ANABOLIC STEROID-INDUCED MYOSITIS AND OSTEITIS.
CASE REPORT THROUGH A RADIOLOGIC APPROACH

Keywords: Myositis; Osteitis; Steroids; Anabolic Agents.

Palabras clave: Miositis; Osteítis; Esteroides; Agentes anabólicos.

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RESUMEN

Introducción. La miositis es una complicación muy rara de las inyecciones extraarticulares de esteroides anabólicos y la osteítis no ha sido reportada como efecto adverso por esta causa. El presente reporte de caso aporta información sobre los hallazgos imagenológicos de estos dos tipos de inflamaciones.

Presentación del caso. Paciente masculino de 37 años, dedicado al fisicoculturismo, quien cinco días después de recibir una inyección de stanozolol presentó dolor y edema en la región glútea izquierda asociados a limitación funcional. El sujeto asistió a consulta por este motivo y se le realizó una ecografía y una resonancia magnética contrastada de pelvis, cuyos resultados permitieron diagnosticarle miositis del glúteo mayor izquierdo y osteítis del hueso ilíaco. Se indicó tratamiento con piperacilina-tazobactam y vancomicina por 10 días y no se requirió manejo quirúrgico dado que se obtuvieron buenos resultados.

Conclusión. La miositis es una complicación rara de las inyecciones de esteroides anabólicos en donde el mecanismo fisiopatológico de estas sustancias es incierto. Por su parte, la osteítis es una complicación aún más rara de la cual se presenta el primer caso conocido por esta causa. Dados los hallazgos se plantea que la miositis reportada es de tipo infecciosa; sin embargo, se requieren estudios adicionales que demuestren la asociación causal real.

ABSTRACT

Introduction: Myositis is a rare complication of extra-articular anabolic steroid injections, while osteitis has not been reported as an adverse effect from this cause. This case report provides information about imaging findings of these two entities.

Case presentation: A 37-year-old male, bodybuilder, presented pain and edema in the left gluteal region, associated with functional limitation, 5 days after receiving an intramuscular anabolic steroid injection (stanozolol). The man underwent an ultrasound scan and magnetic resonance imaging of the pelvis with contrast, which allowed making the diagnosis of myositis of the left gluteus maximus and osteitis of the iliac bone. The patient was treated with piperacillin-tazobactam and vancomycin for 10 days, without complications. No surgical management was required.

Conclusion: Myositis is a rare complication of anabolic steroid injections and the pathophysiological mechanism of this substance is unknown. Osteitis, on the other hand, is an even rarer complication and, to the best of our knowledge, this is the first known case associated with this cause. Given the findings, the myositis reported herein has an infectious nature; however, further studies are required to demonstrate the actual causal association.
INTRODUCTION

Myositis is an inflammation of the muscles that rarely occurs as a complication of extra-articular anabolic steroid injections. The incidence of this condition is not known and, during the preparation of the present study, only 16 publications on this issue were found. Osteitis, on the other hand, is an inflammation of the bones; the occurrence of this complication after the injection of these substances has not been previously reported, which makes the present case unique.

Anabolic steroids are human-made hormones that act on the androgen receptor and they are typically used by bodybuilders. (1) These substances are administered intramuscularly and the most commonly used injectable compounds are testosterone salts (testosterone cypionate, testosterone decanoate, testosterone propionate, testosterone phenylpropionate, testosterone isocaproate, and stanozolol—the compound used by the patient in this case—); 19-nortestosterone, used in the form of nandrolone decanoate and nandrolone phenylpropionate; boldenone undecylate; and methenolone enanthate.

Although the pathophysiological mechanism of anabolic steroids in myositis is unknown, three hypotheses have been put forward: the inoculation of microorganisms from the skin, the hematogenous spread of bacteria from another focus to the injection site, and the innate immune response to steroid ester crystals.

In the absence of abscess or necrosis, the standard of care for steroid myositis includes local anti-inflammatory measures, muscle rest, and intravenous antibiotics. On the contrary, if abscesses are present, they must be drained and, if necrosis is generated, debridement of the dead tissue must be performed; likewise, specific tissue repair techniques must be applied depending on each case.

CASE PRESENTATION

This is the case of a 37-year-old male patient, mestizo, from Bogotá D.C. (Colombia), bodybuilder, with a stable socioeconomic condition and a healthy lifestyle without cardiovascular risk factors. He reported consuming a high-protein diet, as well as amino acids and fatty acids supplements.

The patient attended the emergency service of a quaternary care center on January 6, 2020, due to a feverish sensation (not quantified) for 3 days, stabbing pain and a rash in the left buttock, which prevented normal mobilization of the left leg. A soft tissue ultrasound was performed, which showed no abscesses or necrosis, but did show changes due to myositis of the left gluteus maximus. The subject was discharged with outpatient antibiotic therapy (sultamicillin) and, since he had no appropriate response to the treatment, he attended the same center again on January 8, 2020. The next day, he underwent magnetic resonance imaging (MRI) of the hip with contrast that confirmed the diagnosis of left gluteal myositis. Intravenous antibiotic treatment with 600mg of clindamycin every 8 hours was indicated and he was transferred to the Hospital Universitario Nacional de Colombia (HUN) by ambulance.

