CLINICAL CHALLENGE: CASE 1

Challenge your diagnostic acumen: Study the following x-rays, electrocardiograms, and photographs and consider what your diagnosis might be in each case. While the images presented here are authentic, the patient cases are hypothetical. Readers are welcome to offer their own patient cases and images for consideration by contacting the editors at editor@jucm.com.

42-Year-Old Soccer Player With Knee Injury

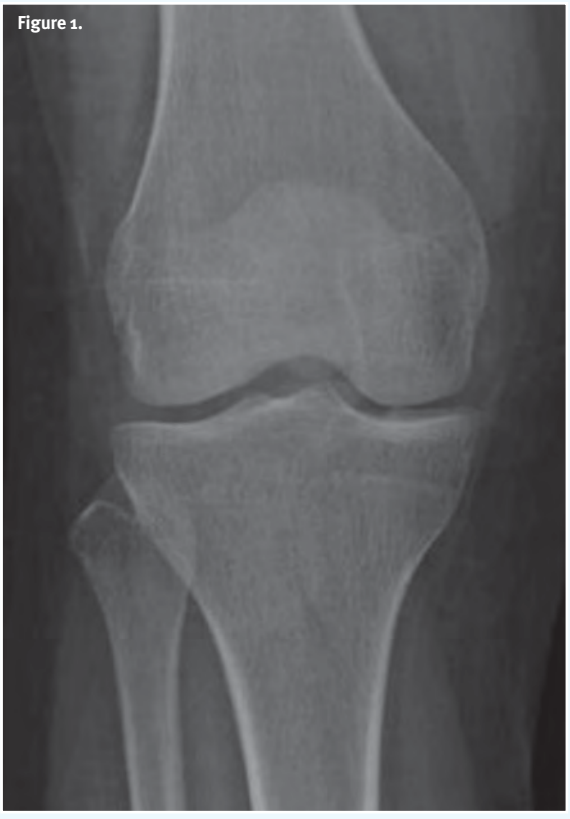


Figure 1.

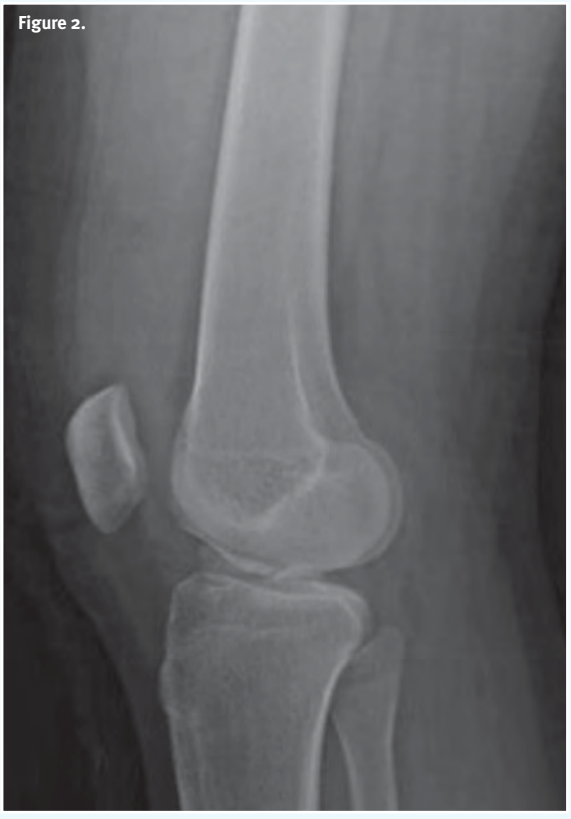


Figure 2.

A 42-year-old man presents to urgent care with knee pain. He says the pain started when he was playing soccer, just as he kicked a ball toward the goal. Ultimately, he had to leave the game because of the pain.

View the image taken and consider what your diagnosis and next steps would be. Resolution of the case is described on the following page.

Acknowledgment: Images and case provided by Experity Teleradiology (www.experityhealth.com/teleradiology).

