



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

28-Year-Old With Foot Pain



A 28-year-old woman presents to urgent care after an indoor rock-climbing event. She fell 10 feet from the wall without any safety gear to break the fall. She describes deep pain in her left foot, and she is limping. An exam reveals dorsal and plantar midfoot tenderness and bruising across the dorsal foot. A 3-view x-ray series is ordered.

Review the image 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 2.



Differential Diagnosis

- Fourth metatarsal fracture
- Lisfranc fracture dislocation
- Middle cuneiform fracture

Diagnosis

The correct diagnosis in this case is Lisfranc fracture dislocation. The anterior-posterior x-ray shows widening of the metatarsal M1-M2 and cuneiform (C) C1-M2 distance as well as a longitudinal fracture of cuboid bone. The oblique view shows a frayed appearing fracture at the base of M2, a possible longitudinal fracture at the base of M3, malalignment of the M2-C2 joint, and longitudinal cuboid fracture. This type of fracture is named after Jacques Lisfranc, a French field surgeon in Napoleon's army, who described a technique for amputation of the forefoot. However, the eponym is used today to describe fractures and dislocations that occur at the junction between the tarsal bones of the midfoot and the metatarsals of the forefoot.

What to Look For

- On x-ray, there are fractures and dislocations at the junctions between the tarsal bones of the midfoot and the metatarsals of the forefoot.
- Significant pain and swelling are usually present, and weight bearing is difficult.
- Neurovascular compromise is possible, so it is especially important to check the dorsalis pedis pulse and evaluate for acute compartment syndrome.

Pearls for Urgent Care Management

- Immobilization with short-leg splint or boot and non-weight bearing status
- Rest, ice, compression, and elevation
- Appropriate pain management
- If available, surgical referral is indicated to ensure healing



36-Year-Old With Hair Loss



A 36-year-old man presents to urgent care with asymptomatic hair loss that developed on multiple sites of his scalp over the previous 2 months. The hair loss developed in round patches that expanded to involve most of the back of his scalp. His past medical history is notable for hyperthyroidism.

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).

**Differential Diagnosis**

- Alopecia areata
- Alopecia mucinosa
- Pseudopelade
- Telogen effluvium

Diagnosis

The correct diagnosis in this case is alopecia areata—an autoimmune disease of the hair follicle resulting in non-scarring hair loss. Most cases are sudden onset and limited to 1 or 2 small patches of alopecia that involve the scalp, eyebrows, eyelashes, or body hair. In severe cases, all hair on the scalp is lost (alopecia totalis), or all scalp and body hair is lost (alopecia universalis). There is an increased incidence of alopecia areata in patients with Down syndrome and those with autoimmune diseases, most commonly thyroid disease.

What to Look For

- Asymptomatic nonscarring hair loss usually in broad confluent smooth circular patches
- Occasionally tingling, burning or pruritus is experienced prior to hair loss
- Exclamation point hairs (short broken hairs with narrow proximal end compared to distal end) are pathognomonic
- Nail abnormalities may also be present

Pearls for Urgent Care Management

- Educate the patient that the course of alopecia areata is unpredictable with wide variation and note that recurrences are common
- Psychosocial support is important
- For adults, recommended therapy is intralesional corticosteroid injections; topical corticosteroids are also recommended if injections are not feasible
- For pediatric patients, recommended therapy is topical corticosteroids



