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Rhabdomyolysis of the Deltoid Muscle in a Bodybuilder Using Anabolic-Androgenic Steroids: A Case Report

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Objective: Rhabdomyolysis (breakdown of skeletal muscle tissue) may be caused by mechanical, physical, chemical, or biological factors. We present the unique case of a bodybuilder who developed localized rhabdomyolysis of the deltoid muscle after injection of steroids into the shoulder region.

Background: A 39-year-old amateur bodybuilder presented to the emergency department with excruciating pain and inability to move his right shoulder after injecting stanozolol, an anabolic-androgenic steroid (AAS), into his right deltoid muscle on the same day. On physical examination, the right deltoid muscle was swollen and tense and the surrounding skin red, tender, and warm. He had no fluctuation or systemic fever and no sensory or motor deficit. His distal pulsations were distinct. Laboratory test results suggested massive rhabdomyolysis. The major magnetic resonance imaging finding was diffuse hyperintensity signals on T2-weighted images of the deltoid muscle, which was consistent with edema.

Differential Diagnosis: Polymyositis and dermatomyositis, mild injury, infectious myositis without phlegmon or abscess formation, radiation therapy, subacute denervation, compartment syndrome, early myositis ossificans, rhabdomyolysis, and sickle cell crisis.

Treatment: The patient was treated with intravenous fluid replacement and sodium bicarbonate to alkalinize the urine. Four days after admission, his pain had decreased, he had regained range of motion, and his renal function remained unaffected.

Uniqueness: Anabolic-androgenic steroid use is associated with various side effects that are generally systemic and dose related. We could not find reports of localized side effects of AAS use, as this case presented, elsewhere in the English-language literature.

Conclusions: "Doping" among amateur athletes occurs frequently. It can cause acute and chronic health problems, most of which are systemic. This is the first description of localized rhabdomyolysis in the area of an AAS injection.

Key Words: anabolic agents, adverse effects

Athletes, especially bodybuilders, use anabolic steroids because they believe that steroids will allow increased periods of intensive training to enhance muscle strength.[1] Products used are mainly anabolic-androgenic steroids (AAS)[2] because of their myotrophic actions and stimulatory effects on the brain.[3] Use of AAS is associated with various side effects that are generally systemic and dose related, such as hepatotoxicity, changes in serum lipid profile, fluid retention, cardiac hypertrophy, hypogonadotropic hypogonadism, gynecomastia in males, hirsutism and menstrual irregularities in females, and behavioral changes.[3,4] Therefore, illicit use of megadoses of AAS can lead to serious and irreversible organ damage. Searching the English-language literature, we could not find reports of localized side effects of AAS use. We present a case of localized rhabdomyolysis (breakdown of skeletal muscle tissue) of the deltoid muscle at the site of AAS injection in a bodybuilder. Our purpose for this case report is to describe this rare side effect and to suggest a mechanism of its pathogenesis.

CASE REPORT

A 39-year-old, previously healthy, amateur bodybuilder presented to the emergency department with excruciating pain and inability to move his right shoulder after injection of anabolic steroids into his right deltoid muscle followed by an exercise session on the same day. The patient engaged in bodybuilding activities 5 times each week for the previous 8 years, and for the last 7 years, used AAS to augment his performance. Using a 23-gauge needle and sterile technique, he injected testosterone derivatives in a 1-mL to 2-mL solution into the lateral aspect of the deltoid muscle 4 times each week. The patient denied any abrupt overstretching movement that may have caused a muscle strain.

On physical examination, the right deltoid muscle was swollen and tense; the skin around it was red, tender, and warm (Figure 1). He had no bruising around the shoulder, no fluctuation or systemic fever, and no sensory or motor deficit. Distal pulsations were distinct. Laboratory investigation revealed considerably elevated levels of enzyme markers of muscle breakdown, with a creatine kinase level of 18 200 U/L (normally $<$195 U/L), which suggested massive rhabdomyolysis (Table). Serum potassium level was 5.9 mmol/L (normally $<$5.2 mmol/L). Renal function and white blood cell count were intact. Urine myoglobin level was tested only on day 3 after admission and was slightly elevated at 63 μg/L (normally $<$60 μg/L). Ultrasound examination showed swollen muscle and inhomogeneous-mixed echogenecity without discrete fluid collection. The major magnetic resonance imaging finding was diffuse hyperintensity signals on T2-weighted images of the deltoid muscle, consistent with edema. No focal abscess or foreign body was identified. However, imaging showed 2 areas suggestive of early focal muscle necrosis (Figure 2).

