

CLINICAL CHALLENGE

This feature will challenge your diagnostic acumen with a glimpse of x-rays, electrocardiograms, and photographs of conditions that real urgent care patients have presented with.

If you would like to submit a case for consideration, please e-mail the relevant materials and presenting information to *editor@jucm.com*.



Man with forehead erythema

Case

An 80-year-old man woke up with a localized area of erythema on his forehead. He didn't think much of it, until the lesion slowly became white and then started turning blue over a few hours. He says that the lesion is painful and is getting larger and darker. He denies the presence of headaches, muscle pain, abdominal pain, sweating, and tremors.

INSIGHTS IN IMAGES: CLINICAL CHALLENGE

THE RESOLUTION

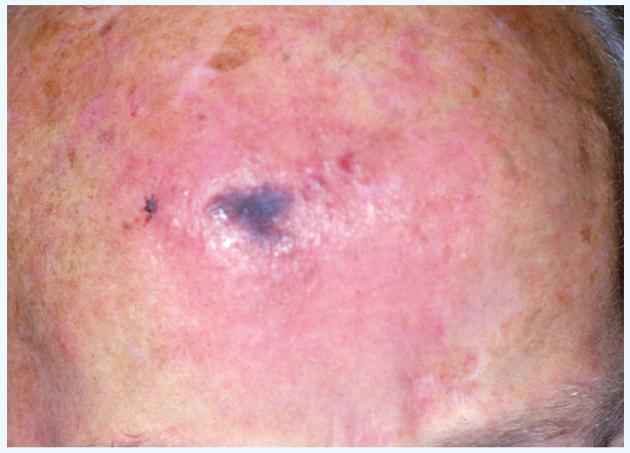


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DDx

Community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) infection Caterpillar envenomation Contact dermatitis Ecthyma Solar purpura

Learnings

Common spider bites usually present with erythema and edema. A necrotic or dusky center within a red, inflammatory plaque is characteristic.

In brown recluse spider bites, vesicles and bullae can present early. Between 12 and 24 hours after envenomation, a large plaque consisting of erythema, ischemia, and necrosis ("red, white, and blue" sign) develops. Later, these lesions can progress into painful, full-thickness necrotic plaques. Patients with black widow bites have local sweating, piloerection, redness, and mild edema. The systemic symptoms of muscle pain, cramps, abdominal pain, salivation, lacrimation, sweating, and tremors are more prominent than the skin findings.

If the victim brings in the spider, seek out a trained arachnologist or entomologist for accurate identification.

Diagnostic Pearls

Look closely for 2 small puncta, the fang marks of the spider.

Most suspected spider bites seen in the United States turn out to be the result of other causes, most commonly cellulitis or furunculosis caused by MRSA. Unless the spider has specifically been identified as the etiology of the symptoms, be cautious about narrowing your differential diagnosis and consider culturing for MRSA.



CLINICAL CHALLENGE



Man with unilateral decreased vision

Case

A 68-year-old man with a long history of uncontrolled hypertension and hyperlipidemia presented with unilateral decreased vision for the last 3 weeks. On exam, you saw a fundus with diffuse retinal hemorrhages and swelling.

INSIGHTS IN IMAGES: CLINICAL CHALLENGE

THE RESOLUTION



Image courtesy of Logical Images, Inc. www.VisualDx.com/JUCM

DDx

Diabetic retinopathy Ocular ischemic syndrome Hyperviscosity retinopathy Papilledema with hemorrhage

Learnings

Central retinal vein occlusion (CRVO) is a potentially blinding subacute vascular occlusion of the eye. The prevalence of CRVO is between 0.1% and 0.4% in individuals who aged 40 or older. Most patients have associated local or systemic disease, with systemic hypertension, diabetes mellitus, and open-angle glaucoma being the most common.

The central retinal artery and vein share a common adventitial sheath as they exit the optic nerve head and pass through the lamina cribrosa. Often in CRVO, a rigid atherosclerotic artery impinges on the nearby vein, causing turbulence and predisposing to thrombus formation. With increased resistance of venous flow, the retina becomes ischemic and fluid leaks out of the vessels. Increased intraocular pressure can also cause turbulence of the central retinal vein and lead to thrombus formation and obstruction. The occluded central vein can lead to intraretinal hemorrhage, exudation of fluid, varying levels of ischemia, and neovascular complications such as neovascular glaucoma. CRVO is commonly classified by severity into two forms: the nonischemic form of CRVO, which is milder and more common, and the ischemic form, which results in more severe retinal damage and vision loss. In ischemic CRVO, more than 90% of patients have vision of 20/400 or worse. One-third of patients with the nonischemic type progress to the ischemic type.

Iris neovascularization, which is associated with neovascular glaucoma, occurs in up to 60% of patients with the ischemic form, usually 3 to 5 months following the obstruction. The main risk factors for development of neovascular glaucoma after a CRVO are the extent of capillary nonperfusion, poor visual acuity, severe venous tortuosity, and retinal hemorrhage.

What to look for

The presenting visual acuity at the time of diagnosis of CRVO is the best predictor of visual prognosis. In a patient with CRVO, there is a 10% risk of developing a CRVO in the fellow eye, especially when there is an underlying systemic abnormality.