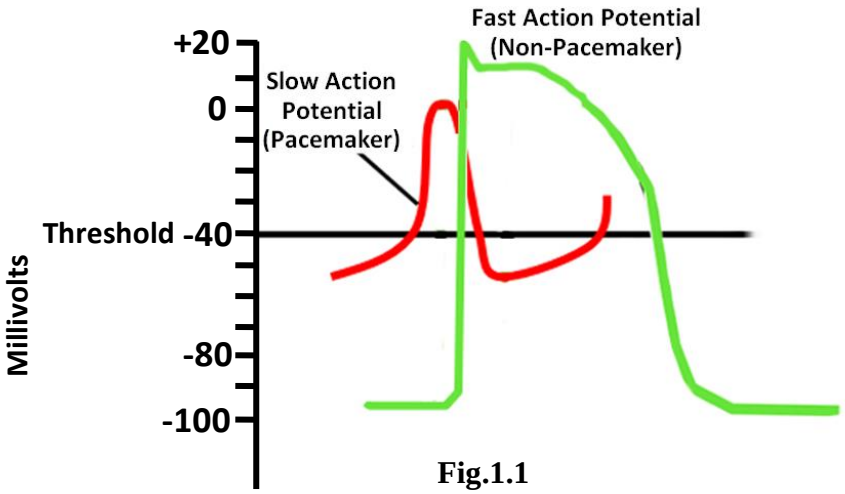

Fig.1.1

There are two sets of action potential :

- **Fast Action Potential** : This potential is seen in contractile cells (atria and ventricles) that are backed up by rapid depolarization due to the opening of fast $\mathbf{Na^+}$channels. Since HIS bundle - purkinje system is connected to the ventricular myocytes , it also shares the fast action potential to allow the impulses to pass rapidly through it.

- **Slow Action Potential** : The SA and AV nodes , being rich in slow $\mathbf{Ca^{2+}}$ channels cause different ion channel kinetics. The initial potential of this nodal system is less negative compared to the RMP of the contractile units of myocytes. This lesser negativity allows the action potential to be automatically activated in a slowly rising semi-curve manner. That's why , this nodal system remains somewhat resistant to the impact of hyperkalemia.

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3. Sequential electrophysiological steps with hyperkalemia

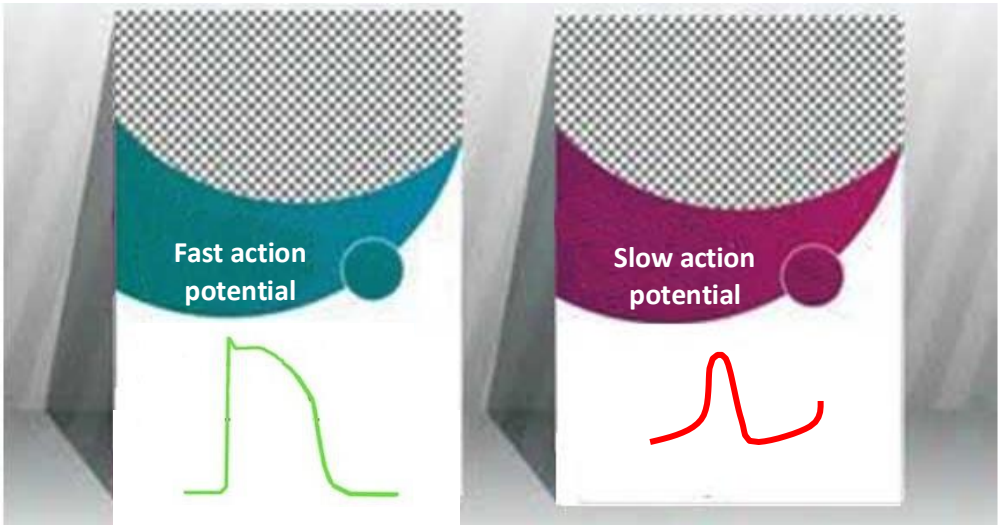


Fig.1.2

Fundamentals :

