Multifocal Osteonecrosis Glucocorticoid Induced

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The present study describes a 29 years old patient diagnosed with aseptic osteonecrosis with multiple localization occurred after a corticoid treatment for chronic toxic hepatitis. The clinical and para-clinical examinations determined the diagnosis of Wilson disease and avascular necrosis with multiple localizations. The evolution of the disease was favourable following the surgical treatment consisting of bilateral total hip arthroplasty with cementless prosthesis, hemi-arthroplasty of the left shoulder with cementless prosthesis, orthotopic hepatic transplantation with an entire liver from donor in cerebral death and immunosuppressive, anticoagulant, antiretroviral and gastro-protective treatment. There is an increase of the number of patients undergoing a glucocorticoids treatment for several months, years or lifelong periods. This type of treatment increases the risk of osteonecrosis depending on the dosage and the duration of the treatment.

Keywords: aseptic necrosis, arthroplasty, corticoid therapy, Wilson disease

Avascular necrosis of the femoral head (ANFH) is a pluri-etiological and mono-pathogenic condition that knows a significant increase of its frequency determined by the technologic development and medical knowledge and the number of patients requiring corticoid treatment. The increasingly used therapy with glucocorticoids (GC) for different systemic pathologies and organ transplantation caused a parallel increase of the incidence of epiphyseal avascular necrosis of the bony extremities. ANFH is a crippling condition posing problems for diagnosis and therapy in order to prevent the occurrence of the musculoskeletal disability in young patients.

Wilson disease, hepatic-lenticular degeneration, represents a rare pathologic condition that is defined by specific fulminant clinical characteristics. It is a neurologic condition with lethal outcome associating a hepatic affectation in correlation with dysfunctions of copper metabolism; the treatment is drastic and it involves orthotopic hepatic transplantation[1,2].

The use of corticoid therapy in different systemic pathologies and organ transplants is considered to be one of the most important causes of the condition and especially the high dosage corticoids therapy has been suggested as an important influential factor in the occurrence of AVNFH[3,4]. Glucocorticoid therapy determines the occurrence of ischemia by increasing the intraosseous pressure due to the apoptosis of the endothelial cells, osteocytes, and the increase of the level and hypertrophy of adipose cells that has as consequence the diminution of the blood flow. The occurrence of AVNFH induced by corticosteroids was documented 1-2 months following the initiation of the high dosage corticosteroid therapy and the probability of the bone epiphyseal extremity to be affected by it is around 80% [5,6]. .

Experimental part

Materials and methods

Interdisciplinary examinations were conducted within the Rehabilitation Hospital. The blood samples were analysed using the GEM Premier 3000 analyser. The imagistic examinations were performed using the X-ray device of the Rehabilitation Hospital.

Case report

This study case brings forward a 29 years old non-smoking patient from the urban area hospitalised within the Orthopaedic and Trauma Clinic in the Rehabilitation Hospital, Iasi, Romania. The patient is not known to have any previous traumas, he is diagnosed with hernia at L5 – S1 that has been surgically treated, obesity type II and toxic-nutritional hepatic cirrhosis with corticoid therapy. The patient presents himself to the clinic for a specialised orthopaedic examination because of persisting acute pain in the hips. The clinical examination finds acute pain during the mobilization of the hips, more intense on the right hip, hypertrophy of the muscle of the right thigh of 4 cm, pain limited mobility of the right hip, bipod walking with a limp on the left side. The radiologic examinations establishes the diagnosis – avascular necrosis of the femoral head, stage IV post-cortisone and fracture of the femoral neck on the right side on pathologic bone (fig. 1).

