Editor's Note: While the images presented here are authentic, the patient cases are hypothetical.

79-Year-Old Female With Acute Back Pain

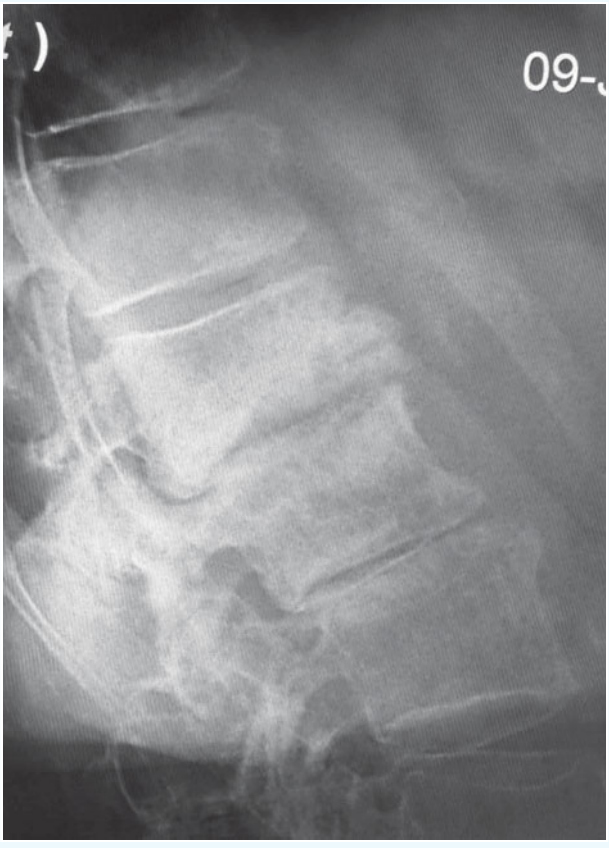
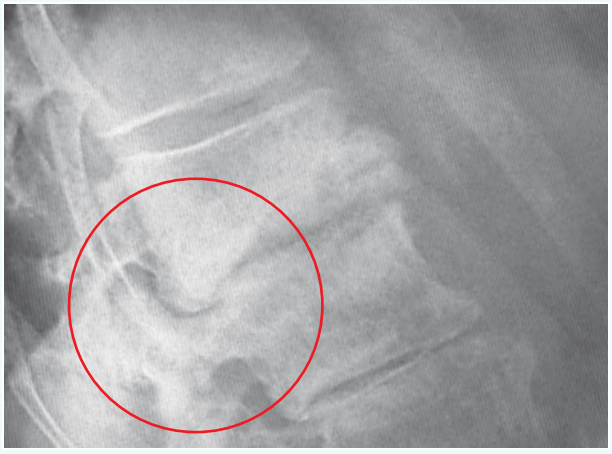


Figure 1: Initial X-ray

A 79-year-old female presents to urgent care complaining of sharp, severe lower back pain for the past 2 months. The pain began when she was bending over to pick something up from the floor and has not improved. The pain occasionally radiates to her flank area but not down her legs, and she denies any bowel or bladder pattern changes. She tried acetaminophen, cold and warm compresses, and rest without any relief. The pain worsens when she sits for long periods of time with slight improvement with rest. Her main complaint is persistent difficulty with bending, lifting, reaching, walking downstairs, and cooking. Physical examination reveals normal vital signs and bony tenderness to her lumbar spine spinous processes, most notably to L1 – L2. Neurologic exam shows normal sensation and 4/5 strength to all extremities.

View the x-ray images 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).



Differential Diagnosis

- Acute discogenic pain
- Vertebral compression fracture
- Soft tissue back injury
- Cauda equina syndrome
- Spondylolisthesis exacerbation

Diagnosis

The radiographic findings include an anterior wedge compression deformity of L1 with loss of anterior vertebral body height, consistent with a vertebral compression fracture. It also shows diffuse osteopenia and intense sclerosis with irregularity of the inferior endplate of L1, which raises concern for pathologic fracture. Multilevel degenerative changes are also visible including disc space narrowing, anterior osteophyte formation, and possible grade I degenerative spondylolisthesis at L4-L5.