- Hyperkalemia slows both resting membrane potential and action potential propagation in cardiac myocytes.
- Surplus extracellular potassium makes the resting cardiac membrane less negative → partial depolarization→ inactivation of channels
  No proper depolarization → impaired myocytes contractibility with conduction delay.
- Both fast and slow-response tissues confront a negative ionic terrain. Since Fast action potential (Na+ channels) is having more negative potential -85 to -90 mV compared to that of slow action potential (Ca2+ channels) having -60 to -65 mV , therefore fast channels are affected earlier (SA and AV nodes use slow Ca2+ channels)

Sequential steps are :

❒ **T-Wave Changes & QT Shortening**

Extra potassium outside cells speeds up repolarization phase 3. This causes tall, peaked T-waves and shorter ST segment and QT interval

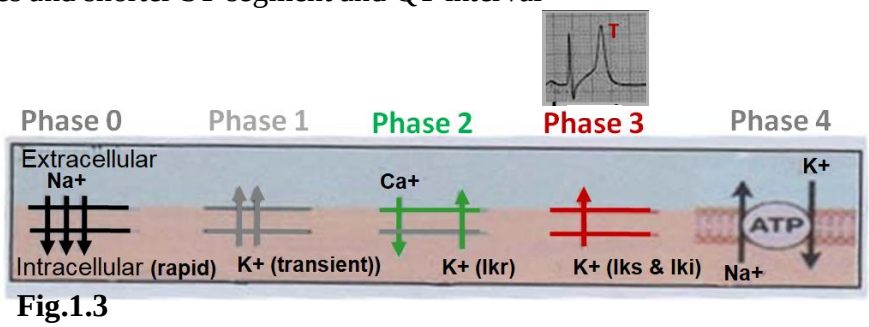
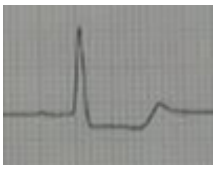


Fig.1.3

❒ **Slowed Atrial Conduction**

Hyperkalemia makes atrial cells less negative, affecting fast Na+ channels first . Atrial myocytes depolarize earlier, leading to slower and weaker conduction , ultimtately sweeping to atrial paralysis (flattened or low-amplitude P-wave with prolonged PR interval).



Flattened P-wave→Atrial standstill

Fig.1.4

❒ **SA & AV Nodes Are Less Affected Initially**

They use slow Ca++ channels, which are less sensitive to early K+ rise but they may be affected later , initially protected by their self automaticity.

❒ **Internodal Tracts Stay Functional Longer**

Though they use Na+ channels, they don't have automaticity , so they resist early effects of hyperkalemia.

❒ **Slowed Ventricular Conduction**

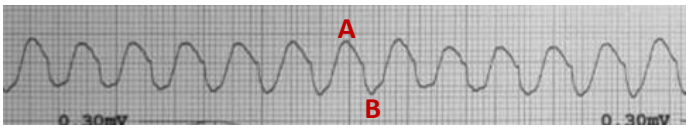
His–Purkinje and ventricles use fast Na+ channels

✓As extracellular potassium rises, it lays its heaviest hand on the most distal segments—be it the Purkinje arborization or the terminal ventricular myocytes.

This descending suppression paints a different electrophysiological spectrum , as illustrated below :

Secondary pacemaker capability of Purkinje fibres is suppressed → infranodal escape pacemaker is unreliable due to its downregulated conduction (either of bundle branches) → associated diffuse intraventricular conduction delay → sine wave → asystole

Fig.1.5



Classical sine wave is displayed here as up (**A** – widened ventricular depolarization phase) and down (**B** – widened ventricular repolarization phase) oscillation

Dual effect of hyperkalemia : Conduction tissue plus myocardial tissue involvement

This classification enables the clinicians to have correlation in between the strength of hyperkalemia and ensuing ECG changes :

Early (K^+ ~5.5–6.0 mmol/L) Slight slowing of SA node; possible mild PR prolongation Tall, peaked T waves (due to faster repolarization)