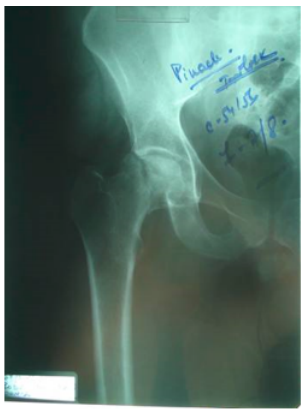

Fig.1 Fracture of the right femoral neck secondary to avascular necrosis of the femoral head

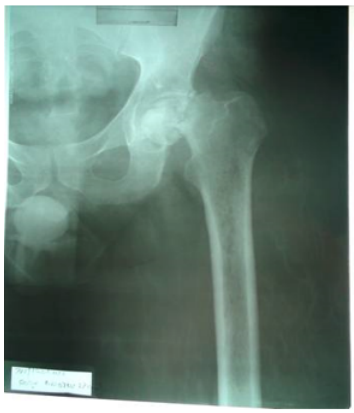

Fig. 2. Fracture of the left femoral neck secondary to avascular necrosis of the femoral head

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The patient undergoes surgical treatment with total hip arthroplasty on the right side using cementless Pinnacle Trilock prosthesis (fig. 6)

The patient returns to hospital 3 months after the surgery experiencing sudden acute pain of the left hip. The clinical examination documents the pain symptoms and a reduced mobility of the left hip, while the radiologic examination establishes the diagnosis of fracture of the left femoral neck secondary to avascular necrosis of the femoral head (fig. 2)

The patient undergoes surgical treatment with total hip arthroplasty on the left hip with cementless Pinnacle Trilick prosthesis (fig. 6) with favourable evolution and per primam scarring and progressive resuming of the hips mobility.

Two years after surgery the patient is hospitalised within the Centre of General Surgery and Hepatic Transplant with fulminant hepatic failure and parenchymalportal decompensation and is subsequently diagnosed with Wilson disease; next, the patient undergoes orthotopic hepatic transplantation intervention with entire liver from a donor in cerebral death.

The particularity of the case determines the patient to return to our clinic 1 year after the hepatic transplant accusing chronic pain in the glenohumeral joints with bilateral involvement. The clinical examination finds the mobility of the shoulders to be limited by pain, more intense on the left side, while the radiologic examination and nuclear magnetic resonance establish the diagnosis of avascular necrosis of the humeral head with bilateral involvement and functional and pain decompensation on the left side (fig. 3-5).

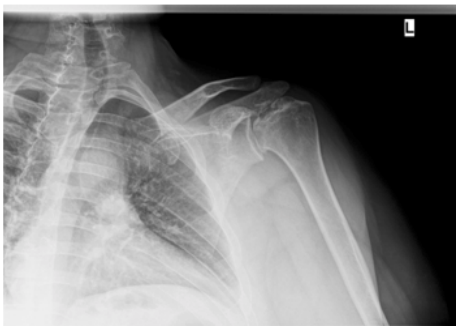

Fig. 3 Avascular necrosis of the humeral head, left involvement

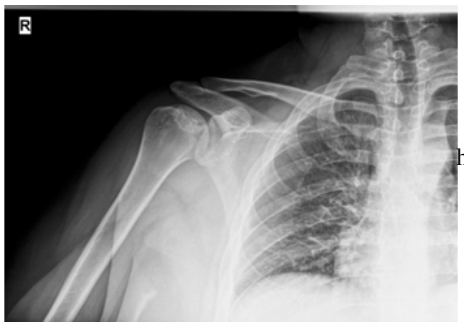

Fig.4. Avascular necrosis of the humeral head, right involvement

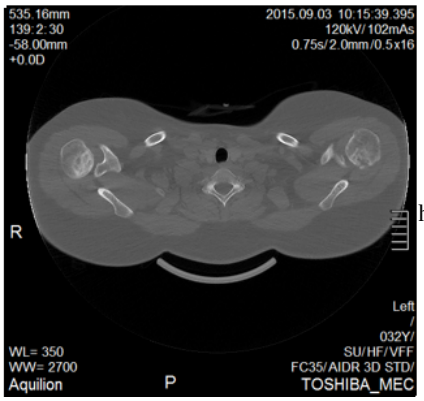

Fig.5. Avascular necrosis of the humeral head, bilateral involvement, NMR image

Following an interdisciplinary preoperative preparation, the patient undergoes surgical intervention with hemiarthroplasty of the left shoulder with cementlessSidus prosthesis (fig. 7) with favourable evolution.