The patient was treated with intravenous fluid replacement and sodium bicarbonate to alkalinize the urine. A

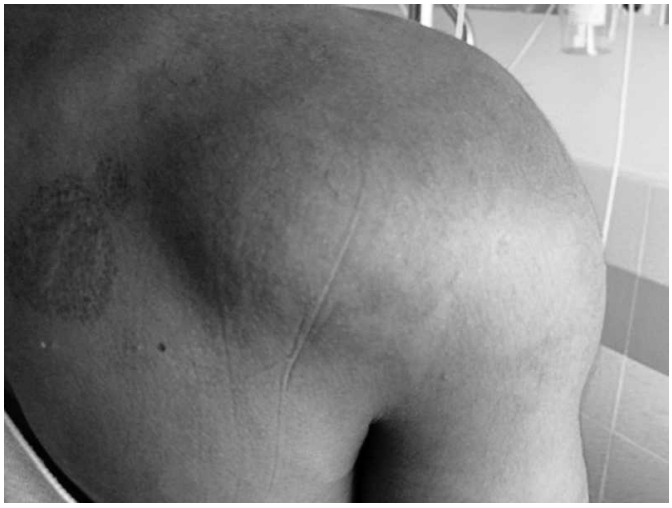

Figure 1. Posterolateral view of the right shoulder region shows swollen deltoid muscle with red, tender, and warm skin around it.

sample of the drug was analyzed and found to contain stanozolol, a synthetic anabolic steroid derived from testosterone. On day 4 after admission, he had decreased pain and regained range of motion; renal function remained unaffected. Because the patient recovered promptly, a muscle biopsy was not performed. On follow-up examination, the patient had recovered completely, returned to bodybuilding training, and regained range of motion and strength in his shoulder.

DISCUSSION

Magnetic resonance imaging has an important role in detecting and characterizing of pathologic conditions of skeletal muscle that cause changes in signal intensity, and it has a higher sensitivity in detecting abnormalities in muscles than computed tomography or ultrasound (100%, 62%, and 42%, respectively).[5] Inflammatory, infectious, traumatic, neurologic, neoplastic, and iatrogenic conditions can affect muscle signal intensity.[6] A pattern of increased signal intensity, which probably reflects edema in the injured muscle as seen in the case presented, narrows the differential diagnosis to polymyositis and dermatomyositis, mild injury, infectious myositis without phlegmon or abscess formation, radiation therapy, subacute denervation, compartment syndrome, early myositis ossificans, rhabdomyolysis, and sickle cell crisis.[6] In our patient, the absence of fever, leucocytosis, and abscess formation and the spontaneous resolution without antibiotic treatment ruled out inflammatory and infectious conditions and made rhabdomyolysis the most probable diagnosis.

Rhabdomyolysis, or acute skeletal muscle destruction, has several causes, including toxic, ischemic, infectious, inflammatory, and metabolic insults.[7,8] Exertional rhabdomyolysis refers to muscle cell damage caused by intense exercise, during which the energy supply to muscles is insufficient to meet demands. Untrained persons who exercise vigorously in hot, humid weather are known to develop the condition, but well-trained athletes and persons whose jobs demand hard physical labor are also at risk.[7] Therefore, rhabdomyolysis should be considered in the differential diagnosis of acute muscle weakness or pain in patients who have recently increased their levels of exercise, regardless of their previous levels of physical fitness.[9]

Breakage of skeletal muscle membrane leads to intracellular content leakage, including creatine kinase, serum glutamic-oxaloacetic transaminase, lactate dehydrogenase, myoglobin, electrolytes (eg, potassium and phosphate), and purines. When blood myoglobin concentrations rise to approximately 3 mg/L, protein crosses the renal threshold and can be found in the urine, a condition known as myoglobinuria. In high amounts, myoglobin can precipitate in kidney tubules and compromise renal function.[8] Myoglobinuria may be visible for only 4 hours, but its presence or absence cannot be related directly to the severity of muscle damage.[7]

Cases of diffuse rhabdomyolysis associated with excessive physical exertion are commonly reported, especially in long-distance runners, weight lifters, football players, and military personnel participating in physical training. However, localized rhabdomyolysis associated with weight lifting is reported infrequently. Bolgiano[10] was the first to report rhabdomyolysis of the biceps occurring after a weight-lifting session in a 40-year-old man who denied use of anabolic steroids or other drugs. Other authors have described rhabdomyolysis of the long head of triceps,[11] bilateral teres major,[12] and bilateral quadriceps[13] muscles after intensive muscular exercise. The exact incidence of rhabdomyolysis among bodybuilders remains unknown. Many cases in the exercise setting are probably unrecognized and diagnosed as simple muscle strain.