Mild-Moderate (K^+ ~6.0–6.5 mmol/L) Flattening of P wave, progressive PR prolongation T waves more peaked; beginning QRS widening

Moderate-Severe (K^+ ~6.5–7.5 mmol/L) Loss of P wave; junctional or atrial standstill begins Widened QRS; decreased contractility

Severe (K^+ >7.5–8.5 mmol/L)Bundle branch blocks, AV block, ventricular escape Further QRS widening, T wave fusion begins

Pre-terminal (>8.5 mmol/L) Complete conduction system collapse QRS and T merge → Sine wave pattern

Terminal (>9.0–10 mmol/L) Electrical silence (asystole)

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4. Overlapping ECG patterns in hyperkalemia : A pertinant consideration

Hyperkalemia often presents with unpredictable and overlapping ECG changes. The proposed concept of overlapping ECG changes in hyperkalemia offers a more realistic and clinically appliciable tool for bedside diagnosis. By moving beyond the rigid sequential model , clinicians can better recognize early warning signs , act promptly and potentially prevent cardiac arrest.

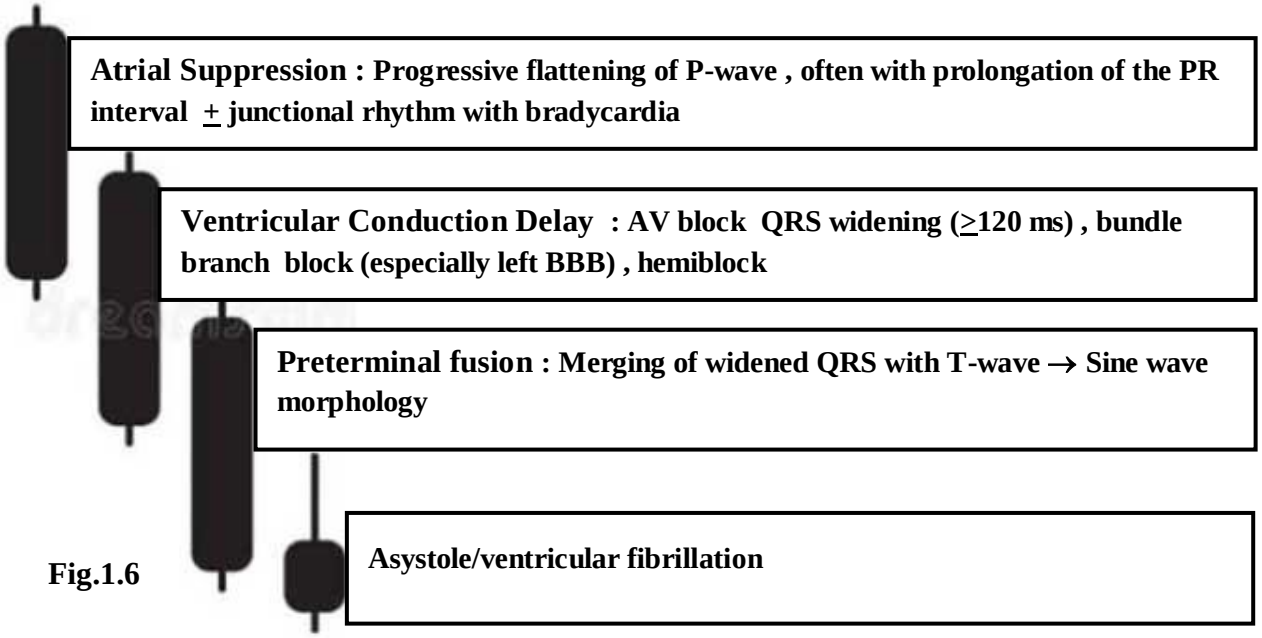

Fig.1.6

With the above sketch , the different stages of hyperkalemia have been illustrated. They may not be limited to stagewise , rather there exists a possibility of overlapping such changes with each other. There may be individual variability in response to hyperkalemia how does it responds to cardiac myocytes and its conductive tissues. The following factors might be responsible for such overlapping :

