CASE REPORT

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Sudden Cardiac Death During Exercise in a Weight Lifter Using Anabolic Androgenic Steroids: Pathological and Toxicological Findings

REFERENCE: Luke, J. L., Farb, A., Virmani, R., and Sample, R. H. B., **"Sudden Cardiac Death During Exercise in a Weight Lifter Using Anabolic Androgenic Steroids: Pathological and Toxicological Findings,"** *Journal of Forensic Sciences*, JFSCA, Vol. 35, No. 6, Nov. 1990, pp. 1441–1447.

ABSTRACT: A 21-year-old, previously healthy weight lifter collapsed during a bench press workout. He had taken anabolic androgenic steroids parenterally for the previous several months. Pertinent autopsy findings included marked cardiac and renal hypertrophy and hepatosplenomegaly, with regional myocardial fibrosis and focal myocardial necrosis. Nandrolone (19-nor-testosterone) metabolites were identified in postmortem urine. The possible etiologies of the cardiac findings are discussed.

KEYWORDS: pathology and biology, steroids, death, sudden cardiac death, anabolic steroids, myocardial hypertrophy, myocardial fibrosis, myocardial necrosis, exercise, athletes

Sudden cardiac death during exercise has not heretofore been listed with the medical and psychiatric complications of anabolic androgenic steroid (steroid) usage [*1*]. Because of the recent vintage of the phenomenon of steroid abuse and the scant numbers of deaths of this type, the cardiac and other systemic pathologic findings of steroid use are just beginning to be described. In the midst of the abundant recent publicity regarding steroid usage among high school [*2*], intercollegiate, and world-class athletes, the authors of this paper are aware of only a limited number of detailed postmortem examinations involving steroid abusers and none in which postmortem steroid analyses have been performed for confirmation and documentation of drug usage.

The opinions contained herein are the private opinions of the authors and are not to be construed as official or as reflecting the views of the U.S. Departments of the Army or Defense.

Received for publication 22 Jan. 1990; accepted for publication 1 Feb. 1990.

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Case Report

A 21-year-old white male complained of dizziness and collapsed during a weight-lifting workout at the gym he frequented on a regular basis for bodybuilding purposes. He had entered a bodybuilding contest scheduled for six months later and was training toward that end. He had left work at 5 p.m. on the day of his death. By 6:15 p.m., he had completed two sets of bench-pressing exercises and other related bodybuilding work when he collapsed. Paramedics responding to the gym found him to be in ventricular fibrillation. He received the standard resuscitative modalities at the scene and subsequentto being taken to the emergency room of a local hospital, including epinephrine, atropine, 50% dextrose, Narcan, dopamine, bicarbonate, Levophed, and multiple attempts at defibrillation, all to no avail. He was pronounced dead at 7:45 p.m.

His past medical history revealed no significant illness. He had graduated from high school three years previously, where he played fullback and middle linebacker on the varsity football team for four years. After graduation, he had worked as an auto mechanic at a local service station owned by his father.

According to family and friends, he had been taking anabolic steroids parenterally in the form of testosterone and nandrolone for the previous several months in an effort to improve his bodybuilding performance. Three factory-sealed glass vials; several unidentified red, tan, and brown pills, presumably vitamins; and two packages of K-Lyte, 25 meq, a potassium supplement, were found by police investigators in his gym bag. The vials consisted of testosterone cypionate, 200 mg/mL (Steris Laboratories, Inc., Phoenix, Arizona); nandrolone (19-nor-testosterone) decanoate, 100 mg/mL; and nandrolone decanoate, 200 mg/mL (both by LyphMed, Inc., Melrose Park, Illinois). According to police, the steroids were injected twice weekly, sometimes with the assistance of others, and the last injection occurred approximately one week prior to death.

Autopsy Findings

The body measured 5 ft, 9 in. (1.75 m) and weighed 213 lb (96.6 kg). Intracaths and other signs of resuscitative intervention were present at the neck and upper extremities. The extremities and torso were heavily muscled, with striae, variably tan to maroon in color and measuring up to 15 cm in greatest dimension, present at the anterior and lateral aspects of the shoulders. The lower extremities had been recently shaved.