Several authors have described rhabdomyolysis after vigorous weight lifting by athletes supplementing their weight-training program with AAS.[14–16] However, these case reports showed generalized rhabdomyolysis that was not isolated to the site of drug injection and, as mentioned previously, may be due to the strenuous physical activity per se.

Localized muscle damage was present at the area of AAS injection, but the causal mechanism for rhabdomyolysis in

Table. Laboratory Test Results of the Patient

			Days After Admission				
	Normal	On Admission	1	2	3	4	7
White blood cell count	4.0–10.8	11.29	13.29	11.67		12.21	
Creatine kinase, U/L	< 195		18 200	17 423	13 281	9925	2615
MB isoenzyme of total creatine kinease, %			1.0	2.6	1.6	1.5	2.3
Serum glutamic-oxaloacetic transaminase, U/L	<40		312	299	247	200	65
Lactate dehydrogenase, U/L	< 260		1136	1093	754	636	431
Potassium, mmol/L	3.5–5.2	4.4	5.9	5.9	5.1	4.7	5.6
Urine red blood cell	0		10–20			10–20	
Urine protein	Negative		Positive			Negative	
Urine color			Amber			Yellow	

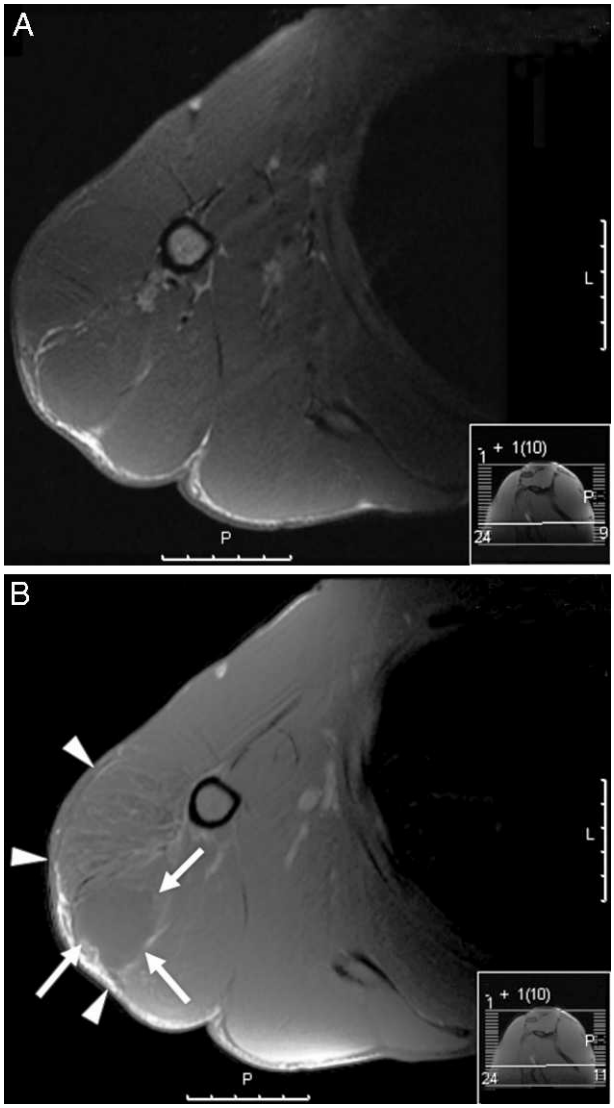

Figure 2. Axial gradient echo images before (A) and after (B) intravenous administration of gadolinium show diffuse enhancement of the deltoid muscle (arrowheads). However, the non-enhancing well-defined region (arrows) is consistent with early necrosis or early abscess formation.

this patient was unclear. Although we could not find a report in the literature describing such a reaction, rhabdomyolysis might be due to a toxic effect of a drug ingredient, or it may be the result of compartment syndrome.

Acute compartment syndrome of muscles surrounding the shoulder is a rare condition, because the arm fascia is yielding and can accommodate swelling without a corresponding increase in compartment pressure. Only 3 such cases are reported in the literature[17,18]; all cases occurred in patients with drug overdosage or intoxication after minor trauma.

For the case described, we hypothesized that a combination of increased compartment volume due to injected fluid and hematoma and decreased elasticity in the fascial envelope caused by the bodybuilder's increased muscle mass resulted in increased intracompartmental pressure. In addition, strenuous physical training may have led to a cascade of events, causing muscle ischemia and rhabdomyolysis.

CONCLUSIONS

"Doping" among amateur athletes occurs frequently. Doping can cause acute and chronic health problems, most of which are systemic. This case report is the first description of localized rhabdomyolysis in the area of AAS injection.

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