On January 11, 2020, at 04:00 a.m., the patient was admitted to the hospitalization service of the HUN with blood pressure of 146/93 mmHg, heart rate of 74 beats/minute, respiratory rate of 19 breaths/minute, temperature of 36.6°C and oxygen saturation of 92% on room air. Physical examination showed edema and erythema of the right gluteal region, induration without areas of fluctuation and loss of thigh extension strength without distal neurovascular deficit. The patient claimed that he did not have any previous recognized medical, genetic, or surgical history, but
reported receiving an anabolic steroid injection into the left buttock in an unprofessional setting.

That same day, at 11:45 a.m., he was treated by the internal medicine service. Laboratory tests were requested, obtaining the following results: C-reactive protein: 28 mg/dL, total leukocytes: 7680/μL, neutrophils: 5450/μL, platelets: 269,000/μL, creatinine: 0.75 mg/dL, blood urea nitrogen: 14.69 mg/dL, and creatine phosphokinase: 205 mg/dL; no liver function tests were performed and no blood or soft tissue cultures were obtained due to the absence of signs of systemic inflammatory response or sepsis. Based on the results, a diagnosis of anabolic steroid-induced myositis and possible fasciitis was suspected. It should be noted that there was no differential diagnosis in his medical record.

On January 12, at 9:40 a.m. (29 hours and 40 minutes after admission to the HUN), the patient underwent a soft tissue ultrasound of the left buttock to confirm the suspected diagnosis (Figure 1). Although no collections suggestive of hematoma and/or abscesses were observed in Figure 1A, there was evidence of increased muscle thickness, diffuse increase in muscle echogenicity and altered fibrillar pattern, findings interpreted as a myositis with dimensions of 14.4x5.6x14.4 cm and volume of 608 cm³. In Figure 1B, no blood collections were observed either, but increased thickness and subcutaneous fat stranding was found; the latter was associated with cellulitis adjacent to the focus of myositis in the left gluteus maximus.

![Figure 1. Ultrasound of the left gluteal soft tissue. A) axial plane of the left gluteus maximus muscle; B) axial plane of the subcutaneous adipose tissue from the superficial left gluteal region to the muscle. Source: Document obtained during the study.](image)

Based on the ultrasound findings, a pelvic MRI with contrast was performed (Figures 2, 3, 4 and 5). In axial T2 (Figure 2), an increase in signal intensity between the muscle fibers of the left gluteus maximus was evidenced due to an edema without disruption of the fascial planes (white arrow) or subcutaneous adipose tissue. Moreover, increased signal intensity was observed from the left iliac bone adjacent to the myositis site (blue arrow). Due to the characteristics, it was suspected that these areas could correspond to fatty infiltration or edema.
The SPAIR (Spectral Attenuated Inversion Recovery) sequence in the axial plane (Figure 3) confirmed increased signal in the described areas. Using the fat suppression technique, all the signal from the fat was eliminated, confirming that the areas with increased signal intensity in the iliac bone and the left gluteus maximus were areas of edema.

Considering the described inflammatory changes in soft tissues, it was suggested that bone edema was secondary to osteitis (reactive) or acute osteomyelitis. Diffusion-weighted images were used to verify this: b-800 (Figure 4A) and apparent diffusion coefficient (ADC) map images (Figure 4B), which showed an area of free diffusion of water molecules in the iliac bone (decrease in signal intensity in b-800 corresponding to an area of increased signal intensity in the ADC map). Instead, acute osteomyelitis presents with abscesses that have diffusion restriction: high signal in b-800 corresponding to an area of decreased signal intensity in the ADC map.

Finally, in T1 without contrast (Figure 5A), it was evident that the area of edema was iso-intense to the rest of the bone. In post-contrast T1 image (Figure 5B), it was found that this area had homogeneous enhancement with round morphology, which could be associated with the bone marrow and the bone cortex, a finding related to osteitis.
No specific signs of necrotizing fasciitis were identified on MRI, so no muscle biopsy was performed.

Based on the results of the laboratory tests and the diagnostic images, a diagnosis of myositis and osteitis was made. The patient received non-steroidal anti-inflammatory drugs (diclofenac), acetaminophen targeting the mechanisms of immune inflammation, and empirical intravenous antimicrobial therapy: first clindamycin and then vancomycin and piperacillin-tazobactam due to the persistence of local inflammatory signs. The general surgery and orthopedics services jointly decided not to perform any surgery because there were no local or systemic complications.

The patient was ordered to continue antimicrobial therapy on an outpatient basis with a home hospitalization plan and telephone follow-up, obtaining a favorable outcome and no anatomical deformities or functional disorder. Tolerance and adherence to treatment were adequate and no adverse reactions occurred.