65-Year-Old With Hypertension

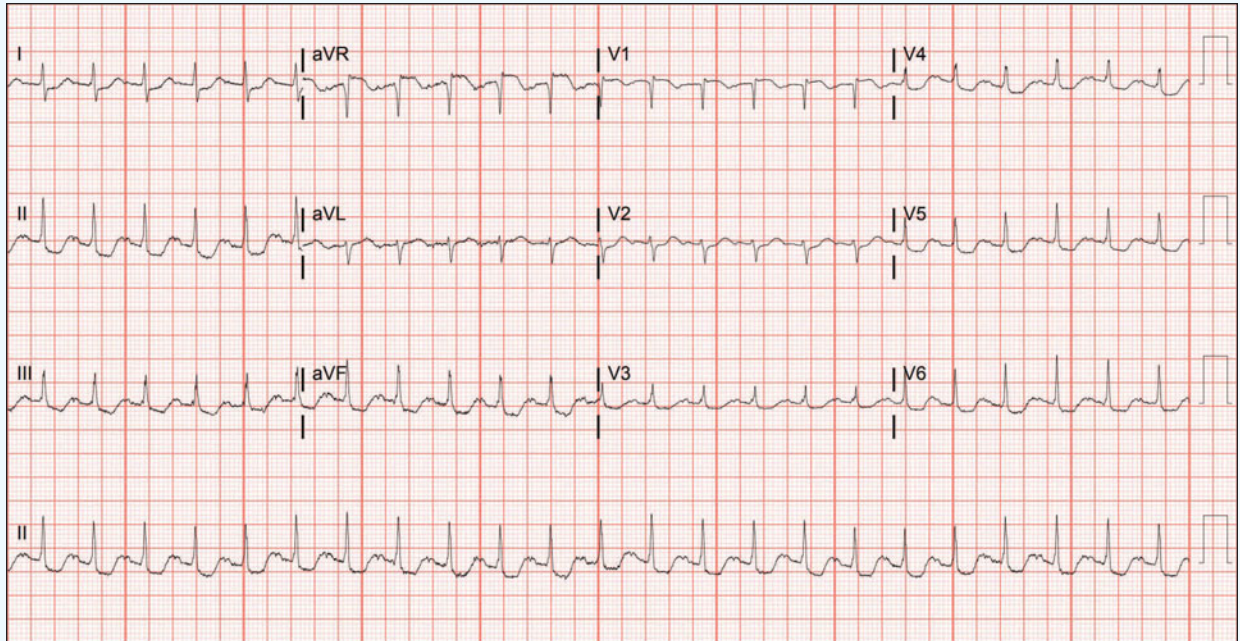


Figure 1: Initial ECG

A 65-year-old female with a history of hypertension presents with chest pain and dyspnea for 1 day. She denies fever, cough, nausea, vomiting, or abdominal pain. Upon arrival to urgent care, her respiratory rate was 28 breaths per minute, blood pressure 179/102, and oxygen saturation 92% on room air. 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

Differential Diagnosis

- ST-Elevation myocardial infarction (STEMI)
- Left ventricular hypertrophy (LVH) with strain
- Diffuse subendocardial ischemia
- Hypokalemia
- Supraventricular tachycardia

Diagnosis

The diagnosis in this case is diffuse subendocardial ischemia from submassive pulmonary embolism. The ECG shows a regular, narrow-complex, sinus rhythm with a rate of 138 beats per minute. There is a normal axis and normal intervals. There are diffuse ST-segment depressions in the inferolateral leads and ST-segment elevation in aVR (**Figure 2**).

This pattern of diffuse ST depression with ST elevation in aVR represents global subendocardial ischemia, and can be caused by many conditions, including left main coronary artery disease or multivessel disease.¹ Any condition with a supply/demand mismatch may have this pattern, including pulmonary embolism, severe anemia, hypoxia, tachydysrhythmias, and shock. Typically, the pattern on the ECG will reverse when the cause is resolved. When this electrocardiographic pattern is encountered, a broad differential should be considered. In a study of 142 ECGs with this pattern, only 27% were associated with acute coronary syndrome.² A targeted history and physical will help to determine the cause, as well as performing adjunct testing such as labs, imaging, or bedside ultrasound.

In this case, the patient was transferred to the emergency department, where she was given adenosine for suspected supraventricular tachycardia, which did not improve her symptoms or resolve the tachycardia. Additional workup discovered a submassive pulmonary embolism. These ECG findings were due to the supply/demand mismatch in the oxygenation of the myocardium caused by her pulmonary embolism.

What to Look For

- Global subendocardial ischemia can result from any disease process that creates a mismatch in the oxygen that the myocardium is requiring and the oxygen that the coronary arteries are supplying.
- When diffuse ST depressions with ST elevation in aVR is encountered, consider conditions like pulmonary embolism, severe anemia, hypoxia, and shock.

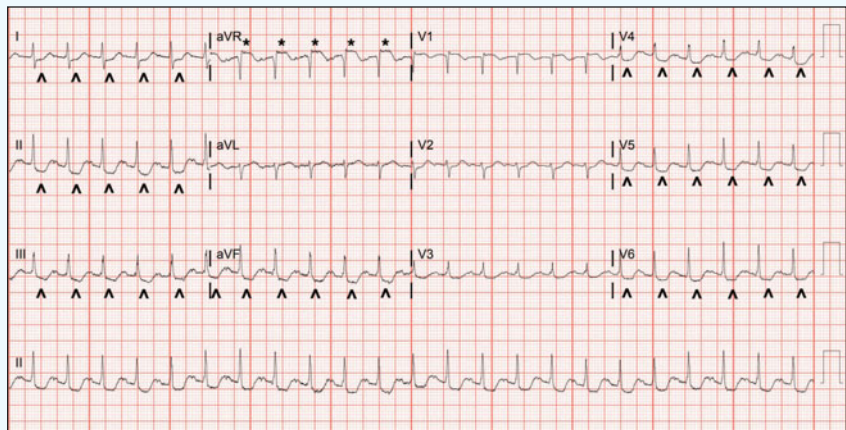


Figure 2: ST elevation in aVR (asterisks) and ST depressions in the inferolateral leads (arrows)

- This ECG pattern is often transient and will likely resolve when the cause is reversed.
- Other electrocardiographic findings of pulmonary embolism include sinus tachycardia, T-wave inversions (especially in anterior and inferior leads), right axis deviation, tall P wave in lead II, and the S1Q3T3 pattern.

Pearls for Management, Considerations for Transfer

- Always consider a broad differential with this ECG pattern and allow an in-depth history and physical guide your workup and next steps.
- Many causes of this pattern are life-threatening, and the urgent care physician should prepare for transfer.

References

1. Kosuge M, Ebina T, Hibi K, et al. An early and simple predictor of severe left main and/or three-vessel disease in patients with nonst-segment elevation acute coronary syndrome. *Am J Cardiol.* 2011;107(4):495-500. doi:10.1016/j.amjcard.2010.10.005
2. Knotts RJ, Wilson JM, Kim E, Huang HD, Birnbaum Y. Diffuse ST depression with ST elevation in aVR: Is this pattern specific for global ischemia due to left main coronary artery disease? *J Electrocardiol.* 2013;46(3):240-248. doi:10.1016/j.jelectrocard.2012.12.016