At the central right buttock was a multicolored tattoo of a weight lifter (Fig. 1). At the skin surface directly superior and lateral to the top portion of the tattoo was a poorly defined, variably tan to yellow region of recent and resolving ecchymosis that measured up to 3 cm. A 1.5-cm region of fresh red-blue hemorrhage was present on sharp dissection within the central confines of the lesion. Approximately eight in-line small (less than 0.1 cm) needle puncture wounds were present directly lateral to the ecchymosis.

The following organ weights were recorded at autopsy: heart, 530 g; lungs, 1050 and 870 g, right and left, respectively; spleen, 520 g; liver, 2500 g; kidneys, 510 g combined; and brain, 1400 g.

Examination of the heart demonstrated marked left-sided hypertrophy. The epicardial and endocardial surfaces were smooth and glistening. The coronary arteries exhibited no evidence of atherosclerosis. The cardiac valves were unremarkable. The sectioned myocardium revealed extensive regional fibrosis (Fig. 2), with principal involvement of the subepicardial and central aspects of the left ventricle and interventricular septum, and relative sparing of the subendocardium of the left ventricle and interventricular septum and of the right ventricle and atria. The fibrotic areas were retracted below the sectioned surfaces and measured up to approximately 8 cm in greatest dimension at the epicardial surface and 1.5 cm centrally. There was extensive left and right ventricular hypertrophy

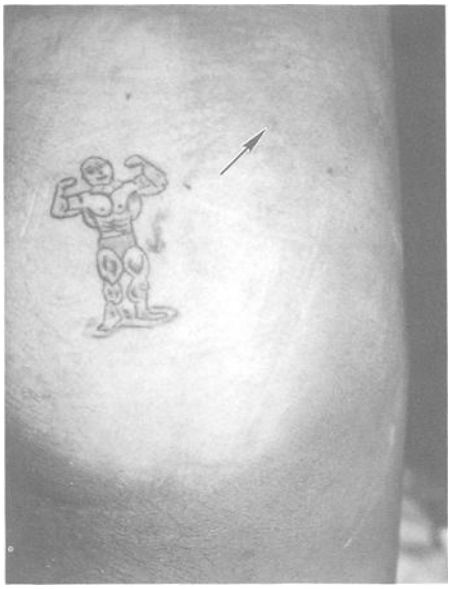

FIG. 1—*Weight lifter tattoo on the right buttock. Note the ecchymosis and needle puncture wounds* (arrow) *superior and lateral to the tattoo.*

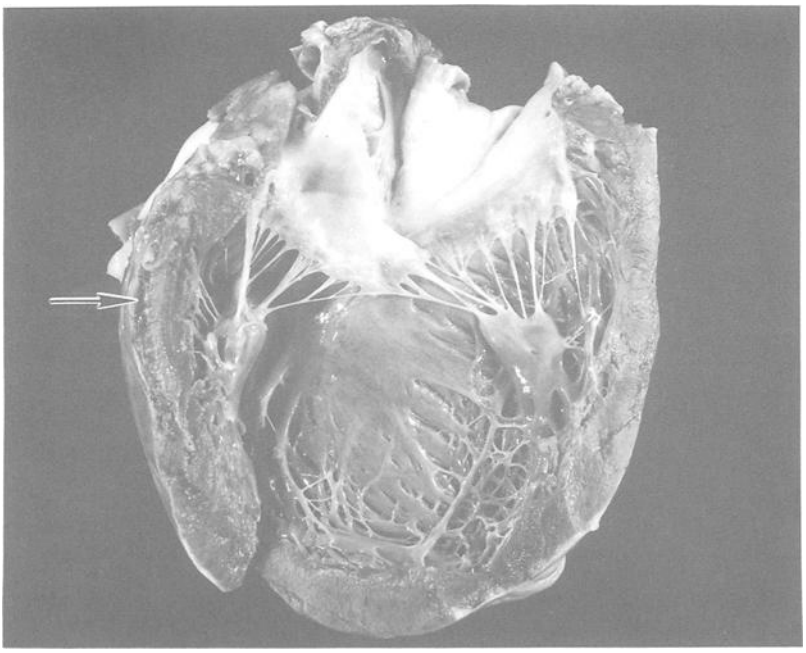

FIG. 2—*The subject's heart, demonstrating left-sided hypertrophy and extensive myocardial fibrosis* (arrow).

and dilatation, with the left ventricular chamber measuring up to 11 cm from base to apex and the left ventricular free wall up to 1.6 cm in greatest width. The right ventricle measured approximately 0.4 cm in thickness.