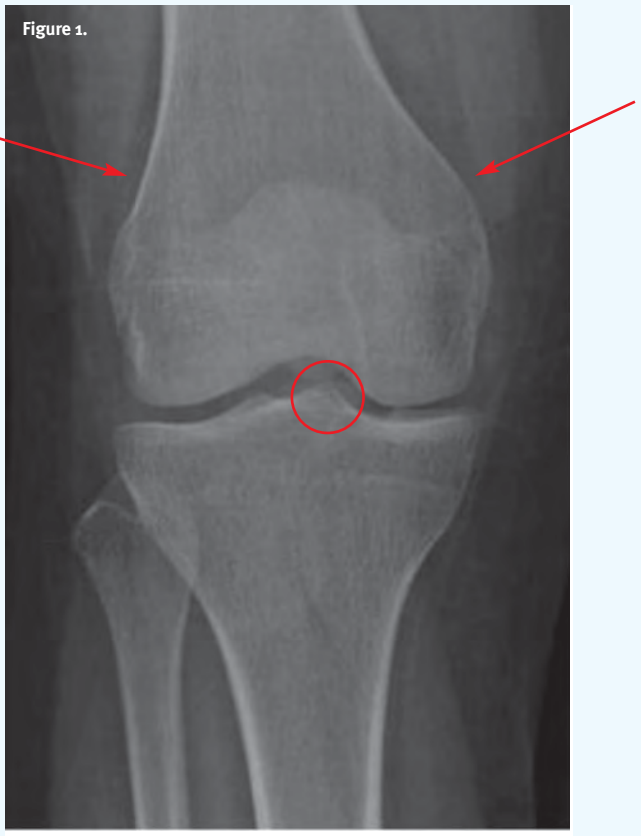


Figure 1.

Differential Diagnosis
- Posterior cruciate ligament (PCL) tear
- Anterior cruciate ligament (ACL) tear
- Tibial plateau fracture
- Patellar tendon tear

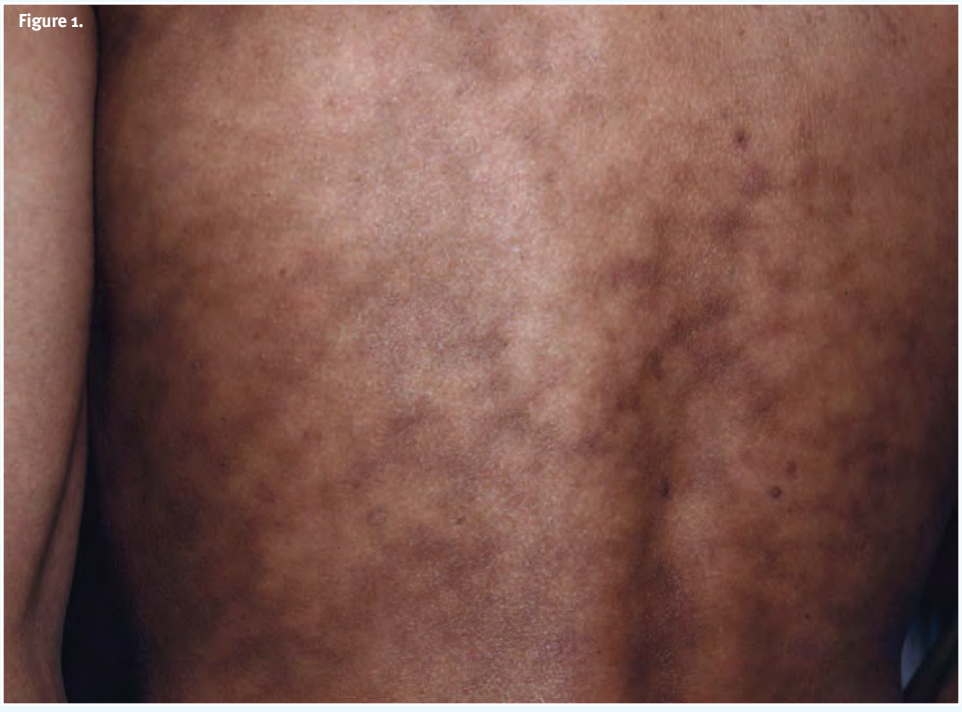
Diagnosis
The correct diagnosis is an ACL tear. In the anterior-to-posterior view, tibial eminence avulsion fracture (circle) overlying tibial spines and large effusion (arrow) can be seen. ACL tears are usually caused by forceful hyperextension of the knee or by a direct blow over the distal end of the femur with the knee flexed. Such an injury typically involves separation of the tibial attachment of the ACL to variable degrees, while separation at the femoral attachment is rare.

