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Peptostreptococcus anaerobius-Related Hepatic Abscess Containing Abundant Monosodium Urate Crystals in a Patient without Gout: Report of a Case

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Abstract

Hepatic abscesses can develop in association with biliary disease, through hematogenous spread via the portal circulation, by continuity with an intra-abdominal infection, as a post-surgical complication or sepsis, and can be either bacterial (pyogenic) or parasitic (usually amoebic). We present the case of an 80-year-old woman with sepsis and a hepatic abscess that on microbiologic culture, grew *Peptostreptococcus anaerobius* but with an unusual finding of abundant monosodium urate (MSU) crystals in the purulent material. There are case reports of gouty tophi presenting as liver nodules as a first manifestation of gout in patients with underlying hepatic disease. However, to the best of our knowledge, this is the first report of MSU crystals within a pyogenic hepatic abscess in a patient without gout. We review the characteristics of gouty tophi and compare them to those found in the abscess cavity, as well as conditions under which MSU crystals are formed.

Keywords

Hepatic Abscess, Anaerobic Bacteria, Purine Metabolism, Monosodium Urate Crystals, Gout

1. Introduction

Hepatic abscess is now considered a rare entity but requires a prompt diagnosis

given its potentially fatal outcome. It is defined as a cavity within the liver that is filled with purulent material and can be either bacterial (pyogenic) or parasitic (usually amoebic) in origin. Hepatic abscesses can commonly develop in association with biliary tract disease but also through hematogenous spread, by continuity with an intra-abdominal infection, with or without sepsis, or as a post-surgical complication [1]. Pyogenic abscesses are usually polymicrobial, but some organisms are more frequently isolated than others. These include: *E. coli*, *Klebsiella*, *Streptococcus*, *Staphylococcus*, and anaerobic bacteria [2]. We present the case of a pyogenic hepatic abscess caused by *Peptostreptococcus anaerobius*, an anaerobic, gram-positive organism, considered to be commensal predominantly in the gastrointestinal and genitourinary tracts [3], but with an unusual additional finding of abundant monosodium urate (MSU) crystals within the purulent material. There are case reports of gouty tophi presenting as liver nodules as a first manifestation of gout in patients with underlying hepatic disease [4] [5]. However, a literature search did not reveal a case of this occurring in a patient without gout.

2. Case Presentation

The case is that of an 80-year-old female with a past medical history of hypertension, hyperlipidemia, hypothyroidism and dementia. She presented to the Emergency Room at Laredo Medical Center, Laredo, TX, complaining of abdominal pain. She was febrile, in hypovolemic and septic shock and in respiratory failure due to right pneumothorax and right lower lobe atelectasis with consolidation; she was intubated and admitted into the ICU. She was also found to have extensive, non-occlusive deep venous thrombosis (DVT) of the left lower extremity. Because of severe anemia (hemoglobin of 4.7 g/dL and hematocrit of 12.9%), she was transfused with 4 units of packed RBCs. An abdominal CT scan showed the presence of a large hepatic abscess (**Figure 1**). The patient was started on broad-spectrum antibiotics (Zosyn and Vancomycin) upon admission due to the septic shock. This regimen was appropriate as Zosyn provides coverage against a broad range of gram-negative and anaerobic organisms, while Vancomycin targets resistant gram-positive bacteria, including methicillin-resistant *Staphylococcus aureus* (MRSA), which are common in critically ill patients. A total of 250 mL of cloudy, brown fluid was drained from the liver abscess. It was noted by those present during the procedure that there were numerous, unusual, grossly evident, short, whitish, threadlike structures thought initially to correspond to a parasite of some sort. However, microbiologic culture of the fluid was positive for *Peptostreptococcus anaerobius* and microscopic examination revealed that the whitish structures actually corresponded to needle-like, crystalline material with the characteristic negative birefringence of MSU crystals intermixed with bacteria and necrotic cellular debris (**Figure 2**). It must be noted that serum uric acid level was normal at 5.6 mg/dL, creatinine was 0.73 mg/dL and all liver function tests were within normal limits. In addition, there was no clinical evidence of gout. During hospitalization, the patient continued on the same antibiotic regimen. The JP drain was left in the liver until the output

of the purulent material was less than 10 mL/day. After nine days, there was notable improvement of her respiratory status and near-total resolution of the abscess (Figure 3). Upon discharge, the infectious disease team recommended continuing the antibiotic regimen for a total of 42 days. At three weeks' follow-up, the patient continued in fair condition.