Osteoporotic fractures are those occurring from a low-impact injury, such as a fall from a standing height or less. Vertebral compression fractures are the most common type of osteoporotic fracture and most often occur at the mid-thoracic (T7-T8) spine and the thoracolumbar junction (T12-L1). These types of fractures can cause significant back pain, limit physical functioning and activities of daily living, and lead to loss of independence, depression, and chronic pain. A history of an osteoporotic fracture is the most important risk factor for subsequent fractures; approximately 19% of patients who have a vertebral compression fracture will have another fracture in the next year.

What to Look For

- Neurologic abnormalities (such as dermatomal sensory deficits, focal weakness, or clonus) may indicate the presence of bone fragments in the spinal canal or foramina, which may require urgent surgical intervention.
- Ask about changes in height. Each vertebral compression fracture causes approximately ≥1 cm loss in height; kyphosis may be an indicator of multiple past vertebral compression fractures.
- In any patient presenting with fever or other signs of infection, a complete blood count and blood cultures should be obtained immediately.

Pearls For Urgent Care Management

- In patients with severe pain and/or suspected fracture-related complications, prompt referral to the emergency department for magnetic resonance imaging is indicated.
- Initial management of osteoporotic vertebral compression fractures should include pain control and activity modification. Patients with mild to moderate pain can be treated initially with nonopioid oral analgesics, considering risks of nonsteroidal anti-inflammatory drug use in older patients.
- Intranasal calcitonin has been shown to hasten the relief of pain from osteoporotic vertebral fracture when initiated early.
- Patients with a tolerable pain level should be encouraged to resume physical activity as quickly as possible. Complete bed rest is not recommended and can lead to further bone loss and deconditioning. Physical therapy and aquatic therapy are recommended for strengthening and pain management.
- Patient Education: fractures may take up to 3 months to heal, and pain and activity intolerance should diminish gradually.
- Bracing and muscle relaxants are not recommended.
- Encourage follow-up with primary care provider for long-term evaluation and management of osteoporosis.
- Consider referral to orthopedic and/or spine specialist, as patients who continue to have refractory pain may sometimes be candidates for surgical interventions for vertebral fractures. ■

8-Year-Old Male with Facial Rash

An 8-year-old male is brought to urgent care by his mother reporting a widespread, itchy facial rash. The patient says that the rash is severely painful and pruritic, and that he feels tingling and pain in his lips. The facial rash presented 3 days prior, and the mother states that her son repeatedly scratches the lesions. The patient and his family are from Honduras; they deny any recent travel. The patient is otherwise well and denies any systemic symptoms. No treatments were tried for symptoms at home. Physical exam reveals normal vitals, and widespread scaly, eroded, and crusted plaques are visible on the child's face and upper lip.

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

Differential Diagnosis

- Arthropod bites
- Excoriated eczema
- Actinic prurigo
- Polymorphous light eruption
- Prurigo nodularis
- Seborrheic dermatitis
- Solar urticaria

Diagnosis

The correct diagnosis is actinic prurigo (AP), also called Hutchinson's summer prurigo, which is a rare, photo-induced skin eruption triggered by exposure to ultraviolet (UV) radiation. AP is a delayed-type hypersensitivity reaction, although its precise pathogenesis remains unclear. Both UVA and UVB radiation have been implicated in triggering symptoms. AP is most frequently seen in indigenous populations of North, Central, and South America and usually first presents during childhood. It is more common in females, although studies from Asia report a male predominance in adult-onset disease.

The diagnosis of AP is often based on history and clinical findings. Up to 50% of patients report a family history of similar eruptions. Additional evaluation may include biopsy, phototesting and laboratory testing including serum immunoglobulin E (IgE), which is nearly always elevated in moderate to severe cases.

What To Look For

- The hallmark symptoms of AP are symmetrically distributed excoriated, pruriginous papules and nodules in photo-exposed areas (especially the face) which are intensely pruritic. Hemorrhagic crusts, eczematous patches, lichenification and/or scarring may also be present.
- Many patients also experience perioral tingling and pain, conjunctivitis, photophobia, and increased lacrimation.
- The onset of AP typically occurs in the spring, when more time is spent outdoors and more skin is exposed; the lesions can persist through the summer.
- AP may have a protracted course that extends into the winter months. Patients with childhood onset may spontaneously improve in adolescence, but the condition can persist into adulthood, with a major impact on the patient's quality of life.