What to Look For
- On exam, there is frequently a large knee joint effusion and initially patient is unable to bear weight
- On exam, a positive Lachman's test or anterior drawer test is present

Pearls for Urgent Care Management
- On exam, ensure that all other ligaments are intact, including the PCL
- MRI is the imaging modality of choice to confirm diagnosis
- Initial treatment is rest, ice, compression, elevation, pain management and non-weight bearing if knee is unstable
- Referral to orthopedic surgery is indicated for further consideration of operative versus non-operative management

38-Year-Old With Rash After Heating Pad Use

Figure 1.



A 38-year-old woman presented to urgent care for rash that had developed on her trunk 2 months prior. On examination, extensive hyperpigmented, reticulated patches were seen on her back. She had no recent history of sunburn, extensive sun exposure, or history of dermatological conditions. Additional history revealed the use of a heating pad for the preceding 3 months to help manage her chronic back pain. She often rested on the electric heating pad for several hours and sometimes fell asleep with the heating pad on.

View the image above and consider what your diagnosis and next steps would be. Resolution of the case is described on the following page.

Acknowledgment: Image and case presented by VisualDx (www.VisualDx.com/jucm).

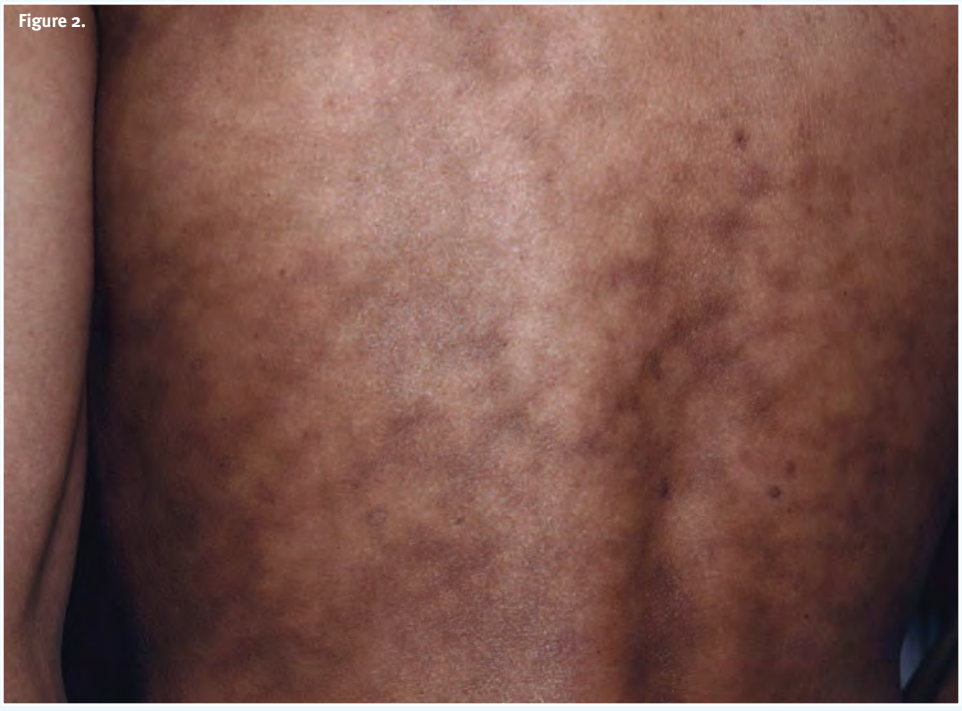


Figure 2.

Differential Diagnosis
- Intravascular large B cell lymphoma
- Erythema ab igne
- Livedo reticularis
- Polyarteritis nodosa

Diagnosis

The correct diagnosis in this case is erythema ab igne (EAI), a disorder of hyperpigmentation caused by prolonged exposure to heat. Heat exposure produces cutaneous hyperthermia, which in turn results in histopathologic changes similar to those seen in sun-damaged skin. Patients who report long-term use of heating pads or electric blankets may experience EAI. Heat exposure from a fireplace or even a laptop computer can also trigger the disorder.