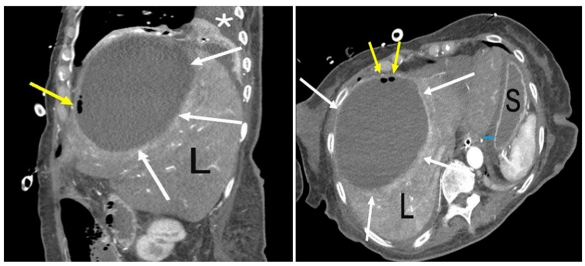

Figure 1. (A) Sagittal CT post-contrast image showing an intrahepatic fluid collection (white arrows). The fluid contains gas (yellow arrow). Incidentally noted is a right pleural effusion (white asterisk). L denotes the liver. (B) Axial CT post-contrast image showing an intrahepatic fluid collection (white arrows). The fluid contains gas (yellow arrows). The stomach wall is thickened at 0.8 cm (blue bar). S denotes the stomach lumen. L denotes the liver.

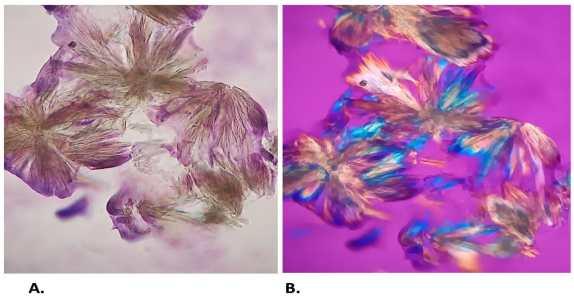

Figure 2. (A) Cytological examination of the intrahepatic fluid shows inflammatory exudate containing abundant monosodium urate crystals (Wright stain, 400×). (B) Monosodium urate crystals viewed under polarized light exhibiting their characteristic negative birefringence (400×).

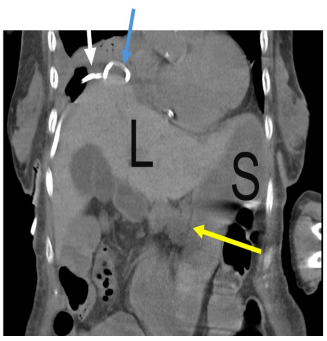

Figure 3. Follow-up coronal non-contrast image of the abdomen obtained 14 days after placement of a pigtail catheter shows the tip of the pigtail (blue arrow) within a subcapsular collection of fluid and air (white arrow). Stomach wall thickening is now resolved (yellow arrow). L denotes the liver. S denotes the stomach lumen. A rounded abscess is also no longer seen.

3. Discussion

Although gout is typically described as an inflammatory arthropathy, it is now well-known that gouty tophi can be present in unexpected, extra-articular locations, sometimes creating diagnostic dilemmas given that they can also be associated with infection, malignancy and other connective tissue diseases and can be the first manifestation of the disease [6]. Furthermore, systemic urate deposition has, until fairly recently, been an unrecognized complication of gout [7]. Since the formation of gouty tophi is characterized principally by a chain of events brought on by deposition of MSU crystals in tissues, it is of importance to understand the factors that contribute to MSU crystallization. A large literature review undertaken by Chhana *et al.* recognizes the main factor for urate crystallization to be a locally elevated urate concentration which leads to three key steps: 1) reduced urate solubility, 2) nucleation through formation of fibril-like nanoprecursors, and 3) growth, or evolution, of the crystal over time, depending on temperature and pH of the local environment [8]. Another key factor is the fact that gut bacteria can impact the host uric acid burden in a setting of metabolic and inflammatory conditions by an associated reduction in circulating levels of uric acid, a proinflammatory molecule. Studies by Kasahara *et al.* have identified bacterial taxa present in the gut microbiome—spanning multiple phyla—that use uric acid and adenine (a key precursor of nucleic acids in intestinal cells) as carbon and energy sources anaerobically. They discovered a gene cluster encoding key steps in purine degradation that is widely distributed in gut-dwelling bacteria. This demonstrates that gut microbes, by utilizing uric acid for their metabolic needs, are important drivers of host global purine homeostasis and uric acid levels and suggests that gut bacterial catabolism of purines may represent a novel mechanism by which the gut microbiome influences overall host health [9] [10]. This is supported by the study of Evgenovich Kondratiuk *et al.* in which synbiotics, defined as a mixture of probiotics and prebiotics (non-digestible fibers), when added to urate-lowering therapy (allopurinol) showed marked reduction in blood uric acid levels, cytokine levels and C-reactive protein and a normalization of the fecal microbiota, when compared to a control group where synbiotics were not added [11].

