

Case Report

Septic Arthritis with Candida in a Young Immunocompetent

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Introduction

Being part of the causes of septic arthritis, fungal arthritis is uncommon and is most often caused by the *Candida* species, especially in immunocompromised subjects [1,2]. This results most often as a complication of a disseminated candidiasis, and rarely as the first location of a *Candida* infection [3,4]. We hereby report a particular case by its clinical presentation in an immunocompetent subject.

Case Presentation

This is a 40-year-old patient with a history of lymph node tuberculosis at the 5th month of antibacillary treatment. He has been hospitalized for a painful swelling of the right elbow with functional impotence evolving for 1 month as well as a pyrexia. The general clinical examination finds a patient who is conscious, afebrile and hemodynamically stable; Osteoarticular examination denotes a flexion deformity of the right elbow, a painful swelling of the soft-tissue parts of the forearm with limitation of the movements of the right elbow. The rest of the physical examination was without detail except for three firm infracentimetric cervical nodes that are painless and movable in the surrounding tissue.

The laboratory tests find normal blood count, elevated CRP at 27 mg/l, accelerated sedimentation rate at 82mm the first hour, normal metabolic workup, with normal renal and liver function. X-rays of the right elbow denoted a diffuse pinch of the joint space with thickening of the soft tissues and subchondral cysts. CT of the right elbow without contrast injection showed a lytic lesion of the medial humeral condyle with a collection of soft parts in favor of an osteoarthritis (Figures 1 and 2).

Discussion

Since the onset of the human immunodeficiency virus (HIV) epidemic, the incidence of fungal arthritis has increased significantly [1]. It accounts for about 4% of all infectious arthritis [5]. Most cases of *Candida* arthritis are limited to individual case descriptions and relatively small sets of cases [6]. In a meta-analysis of a total of 112 published cases of *Candida* arthritis, the median age was 40 years (extremes ≤ 1 month and 84 years??). Sixty-nine patients (62%) were



Figure 1: In view of this clinical presentation and the patient's pathological history, a tubercular origin of the arthritis related to either a relapse or a resistance to the anti-tuberculous treatment was mentioned. However, an additional check-up was not in favour of this diagnosis. This check-up included:

- A surgical bone biopsy of the right humeral condyle with pus sample taken.
- The anatomopathological examination of the taken bone fragments showed an epithelioid and gigantocellular granulomatous process without caseous necrosis (Figure 2).

male and most of them were immunocompetent. History included surgery (35%), haematological malignancies (16%), solid organ transplantation (9%), traumas (9%), intravenous drug use (9%) and solid tumours (4%) [6].

Candida spp are commensal agents commonly found in the gastrointestinal tract, female genital tract, skin, sputum and urine [7,8]. Several species of *Candida* spp are known to cause arthritis, including *C. albicans*, *C. glabrata*, *C. guilliermondii*, *C. lambica*, *C. krusei*, *C. parapsilosis*, *C. tropicalis*, *C. stellatoidea* and *C. zeylanoides* [9-13].

Factors predisposing (to) *Candida* infection are represented by intravenous catheters, intravenous drug use, parenteral hypernutrition, malignant tumours, multiple surgeries, use of broad-spectrum antibiotics and artificial joints [1,10]. *Candida* arthritis may also be secondary to intra-articular corticosteroid injection [1].

According to a recent study of 112 cases of non-prosthetic *Candida* arthritis; 34% of which were immunocompromised; the majority of patients had no apparent underlying immune deficiency [6], which was the case of our observation.

The obvious pathogenesis of most cases of arthritis with *Candida* is that of the haematogenic spread to the joint. The mammalian synovium is extremely vascular and has no basal membrane around it; making it easy for the blood to access the synovial space. Therefore, haematogenic inoculation of *Candida* may affect normal joints [14]. However, direct inoculation or extension from an area adjacent to the infection may also occur [15].

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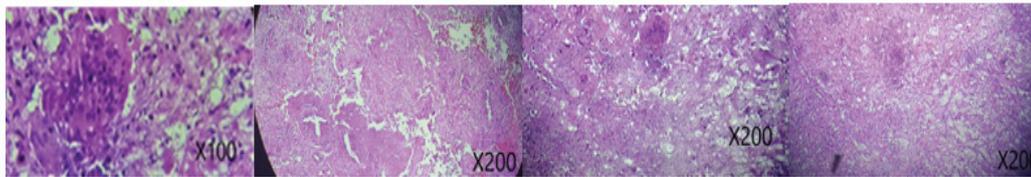


Figure 2: • Cytobacteriological analysis of pus with culture revealed candida spp.

- Culture of bone fragments on lowenstein-jensen media was sterile after 40 days of incubation at 37 degrees.
- Real-time PCR testing of mycobacterium tuberculosis on the taken bone fragments was negative.
- HIV, hepatitis C and B serologies were negative.