Microscopic examination of the heart revealed regional fibrosis, with principal involvement of the subepicardial and central left ventricular and interventricular septal areas, without evidence of inflammation (Fig. 3). In addition, in the multiple sections of heart examined, there were several tiny foci of acute myocardial fiber necrosis, characterized by sparce neutrophilic and round cell infiltrate (Fig. 4). Occasional myocardial fibers exhibited contraction band formation (insert in Fig. 3).

There was marked bilateral renal hypertrophy and hepatosplenomegaly. Otherwise the kidneys, liver, and spleen were normal. Gross and microscopic examination of the remaining organs revealed no significant pathologic abnormalities aside from pulmonary edema. Sections from the right buttock revealed acute and resolving hemorrhage of fibroadipose tissue, with neutrophilic reaction. There was no evidence of granuloma formation or foreign-body reaction.

Complete toxicological examination was negative for drugs of abuse, including ethanol. Postmortem vitreous fluid chemistry revealed values within normal limits for the postmortem interval and the circumstances of death: glucose, 9 mg/dL; urea nitrogen, 21 mg/dL; sodium, 138 meq/L; chloride, 129 meq/L; potassium, 9.9 meq/L; and calcium, 7.0 mg/dL. Blood submitted for a postmortem lipoprotein profile was found to be unsatisfactory for analysis.

Postmortem blood and urine were analyzed (by the Sports Medicine Drug Identification Laboratory, Department of Pathology, Indiana University School of Medicine) for the presence of anabolic steroids. Nandrolone metabolites, 19-nor-androsterone, 19-nor-etiocholanolone, and 19-nor-epiandrosterone, were detected in the urine at levels of 230, 52, and 32 ng/mL, respectively. Failure to identify nandrolone or its metabolites in the blood (detection limit, 5 ng/mL) was most probably due to tissue sequestration of the administered steroid esters, with rapid hydrolysis, distribution, and elimination following

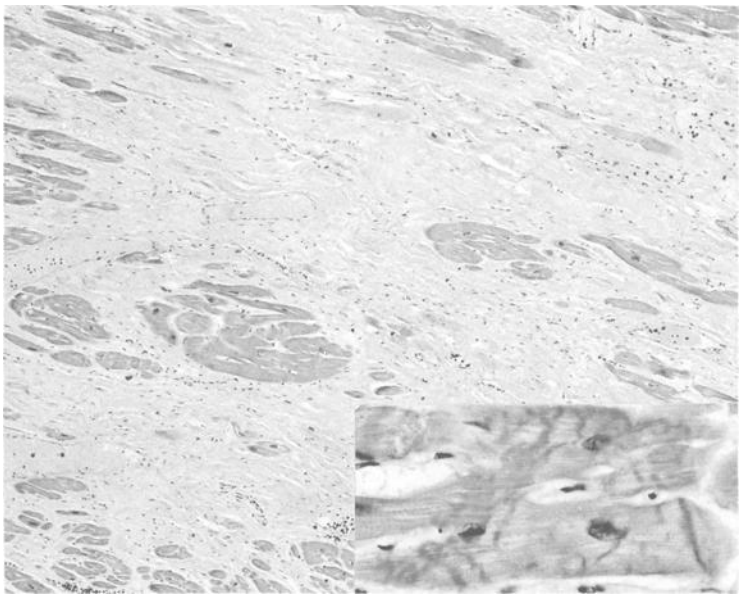

FIG. 3—*Microscopic section of the interventricular septum, showing myocardial fibrosis (×60). The inset demonstrates contraction band formation (hematoxylin and eosin stain, ×300).*