Pearls for Urgent Care Management

- Sun-avoidance and sun-protective practices, including broad-spectrum sunscreens and protective clothing, are the mainstay of the management of AP.
- Mild to moderate disease: Topical corticosteroids and topical calcineurin inhibitors can offer symptomatic relief in mild cases.
- Severe disease: Patients with severe AP require treatment with systemic immunomodulators, immunosuppressants or phototherapy, and should be referred to primary care or dermatology specialists. ■

64-Year-Old With Acute Posterior Ankle Pain

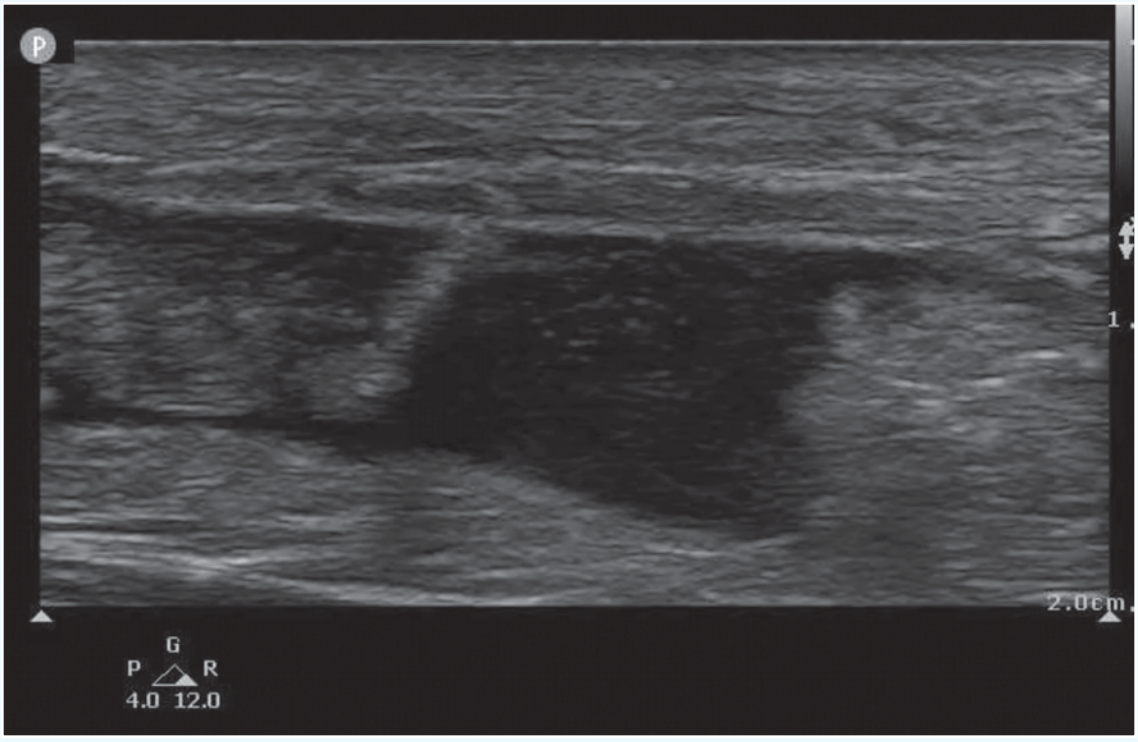


Image 1

A 64-year-old woman with a history of chronic obstructive pulmonary disease, diabetes mellitus, osteoarthritis, and obesity presents to urgent care with acute right posterior ankle pain after stumbling on a curb earlier that day. She says she felt a sudden sharp pain in the back of her ankle and experienced immediate difficulty walking and climbing stairs. She reports 10 days of mild posterior ankle discomfort before this event, which she treated with ice, an elastic wrap, and intermittent crutch use. Approximately 2 weeks prior to presentation, she was treated with levofloxacin for a urinary tract infection.

On examination, there is ecchymosis along the posterior right ankle extending toward the lateral foot. She has tenderness over the distal calf, with only mild tenderness over the foot and bilateral malleoli. She is unable to actively plantarflex her foot or perform a single-leg heel raise. The Thompson test is inconclusive. Point-of-care ultrasound (POCUS) of the posterior right ankle is performed.

View the image above and consider the likely diagnosis and next steps. The resolution of the case is described on the following page.

Acknowledgment: Case presented by Tatiana Havryliuk, MD, an emergency physician based in New York, New York, and the founder of Hello Sono. Case and images provided courtesy of Hello Sono (www.hellosono.com).