Microscopically, gouty tophi are almost instantly recognizable; they form as a response to the presence of MSU crystals in the soft tissues by eliciting an inflammatory infiltrate rich in histiocytes and multinucleated giant cells of foreign body-type. The ideal fixative to demonstrate the crystals in tissues is alcohol-based because formalin fixation tends to dissolve the crystals [12]. Therefore, they are often not present in histologic sections unless the uric acid burden is high enough that not all the crystals are dissolved. However, in synovial fluid and other body fluids, they are usually readily observed, given that synovial fluid is a poor solvent for sodium urate when compared to plasma, and also, because cytologic preparations employ alcohol-based fixatives. Therefore, they are usually easily diagnosed by their needle-like morphology and negative birefringence when viewed under polarized light.

In the case presented here, the hepatic lesion is not a granulomatous tissue response, but, rather, a hypoxic and acidic collection of necroinflammatory and pu-

rulent exudate produced through liquefactive necrosis secondary to infection by *Peptostreptococcus anaerobius*, which is an anaerobic, gram-positive organism that is ubiquitous but found principally in the gastrointestinal tract and genitourinary tracts. Due to their anaerobic nature, they can be found in abscesses outside their natural environment and because these bacteria can facilitate protein synthesis, their demand for purine is essential for their existence [13]. *Peptostreptococcus sp.,* therefore, participates, like other gut microbes, in removal of uric acid, given that this organism, as stated, utilizes it as an energy source. On that note, in the closed setting of an abscess, as in this case, it can be postulated that the uric acid levels far exceeded its ability to degrade it, leading to this increased local, insoluble urate concentration to undergo crystallization. A question remains as to why the presence of MSU crystals is not often seen or reported during the examination of abscess fluids, which often contain anaerobic organisms?

Regarding the appropriate management of pyogenic liver abscesses, Lam and Stokes point out that source control remains the most critical aspect. In other words, control of host factors such as sepsis and mechanisms in the development of the abscess. This is followed by an appropriate antimicrobial therapy individualized according to the etiologic agent, whether it be empirical or based on sensitivity studies [14].

4. Conclusion

We present the case of a septic patient without gout that developed a hepatic pyogenic abscess from which *Peptostreptococcus anaerobius* was cultured. It is an anaerobic, gram-positive organism considered to be a commensal and is predominantly found in the gastrointestinal and genitourinary tracts. It obligately utilizes purine for its survival, degrading it to uric acid, which, along with adenine, it utilizes as a carbon and energy source, thereby participating in removal of uric acid. We believe that, in this case, because of the closed conditions within the abscess, the accumulated excess of uric acid could not be degraded or excreted in the usual fashion and subsequently underwent the complicated process of crystallization, leading to the unusual finding of abundant monosodium urate (MSU) crystals in the purulent material. This differs greatly from gouty tophi, whether they be intra- or extra-articular in location, since they form as a response to the presence of MSU crystals in the soft tissues by eliciting an inflammatory response rich in histiocytes and multinucleated giant cells of foreign body-type, none of which are not present in this case.

Disclosure Statement

The authors declare that written informed consent from the patient is on file. The authors also have approval from Laredo Medical Center for publication of images and clinical details.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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