Results and discussions

The case of this subject is a particular one because of his young age and the diagnosis is a devastating one since the avascular necrosis of the femoral head is in its terminal stage. Late diagnosis leaves no other option than surgical intervention with total hip arthroplasty (fig. 6).

Although the collapse of the femoral head has been noticed late after the initiation of the glucocorticoids therapy, the efforts of reducing the dosage were generally unsuccessful and thus the condition further affected other parts of the body.

Another particularity of the case is the immediate occurrence of the avascular necrosis of the humeral head with bilateral involvement within 3 years, the condition evolving rapidly towards its terminal stage and thus requiring surgical intervention with hemiarthroplasty of the left shoulder (fig. 7)

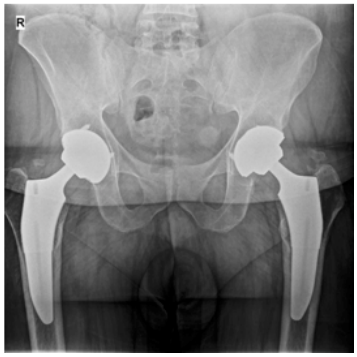

Fig.6. Bilateral total hip arthroplasty, postoperative radiologic examination

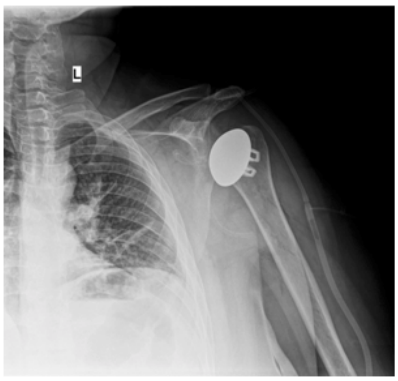

Fig.7. Hemiarthroplasty of the left shoulder, postoperative radiologic examination

Some authors suggest that avascular necrosis with multiple localizations is caused not only by corticosteroids treatment or alcohol abuse but it may have multiple factors etiology[7-10], as it has been observed in the case of our patient, still, we consider that corticosteroids therapy led to the fulminant evolution of the necrosis and its occurrence at the level of more extremities[10-12].

Although avascular necrosis most frequently involves the femoral head, we observed that glucocorticoids therapy may lead to multiple extremities being affected by the condition [11].

Some authors consider that osteonecrosis induced by glucocorticoids therapy may in fact be the apoptosis of osteocytes, a cumulative and irrecoverable defect that can affect the unique mechanical-sensorial role of the osteocytes – channels network and thus leading to the inexorable line of events causing the collapse of the femoral head. The apoptosis of the osteocytes induced by glucocorticoids therapy might explain the correlation between the total dosage of steroids and the incidence of

osteonecrosis and its occurrence after the termination of glucocorticoids therapy [5,6].

Conclusions

The avascular necrosis is a progressive, debilitating condition mainly affecting the young people; the corticoids therapy plays an important role in the occurrence and evolution of the disease. Although the occurrence of the area of necrosis can be observed immediately after the initiation of the glucocorticoids therapy, further efforts of reducing the dosage are generally unsuccessful.

Consequently, an increase of the number of patients undergoing glucocorticoids therapy for months, years or life long period increases the risk of osteonecrosis depending on the dosage and the duration of the treatment.

Despite the increased awareness of the effects of this devastating condition, the mechanism of in situ death of the bone and the imminent collapse remain unknown. Efforts of treating the condition have reduced if any results, while the clinical evolution is progressive and eventually requiring joint replacement.

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