- As potassium rises , atrial and ventricular changes appear on ECG at different rates , and so they may have overlapping pattern.
- Variable expression of ion channels
  - Individuals have genetic and physiological differences in the density and kinetics of ion channels (e.g., Na^+, K^+, Ca^{2+} channels).
  - These variations cause different thresholds for how the atria, ventricles, and nodes respond to rising K^+.
- Differences in Autonomic Tone and Medications
  Vagal tone, beta-blockers, calcium channel blockers, or digoxin can all modulate how electrical signals propagate through different cardiac tissues.
  This can influence which ECG changes appear first or overlap.
- Underlying Cardiac or Renal Disease
  Conditions like left ventricular hypertrophy, ischemia, or renal failure may alter myocardial contractibility or ± the mode of conduction.

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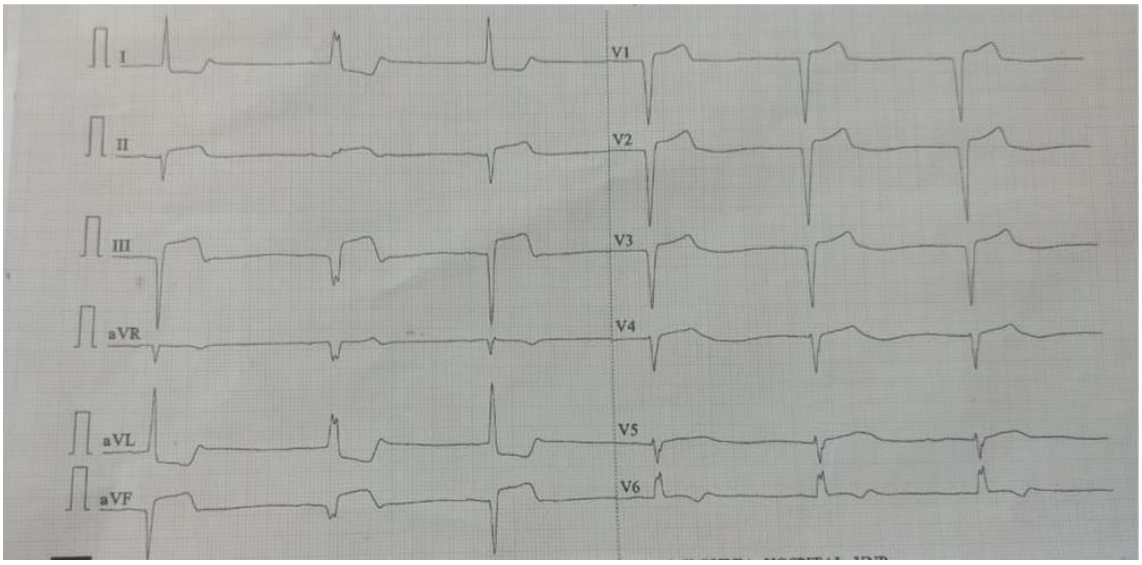
- Rate and Duration of Hyperkalemia
  Acute hyperkalemia leads to more pronounced and synchronized changes.
  In chronic hyperkalemia, the heart may partially adapt, causing some ECG changes to appear blunted or scattered across time and not in a textbook order.

5. An interesting Case study

71 years male presented with one day h/o Diarrhoea , drowsy incoherent state in association .
BP Pulse unrecordable , Non-Diabetic and Non-hypertensive
Serum Na 121 , K 5.86 all in mE/L , 20 ml urine in last 24 hrs , cardiac echo done 2 days back normal



Source : Global Heart Rhythm Forum dated 10th May 2025 , presented by **Dr. R.K Gupta** , Senior Consultant Physician , Yamunanagar , Haryana