The diagnosis of candida arthritis was retained. The patient was treated with fluconazole 400 mg daily and functional rehabilitation for 12 weeks combined with debridement, surgical washing and regular monitoring of hepatic and renal functions. The patient's clinical evolution over a 9-month period was perfect with recovery of almost all of the elbow function.

The most commonly affected joints are in decreasing order: knee (75%), hip (15%), shoulder (7%), ankle (5%), elbow (4%), hand (3%), foot (2%) and wrist (2%) [6]. Candida infection is mostly monoarticular (69%), polyarticular in 22% of cases and oligoarticular in 9% of cases [6]. Candida fungal monoarthritis affecting the elbow in our observation represents therefore a relatively rare clinical entity for infectious arthritis, especially since it occurs in a young immunocompetent person without a significant predisposing factor.

There may be a long latency between fungemia or joint surgery and the onset of arthritis and, in rare cases, symptoms were reported up to 4 years prior to diagnosis [1,16,17]. Clinical symptoms include pain, stiffness and functional impotence [18]. Affected joints are swollen, painful and warm; typically the clinical pattern our patient presented himself with. Histological examination of the infected synovium reveals infiltration by mononucleated histiocyte cells, and fibrosis. Granulomas are rarely present [17,9]. The histological study in the present case is particular by the evidence of an epitheloidal granulomatous and gigante cell reaction without caseous necrosis.

Synovial fluid analysis in Candida arthritis reveals an inflammatory fluid with a predominance of neutrophils, as in pyogenic arthritis. However, this is not specific [6]. The direct examination of synovial fluid by Gram staining allows visualization of the pathogen in only 20% of cases, whereas better results are obtained with culture of synovial fluid or tissue [9]. Blood cultures are often negative [9]. In our observation, Candida was isolated in pus culture taken during the surgical biopsy.

Radiological examination reveals non-specific swelling of soft tissue, joint effusion and destructive joint changes that reflect the virulent nature of Candida arthritis; however, they are not specific. In the case of arthritis on a prosthesis, signs of loosening may appear [1].

The goals of the treatment of arthritis with Candida are multiple: relieve symptoms, eradicate infection, prevent joint damage and restore joint function.

There is no standard evidence-based treatment regimen for patients with fungal osteoarticular infections due to the relatively low frequency of this infectious location [6].

Recommendations for treatment of arthritis with Candida on healthy joints are based on case reports and open series. Recommendations include fluconazole (400 mg/d) for at least 6 weeks or a lipid formulation of amphotericin B (5 mg/kg/d) for at least 2 weeks, followed by fluconazole to complete treatment [19]. Treatments

lasting longer than 6 weeks are often necessary. Fluconazole, if used, may be given orally. Voriconazole and posaconazole appear as effective as fluconazole against *C. albicans* joint infection, but as they have greater intrinsic activity against *C. glabrata* and *C. krusei* which resist to fluconazole, they are potentially an oral option for septic arthritis caused by these pathogens [20]. However, clinical experience in septic arthritis with Candida is still limited with these new triazoles. Similarly, echinocandin-based antifungals (caspofungin, micafungin and anidulafungine) have been used successfully and may be an alternative to the treatment of arthritis with Candida spp, including those with resistance to triazole-containing drugs [19].

Surgery can also be an important complement to the medical treatment of arthritis with Candida. Although there is no significant difference in the therapeutic response between the medical treatment associated with surgical treatment versus medical treatment alone [6], this cannot justify medical treatment alone for any fungal arthritis, especially since there have been more deaths and disseminated candidiasis in the case of medical treatment alone. However, this difference may be due to an incomplete control of the source of infection or to underlying immunodepression in patients [6].

On the other hand, although combined medical and surgical procedures are certainly considered a standard of treatment for septic infections, severe comorbidities may prevent surgery in some patients [6]. We therefore suggest that a prospective study can help to define in detail the factors guiding the decision to combine surgical treatment with medical treatment in arthritis with Candida. In our observation, the patient received medical treatment based on fluconazole 400 mg/d for 3 months combined with a debridement and a surgical washing, all with both good clinical and biological evolution. This could illustrate the correct therapeutic response to combined medical and surgical treatment in the immunocompetent young person.

Eradication of joint Candida infection may be confirmed by analysis and culture of synovial fluid after treatment. In the case of fungal arthritis on prosthesis, the prosthesis must be removed. The use of intra-articular antifungal chemotherapy is controversial [15].

Conclusion

Candida fungal arthritis must remain in the minds of practitioners who manage patients with septic arthritis because only early management would prevent the subsequent joint damage. Understanding host factors and microbiology are essential and required for establishing good practice recommendations.

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