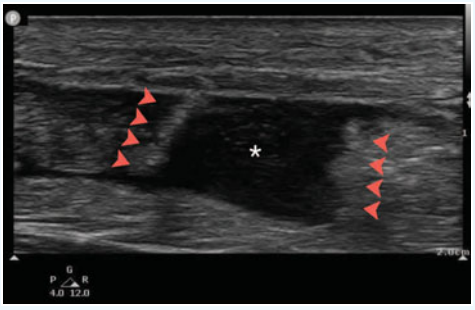


Image 2

Differential Diagnosis

- Ankle sprain
- Calf muscle injury
- Complete Achilles tendon rupture
- Partial Achilles tendon tear
- Achilles tendinopathy
- Deep vein thrombosis
- Calcaneal avulsion fracture
- Retrocalcaneal bursitis

Diagnosis

The correct diagnosis in this case is complete Achilles tendon rupture. This classically occurs after a sudden forceful push-off, often during sports, with acute posterior ankle or calf pain and an audible or palpable "pop." However, presentations may be subtle, particularly when patients have preceding tendinopathy or subacute tendon pain before complete rupture. Misdiagnosis of an ankle sprain or calf strain may occur when pain partially improves or patients retain some plantarflexion through accessory muscles.

Discussion

This case highlights an important urgent care clinical pearl: Achilles tendon rupture should remain on the differential for posterior ankle pain or ecchymosis, loss of plantarflexion, or inability to perform a single-leg heel raise, even when the Thompson test is equivocal. Recent fluoroquinolone exposure further increases clinical suspicion. Other reported risk factors include steroid use, oral bisphosphonate use, male sex, obesity, diabetes mellitus, chronic kidney disease, hyperparathyroidism, and intermittent athletic activity in deconditioned individuals.[1]

POCUS can rapidly confirm the diagnosis at the bedside. A high-frequency linear transducer should be used to evaluate the Achilles tendon in long- and short-axis views, scanning from the myotendinous junction to the calcaneal insertion. A normal tendon appears as a continuous fibrillar band, whereas a complete rupture demonstrates loss of fibrillar continuity, tendon retraction (arrowheads in image above), and a fluid-filled gap (asterisk). For complete Achilles tendon rupture, ultrasound has a reported sensitivity of 94.8% and specificity of 98.7%.[2]

Partial tears are more challenging and may require comparison with the contralateral side or advanced imaging.[3] Dynamic assessment with a real-time Achilles ultrasound Thompson test may also support the diagnosis when differentiating partial from complete rupture.[4] The retrocalcaneal bursa should not be mistaken for a tendon rupture, as it may contain fluid in cases of bursitis.

Once a complete rupture is identified, or when clinical suspicion remains high despite an equivocal study, the patient should be splinted in plantarflexion, kept non-weight-bearing, and referred urgently for orthopedic evaluation.

What To Look For

- In complete tears, the tendon defect is usually readily apparent, with discontinuity of the Achilles tendon fibers and retraction of the tendon ends.
- Look for anechoic or hypoechoic fluid within the defect, consistent with hemorrhage.

Pearls For Urgent Care Management

- POCUS is easy to perform and highly sensitive for complete Achilles tendon ruptures, helping to quickly make the diagnosis.
- Scan the entire tendon in both the long- and short-axis views.
- Partial tears are more subtle. Compare with the contralateral tendon and use dynamic maneuvers to improve diagnostic accuracy.
- Patients with current or recent fluoroquinolone, steroids or oral bisphosphonate use are at increased risk for Achilles tendon rupture.

References

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CLINICAL IMAGE CHALLENGE
ECG

