Introduction
Arthritis of the first metatarsophalangeal (MTP) joint of a different etiology than gout is known by the term pseudopodagra. Although the majority are due to deposits of other micro crystals, there has also been descriptions of other entities, such as infectious arthritis or lesion by foreign bodies. We present the case of a patient with a septic pseudopodagra due to Streptococcus agalactiae with a refractory evolution.

Clinical Case
The patient is a 71-year-old male, with a history of pulmonary tuberculosis, chronic simple bronchitis, sigmoidectomy due to adenocarcinoma, and now free of neoplasm and chronic liver disease of unknown origin, probably due to the ingestion of alcohol. He came for the first time to the emergency department of our center in the month of August 2004, due to pain and swelling of the first MTP joint of the right foot, presenting during the past 24 hours fever. The physical exploration showed fever (37.5°C), mucosal jaundice, stigmas of liver disease, a 3 cm liver enlargement, and mono arthritis of the first MTP of the right foot. On further examination, he showed a mild normocytic, normochromic anemia, 77 000/µL platelets, prothrombin time of 29% y and GOT 47 U/L, with the rest of the hemogram being normal, biochemistry, and urine sediment also normal. In spite of the patient not knowing that he had hyperuricemia and denying previous episodes of podagra, he was approached as gouty mono arthritis, and treatment with colchicine in a descendent pattern was established, as well as paracetamol and cryotherapy. A month later, the patient visited the outpatient rheumatology clinic with the persistence of pain and signs of inflammation, as well as fever in spite of the installed treatment. Due to the refractory podagra, a foot x-ray was taken, observing important bony erosions with joint destruction (see figure). With a diagnostic suspicion of septic mono arthritis in a patient with chronic liver disease in spite of systemic affection, hospitalization was decided upon to his study. Physical exploration and revealed arthritis of the first MTP with signs of inflammation and the analysis showed: erythrocyte sedimentation rate of 8 mm/h, C reactive protein, 7.98
mg/L; urates, 4.1 mg/dL; albumin, 22.6 g/L; bilirubin, 2.43 mg/dL; GOT, 37 U/L; GPT, 28 U/L; prothrombin time, 33%; and 146,000/µL platelets. Arthrocentesis of the first MTP was done, obtaining a drop of joint fluid that enabled us to do a culture and start empiric antibiotic treatment with ceftriaxone 2 g/24 h and cloxacylin 2 g/4 h intravenously while we awaited the results of the microbiologic study. In the joint fluid *Streptococcus agalactiae* sensitive to penicillin was isolated. After doing a transthoracic echocardiogram, endocarditis was ruled out. Antibiotic treatment was completed with the intravenous ceftriaxone during 2 weeks and posteriorly during 3 weeks after discharge. Evolution since that has been satisfactory with no functional consequences.

**Discussion**

The differential diagnosis of an acute monoarthritis is ample and its approach is based on arthrocentesis and the analysis of joint fluid, including biochemistry with glucose, proteins and a cell count, urgent Gram stain and culture and cytologic analysis with polarized light searching for crystals. Occasionally, the clinical scenario in which a monoarthritis presents itself is characteristic of a disease, making it possible to omit the arthrocentesis. This occurs especially if the affected joint is small, such as the first MTP, a situation that is highly suggestive of gout. In spite of this, there have been other described causes of first MTP joint arthritis that received the name of pseudopodagra that we must remember. The literature has well described cases of pseudopodagra due to hydroxiapatite. This entry is due to the deposit of hydroxiapatite crystals in the soft tissue adjacent to the first MTP joint (periartitis) or in the joint space, causing full-fledged arthritis. The deposit of calcium pyrophosphate crystals be it around the joint or inside the joint, has also been described as a cause of pseudopodagra. The presence of radio graphically evident periaricular calcifications in a patient with podagra must make a suspect any of these 2 entities. Spondyloarthropathies can present as an asymmetric oligoarthritis, and the MTP joint synovitis is a common manifestation. In a series of 143 patients with spondyloarthritis, 17 cases of pseudopodagra were found. Rheumatoid arthritis also frequently affects the MTP joints, although this is in the context of a symmetric polyarthritis, making it a rare differential diagnosis. Belsert’s disease, osteonecrosis, sesamoiditis, and hallux rigidus can also be included in the differential diagnosis of pseudopodagra. Finally, one must not forget that when faced with an acute monoarthritis, one is obliged to rule out an infectious cause through arthrocentesis and appropriate cultures, especially when a cause cannot be identified based on previous diagnoses. In our case, in spite of having a patient with a moderate consumption of alcohol, there were no previous indications of hyperuricemia or gout. The absence of septic data and the overall good state of our patient may does skip the arthrocentesis on the first encounter. Among the bone and joint infections that can present as a pseudopodagra, the most frequent one is tuberculosis, while the pyogenic are less frequent and are described in the literature as isolated cases, among them *Haemophilus influenzae, Pasteurella multocida, Bacillus, and Brucella*. There is also one described case of fungal infection. It must be emphasized that it is important to adequately approach the patient with acute monoarthritis in an emergency department based on the presence of an underlying illness, such as diabetes, chronic liver disease, or chronic kidney disease. Apart from being more susceptible to infections, these diseases can present with the more difficult evolution and hide serious disease, such as the present case.

**References**