DISCUSSION

In the present study, it was found that the availability of multiple imaging techniques and clinical follow-up are strengths for the diagnosis and treatment of myositis and osteitis as complications of anabolic steroid injection. On the other hand, the lack of specimens for histopathological and microbiological analysis was identified as a weakness.

Anabolic steroids are hormones injected intramuscularly, which act on the androgen receptor, and the population most often associated with their use are bodybuilders. (1) The compound used by the reported patient was stanozolol, which corresponds to the testosterone salt group.

Myositis as a complication of extra-articular anabolic steroid injections is a finding barely reported in the literature (16 results in the PubMed search), and there are no case series or specific systematic reviews addressing the topic. As for osteitis, no literature report describes it as a complication of the administration of these substances.

Myositis is classified as a serious local complication of steroid injection, and although the pathophysiological mechanism is unknown, three main hypotheses on the causes have been proposed: inoculation of microorganisms from the skin, hematogenous spread of microorganisms from another focus to the injection tract, and innate immune response to steroid ester crystals. In cases reported in the literature, the methicillin-resistant Staphylococcus aureus...
bacteria (2-8) has been frequently isolated, so most anabolic steroid-induced myositis is believed to be infectious (pyomyositis). However, this conclusion cannot be reached since there are no studies that prove the causal relationship.

The most common sites for anabolic steroid injection are the gluteus maximus, the deltoid, and the vastus lateralis muscles. (9) Therefore, infectious signs should be looked for in these sites in the first instance.

Pyomyositis occurs in three stages: 1) invasive stage, characterized by muscle edema and pain from bacterial seeding; 2) suppurative stage, characterized by abscess formation that occurs 10-21 days after injection; and 3) late stage, characterized by multiple organ failure. It should be noted that if the first two stages are not treated promptly, the third stage can lead to death. (10)

In ultrasound, myositis is characterized by increased thickness and echogenicity of the muscle, both focal and diffuse, and altered fibrillar pattern secondary to edema and hyperemia.

The characteristics of myositis and osteitis observable on MRI and CT scans are summarized in Table 1:

| Technique/Pathology | Myositis                                                                                                                                    | Osteitis                                                                                                                                             |
|---------------------|--------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------|
| Magnetic Resonance Imaging | Increased muscle thickness and signal intensity on T2-weighted or STIR sequences, decreased T1-weighted signal between muscle fibers, and secondary alteration of muscle fibrillar pattern. There is no diffusion restriction to the flow of water molecules but a homogeneous enhancement of the inflammation with the contrast agent. | Edema of the bone cortex next to the focus of infection in the soft tissues: ill-defined foci of hyperintensity on T2, with no decrease in signal in the fat suppression sequences, and isointense on T1-weighted imaging. There are no foci of diffusion restriction of free water molecules and the enhancement pattern is usually homogeneous and generally confined to the bone cortex. |
| Computerized axial tomography | Increase in muscle thickness, which becomes asymmetric with respect to the contralateral side, and homogeneous decrease in the attenuation values with loss of fibrillar pattern and definition of the borders between the muscle and the subcutaneous cellular tissue. | Signs with low sensitivity.                                                                                                                                |

In the MRI, the areas of myositis have a homogeneous enhancement with the use of gadolinium-based contrast agent, while the areas of muscle necrosis do not present any enhancement. If abscesses coexist, they have well-defined walls (often thick) that are enhanced by the contrast agent.

Muscle tumors, both benign and primary malignant, are an important differential diagnosis, since they can be differentiated by diffusion restriction on MRI. In addition, malignant tumors are very likely to cause disruption of the fascial planes with invasion of adjacent structures.

Another relevant aspect of MRI is the identification of specific signs of necrotizing fasciitis, such as extensive involvement of the deep fascia (intramuscular or contacting the superficial fascia in more than three locations),
absence of fascial enhancement with gadolinium-based contrast agent, and, less frequently, the presence of gas. Computed tomography (CT) is more sensitive in detecting gas using bone window but the detection of myositis is more difficult with this technique due to its poor soft tissue resolution.

As mentioned above, osteitis is a rare complication of extra-articular anabolic steroid injection that is not reported in the literature, which makes the case presented herein valuable. In that sense, it is worth mentioning that such inflammation is characterized by an edema of the bone cortex adjacent to the focus of the infection in the soft tissues, in addition to ill-defined foci of hyperintensity on T2 and isointense on T1-weighted imaging. In osteitis, there are no diffusion restriction foci on MRI and the enhancement pattern is usually homogeneous and confined to the bone cortex. (10,14)

CONCLUSION

Myositis is a rare complication of anabolic steroid injections and the pathophysiological mechanism of this substance is unknown. Osteitis, on the other hand, is an even rarer complication and, to the best of our knowledge, this is the first known case associated with this cause. Given the findings, the myositis reported herein has an infectious nature; however, further studies are required to demonstrate the actual causal association.

PATIENT’S PERSPECTIVE

The patient stated that he received a high-quality, comprehensive, and timely treatment.

ETHICAL CONSIDERATIONS

This case report was prepared after obtaining the informed consent of the patient.

CONFLICT OF INTEREST

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