83-Year-Old Female With Palpitations

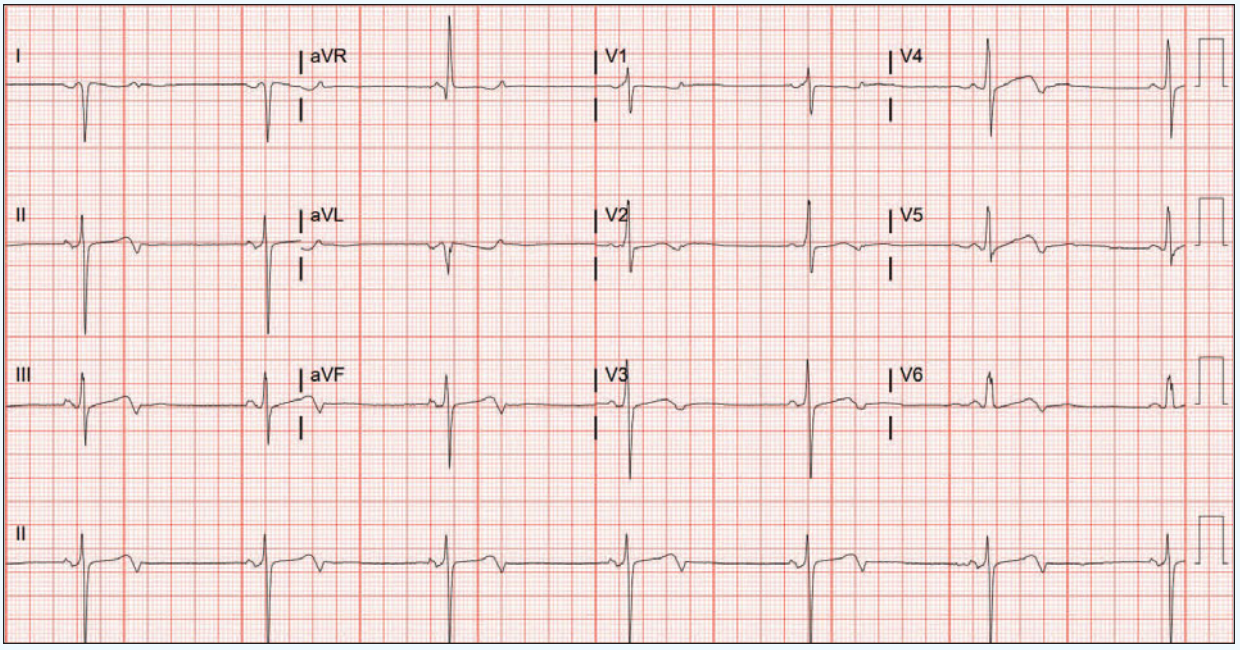

Figure 1: Initial ECG

An 83-year-old female with a past medical history of hypertension, diabetes mellitus, and hyperlipidemia presents to urgent care with palpitations and dizziness for the past 2 weeks. She does not have any family members present. An ECG is obtained.