ECG Findings	Interpretition
i. Flat P-wave → prolonged PR interval (0.22 sec) → Junctional escape rhythm (with bradycardia) → LBBB pattern (no tall T anywhere on ECG) **NB** : Zoom the ECG tracings to its maximum to view flat P-wave with prolonged PR interval	• A flat or absent P wave does not always signify SA node failure—it often reflects atrial conduction block, while the SA node silently endures (with PR>) • A nodal pattern emerging after flat P waves with bradycardia should rightly be termed a "junctional escape rhythm" • LBBB in association suggests advanced conduction system involvement ± Intermittent LBBB pattern points to somewhat fluctuating level of hyperkalemia
ii. Q-wave with ST elevation in inferior leads (II, III , aVF) with reciprocal ST depression in leads I and aVL	Discordant STE especially in context with lead III with > 1 mm STE > 25% of the depth of the preceding wave (at the LBBB site) – with the presence of Q and ST elevation in other inferior leads suggests inferior STEMI (modified Smith-Sgarbossa criteria)

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The stepwise detailing of the entire clinical scenario :

✓ Please read through the investigation reports as laid down in the previous page with red outlines

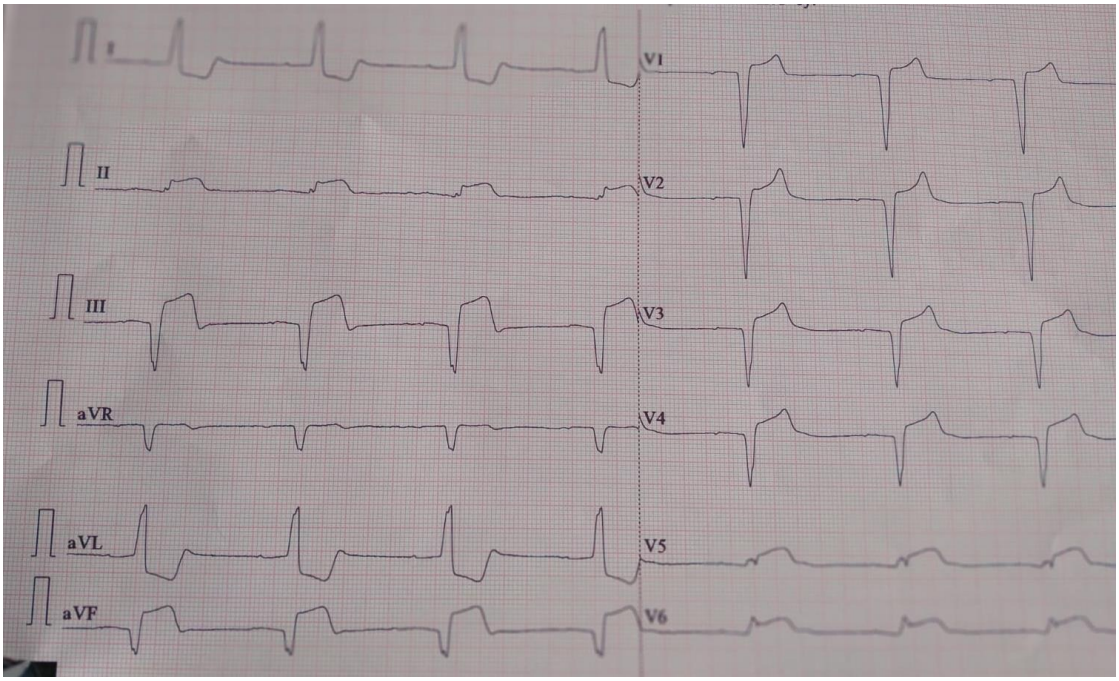
Step 1 : Most possibly the patient had passed into hypovolemia related Acute kidney injury (AKI)

Step 2 : Possibly further progressed to the stage of Hyperkalemia -renal induced

Step 3 : ECG findings suggestive of hyperkalemia , as elaborated in the previous page.

Step 4 : Evidence of Acute STEMI (inferior) , the details discussed in the previous page.

