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Ma. Paula Engedi M. Delmendo, MD Ann Margaret V. Chang, MD

Institute of Pathology St. Luke's Medical Center, Quezon City

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Nasal Gouty Tophus

A 48-year-old, non-hypertensive, non-diabetic man with uncontrolled gouty arthritis presented with a four-day swollen nasal mass. He was assessed to have a nasal abscess at the emergency room and was admitted for urgent management. Paranasal computed tomography (CT) scans showed a heterogeneously enhancing focus with areas of hypodensities in the nasal apex and dorsum extending into the right ala measuring 1.5 x 2.8 x 3.4 cm. with associated erosion of the cartilaginous part of the anterior nasal septum, soft tissue swelling and skin thickening in the nasal dorsum, nasal tip and right zygomatic region that was suspected to relate to an aggressive etiology. Tissue correlation was therefore recommended, and he underwent endoscopic-quided incision and drainage with biopsy and debridement of the nasal mass.

The specimen submitted consisted of red to white, irregular, soft tissue fragments with an aggregate measurement of $1.5 \times 1.5 \times 0.5$ cm. Microsections showed deposits of amorphous white to pink material with surrounding fibrosis and acute and chronic inflammatory cell infiltrates and foreign body giant cells. (*Figures 1 and 2*) Also seen in the background were fragments of sclerotic bone and bacterial colonies. These findings were consistent with gouty tophus with acute and chronic inflammation and bacterial colonization. The culture and sensitivity test of the nasal discharge showed growth of *Enterobacter aerogenes* (currently named *Klebsiella aerogenes*) which was identified by an automated mass spectrometry microbial identification system (VITEK[®] MS). Work-up also included uric acid levels which were within the reference interval at that time (6.57 mg/dL).

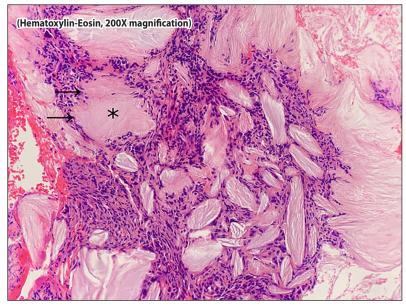


Figure 1. Haematoxylin and eosin stained slide showing nodular aggregates of acellular, amorphous, pale eosinophilic material (asterisk) surrounded by histiocytes in a palisading pattern (arrows) (200x)

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Correspondence: Dr. Ann Margaret V. Chang Institute of Pathology St. Luke's Medical Center 279 E Rodriguez Sr. Ave, Quezon City 1112 St. Luke's Medical Center Sth Ave, Taguig 1634 Philippines Phone: (632) 8723 0101 local 4149 Email: amychang@stlukes.com.ph

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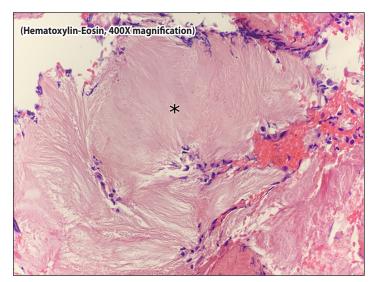


Figure 2. Haematoxylin and eosin-stained slide showing a higher magnification of the pale pink, amorphous (cotton candy-like) material (asterisk) (400x)

Gout is a disease that occurs in response to the presence of monosodium urate (MSU) crystals in joints, bones, and soft tissues and may result in one or a combination of acute arthritis (a gout flare), chronic arthritis (chronic gouty arthritis), and tophi (tophaceous gout).¹ Tophaceous gout typically occurs more than a decade after first presentation of gout in the context of untreated hyperuricemia but some patients present with gouty tophi earlier.² Typical sites for tophus deposition include the olecranon bursa, the Achilles tendon, the first metatarsophalangeal (MTP1) joint, the ear and the finger pulps.² Occurrence of gouty tophi in the nose is rare and only sixteen other case reports published in English literature were found from 1978 to 2022.^{3,4} Similar to this case, all the other published cases of nasal gouty tophi involved male patients with ages ranging from 33 to 69 years old.^{3,4}

A tophus is composed of three main zones, namely the central crystalline core of MSU crystals, the surrounding highly cellular corona zone and the outer fibrovascular zone.² The tissue reaction to a tophus is generally chronic inflammatory in type and involves both innate and adaptive immunity, thus, the coronal and fibrovascular zones both contain numerous CD68+ macrophages and plasma cells, fewer mast cells and scattered T and B lymphocytes.^{1,2} Although clinical signs of acute inflammation sometimes occur in tophi, this is unusual, and neutrophils are rarely observed.^{1,2} However, in the case of the patient, acute inflammatory cells were present because secondary infection already occurred. Infection is one of the complications of gout along with significant structural damage to surrounding tissues including cartilage and bone which is a late feature of this disease.^{2,5} A possible differential diagnosis for this case is tumoral calcinosis which has

amorphous or granular calcium crystals (hydroxyapatite crystals) with surrounding foreign body giant cell reaction.⁶ However, calcification was not appreciated in the specimen and the pale pink, amorphous (cotton-candy-like) material in the specimen is unlike that of the amorphous substance previously described in tumoral calcinosis. Although polarizing microscopy is better suited for differentiation between the two entities, the crystals are frequently lost in routine H&E-stained sections and a special method such as nonaqueous alcoholic eosin staining (NAES) method is required to demonstrate these.⁶ In addition, given the clinical history of uncontrolled gouty arthritis, the diagnosis of gouty tophus is favored. Since histiocytes surrounding the amorphous pink substances form a palisading granuloma in the specimen, possible differentials include a rheumatoid nodule and granuloma annulare which have necrobiotic granulomas characterized as having a central area of fibrinoid necrosis or degraded collagen, respectively, surrounded by palisading histiocytes and lymphocytes.7 However, given the clinical picture, these two entities are unlikely.

This case demonstrates occurrence of gouty tophi in an unusual location, the nose, with erosion of cartilage and secondary infection. It is important to include this entity in the differential diagnosis even when a mass presents in an unusual location especially in male patients with a history of uncontrolled gout.

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