What to Look For
- The rash appears as mottled, reticulated, pink, reddish, or violaceous patches that eventually become brown from melanin deposition
- While usually asymptomatic, there may be pruritus or mild burning paresthesias
- Rarely, vesicles or bullae have been described in affected areas

Pearls for Urgent Care Management
- Treatment is removal of the heat source
- Skin changes usually clear without further intervention in weeks to months, however, some may become permanent

CLINICAL CHALLENGE: CASE 3

55-Year-Old Male With Dyspnea

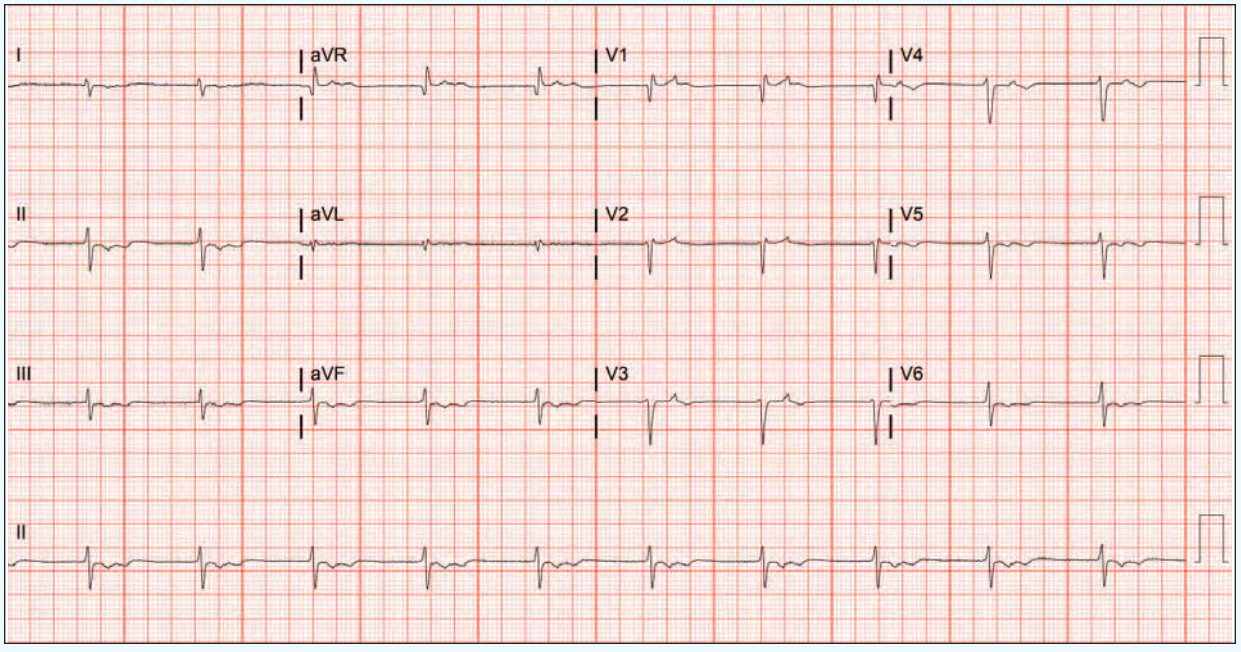


Figure 1: Initial ECG

A 55-year-old male with a history of heart failure presents to the urgent care with dyspnea. An ECG is obtained.

View the ECG captured above and consider what your diagnosis and next steps would be. Resolution of the case is described on the next page.

Case presented by Benjamin Cooper, MD, McGovern Medical School, The University of Texas Health Science Center at Houston, Department of Emergency Medicine

Case courtesy of ECG Stampede (www.ecgstampede.com).