ECG of the same patient recorded after 1 hour : the findings are more or less the same except there is also evolution to anterolateral STEMI (V5 V6) in addition



Discussion :

This case is indeed stirring and educationally rich—a dramatic yet tragically oriented cascade that deserves thoughtful exploration – diarrhoea of one day standing in this old patient → hypovolemia-induced AKI → secondary hyperkalemia → electrocardiographic manifestations of acute STEMI , as discussed above. Further to say , the causative factors involved here with the absence of tall T-wave inspite of this cascade of hyperkalemia would be worth to be discussed here :

- Myocardial tissue by acute MI was most possibly so suppressed that ventricular repolarization (T-wave) no longer appears exaggerated.
- The existing LBBB pattern on ECG might had masked typical tall T-wave changes
- The history by itself suggests the rapid evolution of renal induced secondary hyperkalemia which might have overwhelmed the myocardium before repolarization changes to manifest.

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- T-wave may be deceptively normal in hyperkalemia
  In approximately 20–25% of hyperkalemia cases, the T wave may remain normal in shape and amplitude throughout, even with moderate to severe elevations of serum potassium. This variant presentation is often termed **"electrocardiographic silence"** of hyperkalemia.

This would be worthwhile to discuss here some pertinent issues concerned with this case :

- ❒ Here there is involvement of left bundle branch block (LBBB) as a marker of conduction delay. Hyperkalemia causes generalized conduction slowing, but the anatomical complexity, higher sodium-channel dependence, and greater functional demand of the left bundle make it more likely to show this block earlier and more obviously than the right bundle.
- ❒ It is also known fact that acute MI may also cause LBBB pattern on ECG.
  Patients presenting with acute myocardial infarction (MI) and a left bundle branch block (LBBB), the presence of hyperkalemia (elevated potassium levels) can complicate the diagnosis. It is crucial to recognize and treat hyperkalemia promptly to prevent adverse cardiac events with improved outcomes.
- ❒ The association of Hyperkalemia with acute myocardial infarction exposes the patient to increased mortality by further deteriorating the renal function.

6. Take Home Message

- ☐ The ECG is a rapid and invaluable bedside diagnostic tool in hyperkalemia
- ☐ The status of hyperkalemia is strengthened in the presence of symptoms such as nausea , vomiting , paraesthesia , muscle weakness , palpitation , unability to standing up ± drowsy status in association with the background of hyperkalemia predisposing conditions.
- ☐ The waves suggestive of hyperkalemia on ECG are more dramatic in its behaviour with faster evolution toward its lethal journey (sine wave , cardiac asystole / ventricular fibrillation). There should be no delay in the institution of its treatment , as needed
- ☐ Hyperkalemia narrates a tortoise's journey—not one of triumph, but of a terminal slowing. It begins quietly, flattening the P wave, prolonging the PR, widening the QRS. At each step, the conduction tissues tire, the myocardium dulls, and what was once a brisk sprint of depolarization becomes a heavy, labored march. Eventually, the tortoise halts—not from exhaustion, but from electrical silence with a preterminal warning as the 'sinus wave'.
- ☐ The proposed concept of overlapping ECG changes in hyperkalemia offers a more realistic and clinically appliciable tool for bedside diagnosis. By moving beyond the rigid sequential model , clinicians can better recognize early warning signs , act promptly and potentially prevent cardiac arrest (for details see page 5-6)

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☐ Some pertinent consideration :

- In approximately 20–25% of hyperkalemia cases, the T wave may remain normal in shape and amplitude throughout, even with moderate to severe elevations of serum potassium. This variant presentation is often termed **"electrocardiographic silence"** of hyperkalemia
- There may be involvement with left bundle branch block (LBBB) , more common compared to RBBB as a marker of conduction delay. Hyperkalemia causes generalized conduction slowing, but the anatomical complexity, higher sodium-channel dependence, and greater functional demand of the left bundle make it more likely to show this block earlier and more obviously than the right bundle.
- The association of Hyperkalemia with acute myocardial infarction exposes the patient to increased mortality.

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