View the ECG 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

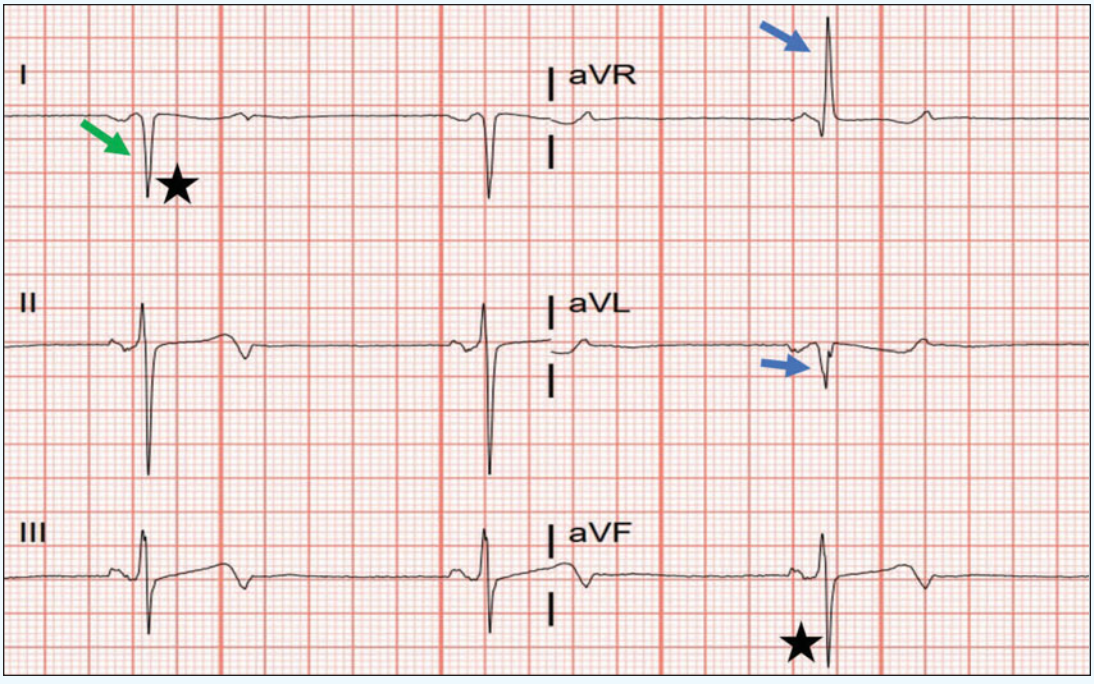

Figure 2: Inversion of lead I (green arrow), swapping of leads aVL and aVR (blue arrows), and marked right axis deviation with negative QRS complexes in lead I and aVF (stars).

Differential Diagnosis

- Acute coronary syndrome
- Left posterior fascicular block
- Dextrocardia
- Limb lead reversal
- Marked (extreme) right axis deviation

Discussion

The diagnosis in this case is limb lead reversal. The ECG shows a regular rhythm with visible P waves before each QRS complex, consistent with sinus rhythm. There are deep S waves and negatively deflected P waves in Lead I as well as a globally positive QRS complex in aVR. These findings should raise suspicion for a limb lead reversal, specifically right arm-left arm (RA–LA) reversal, which is the most common type of electrode misplacement.[1]

The ECG changes seen with RA–LA reversal can be understood as a 180° horizontal rotation of Einthoven's triangle around the aVF axis.[2] This results in inversion of lead I, swapping of aVR and aVL, an apparent marked right axis deviation, and swapping of leads II and III **(Figure 2).**[3,4] In addition, in limb lead reversal, the precordial leads typically retain normal morphology and R-wave progression, as chest lead placement remains unaffected. Electrode misplacement can lead to significant ECG misinterpretation and may result in unnecessary cardiac workup in patients without true pathology.[5]

A clinician may mistake this ECG for acute coronary syndrome given the T-wave inversions in leads I and aVL and the biphasic appearing T-waves in inferior and precordial leads. However, acute coronary syndrome would not cause global inversion of lead I along with a positive aVR. This pattern should alert the clinician to repeat the ECG and confirm proper lead placement prior to making an interpretation. Additionally, there are no acute ST elevations or ST depressions in contiguous leads, ruling out ST-segment elevation myocardial infarction.

Left posterior fascicular block may be considered, as the QRS complexes in leads I and aVL are negatively deflected, leading to the appearance of a marked right axis deviation. However, left posterior fascicular block does not cause the P wave in lead I to be inverted. Additionally, isolated left posterior fascicular block is rare in the general population, making this diagnosis less likely.[6]

RA-LA limb lead reversal can be mistaken for dextrocardia, as both can present with an inverted lead I and a positive aVR. However, the key distinguishing feature is the R-wave progression in the precordial leads. In dextrocardia, there is reversed R-wave progression, with leads V3-V6 showing predominantly negative QRS complexes.[7] In contrast, RA-LA limb lead reversal affects only the limb leads, so the precordial leads remain normal, as seen in this patient's ECG. This finding supports limb lead reversal as the correct diagnosis.

THE RESOLUTION

Marked (extreme) right axis deviation may be considered given the negative QRS complexes in lead I and aVF. However, limb lead reversals are known to mimic axis abnormalities and true marked right axis deviation does not cause global inversion of lead I or a positive aVR. Therefore, this ECG does not reflect true physiological axis deviation. Clinicians should consider repeating the ECG and confirm that all leads are placed in their proper position.

What To Look For

- The key ECG clues for RA-LA lead misplacement include positive P wave, QRS complex, and T wave in aVR, apparent marked right axis deviation, and global inversion of the P wave, QRS complex, and T wave in lead I.
- If limb lead reversal is suspected, repeat the ECG and confirm proper lead placement before making an interpretation.

Pearls For Initial Management, Considerations For Transfer

- Limb lead reversal can mimic myocardial infarction and other serious pathologies. Clinical judgement is essential, and providers should have a low threshold to repeat the ECG when findings do not match the clinical picture.
- If concern for acute coronary syndrome persists after correcting lead placement, then escalating transfer to an emergency department is imperative. Recognizing limb lead reversal can help avoid unnecessary workup and inappropriate transfers.

References

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