ECG STAMPEDE

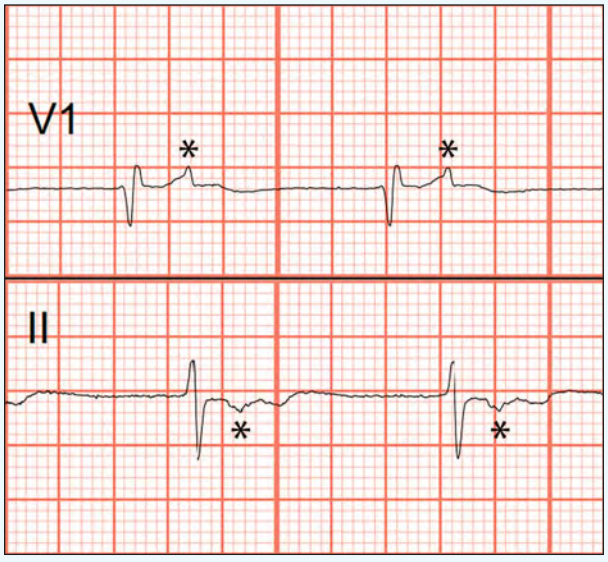


Figure 2. Retrograde p' waves in leads V1 and II (asterisks).

Differential Diagnosis

- Sinus bradycardia
- Junctional escape rhythm
- Complete heart block
- Hyperkalemia

Diagnosis

The diagnosis is junctional escape rhythm. The ECG reveals narrow escape complexes at a rate of 60 beats per minute. Retrograde p' waves are visualized immediately following the QRS complexes (**Figure 2**). Pacemaking cells exist throughout the conduction system, from the atrioventricular node through the distal Purkinje fibers. Inferior pacemakers are suppressed by the most superior (and dominant) one—usually the sinoatrial node. When impulses from the dominant pacemaker fail to conduct distally or an ancillary pacemaker outpaces and usurps control, an "escape" rhythm results.[1] When a superior pacemaker fails to generate impulses at a rate faster than an inferior one, then the faster, more inferior, one will "escape." In our case, the sinus node failed to generate a rate that outpaced the junction, which led to a junctional escape rhythm.

The junction refers to the part of the conduction system involving the atrioventricular node and the proximal His bundle (immediately inferior to the atrioventricular node). It is the only part of the conduction system capable of producing a narrow QRS complex, provided that the bundles remain intact. Junctional escape rhythms generally produce a rate of 40 to 60 beats per minute, whereas more distal ventricular pacemakers are slower and less reliable.[2]

Normally, the atria depolarize from superior to inferior given the superior location of the sinoatrial node in the right atrium. The normal wave of atrial depolarization creates a positive deflection in the inferior leads (ie, II, III, and aVF), and a negative deflection in aVR—the typical morphology of P waves when the impulse originates from the sinoatrial node. Sometimes, when the impulse originates from the junction (or below), the atria will depolarize in the opposite direction (ie, from inferior to superior), and retrograde p' waves can be seen immediately following the QRS complexes. Retrograde p' waves are negatively deflected in the inferior leads (ie, II, III, and aVF) and positively deflected in aVR and V1 (**Figure 2**). Our patients ECG lacks typical P waves, indicating that the sinoatrial node failed to outpace the junction and resulting in a junctional escape rhythm with retrograde p' waves. In the setting of preexisting heart failure, the decreased cardiac output associated with acute (relative) bradycardia can become symptomatic, as with this patient. Immediate transfer to an electrophysiology-capable facility is indicated.

There is no evidence of complete heart block given the lack of observed sinoatrial activity. While hyperkalemia is possible, there are no features to suggest that is the case (eg, peaked T waves or QRS widening).

What To Look For

- Junctional escape rhythms are narrow, and typically produce rates between 40 and 60 beats per minute.
- Retrograde p' waves can be seen immediately following the QRS complexes, are upright in aVR and V1, and negatively deflected in the inferior leads.

Pearls For Initial Management And Considerations For Transfer

- Patients with symptomatic bradycardia warrant transfer to an electrophysiology-capable facility.
- If unstable, consider transcutaneous pacing.

References

1. Mattu A, Tabas J, Brady W. *Electrocardiography in Emergency, Acute, and Critical Care*. 2nd ed. The American College of Emergency Physicians; 2019.
2. Wagner GS, Strauss DG. *Marriott's Practical Electrocardiography*. 12th ed. Lippincott Williams & Wilkins; 2014.

