

# Photo Quiz: Unusual lumps to the lower leg with fat atrophy of the ankle

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**ANSWER: Erythema Nodosum (EN)**

## Discussion

Erythema nodosum (EN) is an acute, nodular, erythematous eruption primarily isolated to the lower legs. The condition is secondary to a primary disease and can be idiopathic in nature. EN is thought to be a delayed hypersensitivity to circulating antigens.<sup>1</sup> The most common primary causes of EN include streptococcal infections and other bacterial infections such as *Yersinia enterocolitica*, *Mycoplasma pneumoniae*, *Lymphogranuloma venereum* (LGV), *Salmonella* and *Campylobacter*. Fungal infections such as *coccidioidomycosis* (San Joaquin Valley Fever), *histoplasmosis* and *blastomycosis* can cause EN.<sup>1</sup> Systemic diseases such as *Sarcoidosis*, *TB*, *Leprosy*, *Behcet's syndrome*, *Crohn's disease* and *chronic ulcerative colitis* have been associated with EN eruption.<sup>1,2</sup>

In the eruptive phase of EN, malaise and fever are accompanied by acute nodular pain to the legs. This can be associated with general aches and myalgia. Arthralgia may occur and usually precedes the eruption or occurs during the eruptive phase.<sup>1</sup> In febrile illness with associated dermatologic findings, abrupt onset of illness with initial fever is followed by a painful rash within 1-2 days.<sup>1</sup> The lesions, depending of source of infection, can resolve within 7 weeks and may persist over 6 months.

The primary skin lesions include tender, red nodules below the knee and isolated primarily to the lower leg. The lesions can be poorly circumscribed and defined and range from 2 to 6 cm in diameter. As the lesions regress and absorb, the color gradually fades to a yellow hue resembling a bruise. As the lesions disappear, fat atrophy can occur and the overlying epidermal tissue will desquamate.<sup>1</sup>

This is a very interesting case because the presentation was so multifaceted. The patient first developed a lymphadenopathy. Why the patient developed acute groin adenopathy before EN is still in question. The patient has a complicated history of respiratory infection and it could be surmised that this may have been a result of respiratory infection. She had previously been hospitalized for respiratory infection prior to this episode. However, the lab values including WBC were within the normal range. Also, the fungal panel that included tests for *histoplasmosis*, *blastomyces* and *candida* was negative.

The patient began to take Cleocin 300mg, three times a day for 7 days and developed a significant skin rash with associated erythema nodosum. The EN was not initially diagnosed until the patient saw her gynecologist and dermatologist after two previous visits to the ER without improvement. She was subsequently placed on Naprosyn 250mg TID and Prednisone 20mg/day for two months. This eventually resolved the EN. What also made this case interesting was the fact that the patient developed fat atrophy and tissue induration as the nodules regressed. This has been well documented. This would likely explain the induration of tissue to both legs and the fat atrophy within Kager's triangle of the ankle.

A 'red herring' in the clinical presentation was the patient's inguinal hernia. This was purely incidental and could also be a result of the patient's high steroid use and tissue weakening along the inguinal canal. There is no doubt she had inguinal lymphadenopathy, which associated with the patient's COPD required testing for Lyme disease (i.e. lymphadenopathy and skin rash), Chlamydia trachomatis (groin and inguinal pain that can result from pelvic inflammatory disease or lymphogranuloma venereum (LGV) and fungal infection (i.e. patient history of chronic respiratory disease and infections).

The patient's BOOP posed clinical differentials that had to be ruled out before her diagnosis could be confirmed.

Erythema nodosum induced by Clindamycin does not appear to be reported in the literature. This may be the first case of Cleocin induced EN in a compromised patient, although it cannot be 100% confirmed in this case report. There have been reports of EN induced by other drugs that primarily include sulfonamides and halide agents.<sup>1</sup> A strong argument that Clindamycin caused EN in our patient is the fact that EN is commonly associated with hypersensitivity reactions caused by drugs, several systemic diseases and prolonged drug therapies. It also occurs in the panniculus adiposus or fatty layer of the skin.<sup>1</sup>

EN is considered a self limiting disease. EN responds well to NSAIDS producing an analgesic effect, anti-inflammatory and anti-pyretic properties. Other treatments have included Corticosteroids, Colchicine, and potassium iodide to relieve topical tenderness of the lesions.<sup>1</sup>

The other choices in this quiz included discoid lupus, fibromyalgia, histoplasmosis, Sarcoidosis, Scleroderma and Sjogren's disease. Discoid lupus is more confined to the face, scalp and hands and only occurs in patients with Systemic Lupus Erythematosus (SLE).<sup>3</sup> The lesions are scaly and psoriatic in nature. Fibromyalgia does not present with dermatologic nodules to the lower extremity, so this could immediately be ruled out. Histoplasmosis was ruled out through fungal culture. Sarcoidosis is a multisystem inflammatory disease predominantly affecting the lungs and intrathoracic lymph nodes. Sarcoid primarily presents with noncaseating granulomas (NCGs) affecting organ tissues.<sup>4</sup> When sarcoidosis is associated with EN, it is called Löfgren's syndrome. If Löfgren's syndrome is suspected this can be confirmed by mediastinal lymph node biopsy and the presence of bilateral hilar adenopathy demonstrating noncaseating granulomas.<sup>5</sup>

Scleroderma is characterized by indurated, hard nodules of soft tissue, hence the term *Sclerosis* or hard/indurated and *derma* or skin.<sup>6</sup> The ACR or American College of Rheumatology provides criteria for the classification of scleroderma. The classification is based on the fulfillment of one major criterion and two minor criterion. Major criterion includes proximal scleroderma as characterized by symmetric thickening, tightening, and induration of the skin of the fingers and the skin that is proximal to the metacarpophalangeal or metatarsophalangeal joints. These changes may affect the entire extremity, face, neck, and trunk.<sup>6</sup> Minor criterion include 1) sclerodactyly including a major criterion characteristic, but limited to only the fingers, 2) digital pitting scars or a loss of substance from the finger pad: As a result of ischemia, depressed areas of the fingertips or a loss of digital pad tissue occurs, and 3) basilar pulmonary fibrosis includes a bilateral reticular pattern of linear or lineonodular densities most pronounced in basilar portions of the lungs on standard chest roentgenograms. These densities may assume the appearance of diffuse mottling or a honeycomb lung and are not attributable to primary lung disease.<sup>6</sup>

Sjogren's syndrome is an autoimmune disease characterized by abnormal production of blood antibodies that are directed against normal tissues, particularly glands that produce water such as lacrimal and salivary glands. This causes a significant dryness of both the eyes and mouth.<sup>7</sup> This disease rarely causes dermatologic lesions to the legs.

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