CLINICAL CHALLENGE: CASE 4

53-Year-Old Female With Fatigue

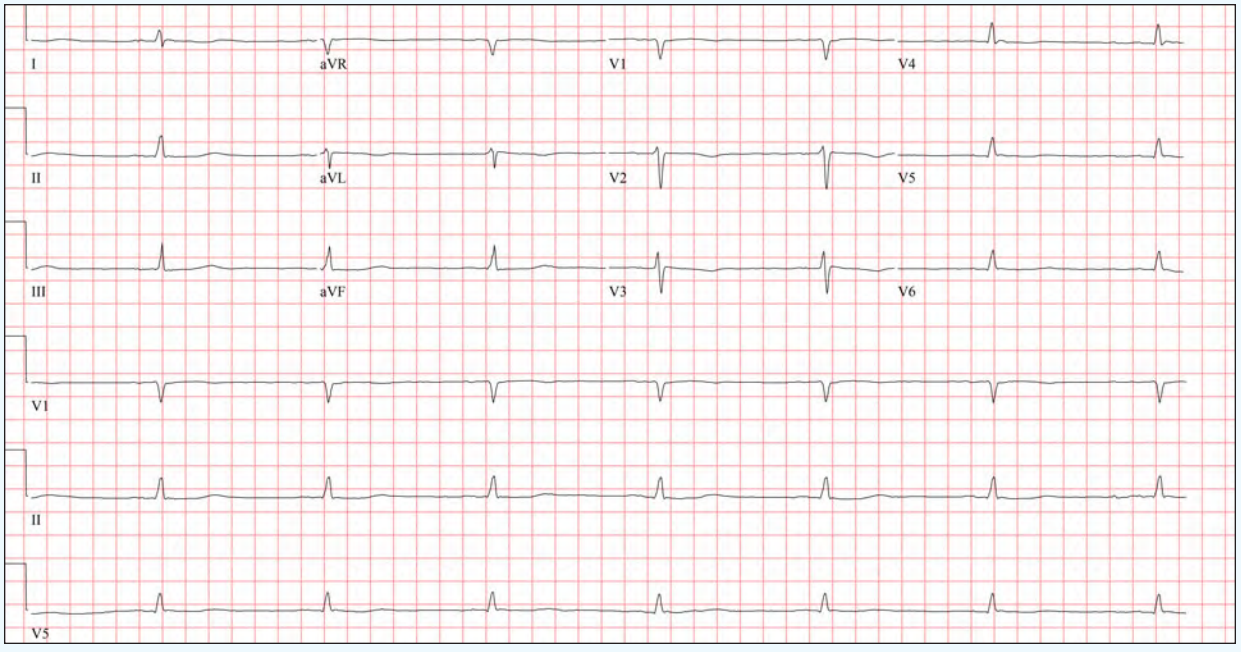


Figure 1: Initial ECG

A 53-year-old female with no significant past medical history presents to the urgent care with progressive fatigue for several weeks. An ECG is obtained.

View the ECG captured above and consider what your diagnosis and next steps would be. Resolution of the case is described on the next page.

Case presented by Catherine Reynolds, MD, McGovern Medical School at UTHealth Houston.

Case courtesy of ECG Stampede (www.ecgstampede.com).

ECG STAMPEDE

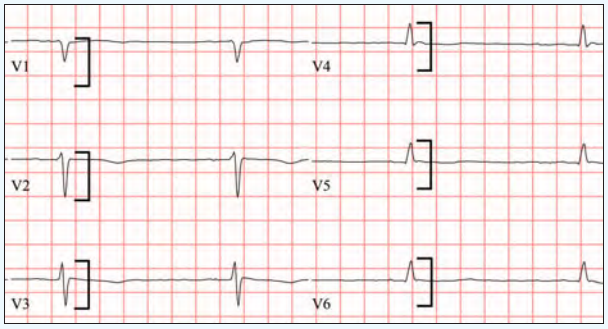


Figure 2: 10 mm of amplitude as depicted by the closed brackets

Differential Diagnosis

- Sinus bradycardia
- Myxedema coma
- Pericardial effusion
- Complete heart block
- Hypokalemia

Diagnosis

The diagnosis is myxedema coma. The ECG reveals sinus bradycardia with a rate of 40 beats per minute and low voltage. Atrial activity is difficult to see but careful analysis reveals blunted P waves. There is also a prolonged QT interval. The traditional definition of low voltage is an amplitude less than 5 mm in the QRS complexes of all limb leads or an amplitude of less than 10 mm in the QRS complexes of all precordial leads.[1] This patient had a QRS amplitude of less than 10 mm in all precordial lead (**Figure 2**). The differential for low voltage can be broken down into two categories: cardiac abnormalities resulting in diminished impulse generation; and increased impedance due to attenuating substances between the heart and the surface leads (**Table 1**).

When low voltage is encountered, the provider must consider the listed conditions in the differential diagnosis. Many causes can be ruled out with a comprehensive physical examination (eg, obesity, peripheral edema) and ancillary testing as needed (eg, COPD, pneumothorax), or bedside ultrasound when available (eg, pericardial effusion). Other conditions, such as hypothyroidism, may require additional testing. The combination of low voltage and bradycardia should raise the concern for hypothyroidism/myxedema.[3] Concern for severe hypothyroidism or myxedema should trigger additional laboratory testing and/or transfer to higher level of care. Interestingly, hypothyroidism can cause low voltage through two mechanisms: the direct effects of hormonal deficiency on the generation of cardiac action potentials and via the presence of a pericardial effusion, seen in up to one-third of patients with hypothyroidism and, rarely, leading to tamponade.[3,4] Patients with tamponade or large effusions due to hypothyroidism will characteristically lack a compensatory tachycardic response and will be bradycardic or normocardic.[5,6] Other electrocardiographic findings of hypothyroidism include sinus bradycardia, low voltage, and prolonged QT interval. Occasionally, dysrhythmias like torsade de pointes can result.[3,7] The presence of conducted P waves precludes the diagnosis of complete heart block. While hypokalemia can cause QT prolongation, it is not known to cause low voltage or bradycardia.

Table 1. Causes of Low Voltage

Increased Impedance
Pericardial
Effusion
Constrictive pericarditis
Pneumopericardium
Thoracic
Intra-pleural
Pneumothorax
Pleural effusion
Pulmonary
COPD
Pulmonary edema
Mediastinum
Pneumomediastinum
Soft tissue
Peripheral edema
Obesity
Decreased Impulse Generation
Prior myocardial infarction
Infiltrative cardiomyopathy (amyloidosis, sarcoidosis)
Myocarditis
Hypothyroidism

What To Look For

- Low voltage is caused by decreased impulse generation or increased impedance due to extracardiac causes like pericardial effusion, COPD, or obesity.
- Consider all causes of low voltage.
- In patients with bradycardia and low voltage, consider hypothyroidism/myxedema coma.

Pearls For Management, Considerations For Transfer

- Patients with severe hypothyroidism require transfer for admission, typically to an ICU.
- Be aware that hemodynamic instability could be caused by cardiac tamponade in patients with hypothyroidism, in which case fluid administration while preparing for immediate transfer is indicated.

References

1. Madias JE. Low QRS voltage and its causes. *J Electrocardiol*. 2008;41(6):498.
2. Cooper BL, Giordano JA, Fadial TT, Reynolds CE. *ECG Stampede: A Case-Based Curriculum in Electrocardiography Triage*. 1st ed. (Cooper BL, ed.). Null Publishing Group; 2021.
3. Danzi S, Klein I. Thyroid disease and the cardiovascular system. *Endocrinol Metab Clin North Am*. 2014;43(2):517-528. doi:10.1016/j.ecl.2014.02.005
4. Tajiri J, Morita M, Higashi K, Sato T, Fujii H, Nakamura N. The cause of low voltage qrs complex in primary hypothyroidism pericardial effusion or thyroid hormone deficiency? *Jpn Heart J*. 1985;26(4):539-547. doi:10.1536/ihj.26.539
5. Wang JL, Hsieh MJ, Lee CH, et al. Hypothyroid cardiac tamponade: Clinical features, electrocardiography, pericardial fluid and management. *American Journal of the Medical Sciences*. 2010;340(4):276-281.6. Cooper BL, Ducach GJ, Fadial TT. Low and Slow. *Ann Emerg Med*. 2021;77(6):601-603. doi:10.1016/J.ANNEMERGMED.2020.12.010
7. Wei Mak W, Nurazni Raja Azwan R, Badrulnizam Long Bidin M. Severe hypothyroidism presenting with supraventricular tachycardia. *Med J Malasia*. 2018;73(5):349-350.