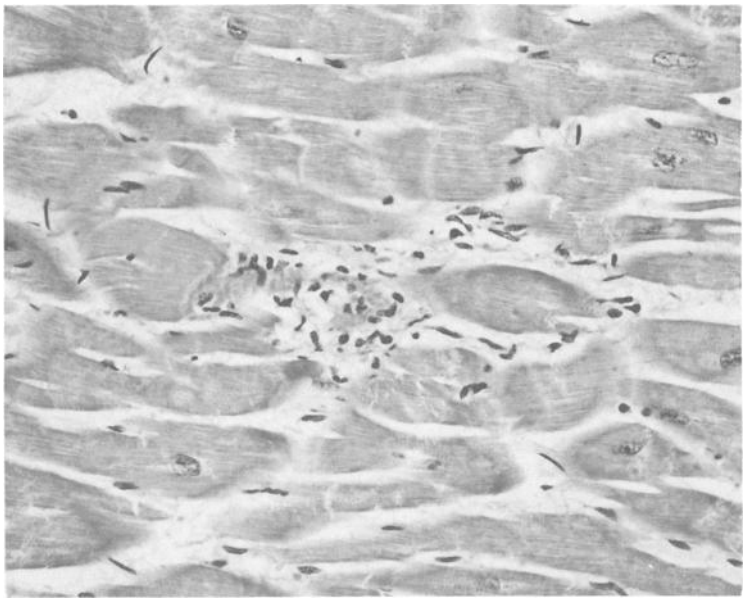

FIG. 4—*Microscopic section of the left ventricle demonstrating focal necrosis of myocardial fiber with mixed cellular inflammatory reaction (hematoxylin and eosin stain, ×300).*

their release into the general circulation. An extended interval prior to submitting the blood for testing raises the additional possibility of sample autolysis. To our knowledge, identification of anabolic steroids in postmortem blood has yet to be accomplished.

Discussion

It is our understanding that the cardiac changes described in deaths of athletes using steroids are primarily those of myocardial hypertrophy (E. K. Balraj, coroner, Cuyahoga County, Ohio, personal communication 1989). We also document myocardial hypertrophy in this case report. Given the remarkable growth-stimulating capacity of anabolic steroids [*3*], such a finding would be the expected result of this sort of pharmacological experimentation. Unfortunately, most available information on the subject is anecdotal in nature. The current literature does not extend to fatalities of steroid users in general or to deaths during exercise of these persons in particular. Moreover, the use by abusing athletes of an increasingly wide range of steroid products, taken in various forms, singly and in different temporal combinations and sequences [*3*], makes interpretation of pathologic findings extremely difficult. This is particularly the case given the lack of postmortem analyses to confirm or document specific steroid use in deaths of this type.

The myocardial findings of focal necrosis and regional fibrosis, noted in the case presented here, may conceivably represent an occult episode of viral or toxic myocarditis. Neither the minimal extent of the inflammatory reaction present, nor the distribution of such changes, nor the restriction of their involvement to the left ventricle and interventricular septum rules out this hypothesis. The history of long-term vigorous physical activity and athletic achievement in this case, without past signs or symptoms of serious cardiac disease, does not exclude myocarditis as a possible causative factor.

On the other hand, it is possible that the particular steroids used here or the specific

combination or sequence of those steroids injected may have caused myocardial growth in so rapid a fashion as to outgrow the blood supply of the fibers involved, generating ongoing piecemeal necrosis of myocardial fibers and the fibrotic changes noted. The marked cardiac and renal hypertrophy and hepatosplenomegaly present in this case would certainly lend credence to this hypothesis.

There are a number of additional steroid-related pathophysiological mechanisms that might have played a role in causing the cardiac pathology noted here. Elevated low-density lipoprotein (LDL) and reduced high-density lipoprotein (HDL) levels are a well-recognized complication of steroid usage, changes that have been associated with platelet hypercoagulability, increased vascular response to norepinephrine, and coronary vasospasm [*4–9*]. Elevated circulatory levels of catecholamines have been implicated in cases of myocarditis (by means of microvascular spasm) and chronic myocardial fibrosis [*10–11*]. Unfortunately, serum lipoprotein and catecholamine levels were not obtained in the present case.

McNutt et al. [*12*] describe a myocardial infarct, documented clinically, in a 330-lb (150-kg) weight lifter taking steroids orally and parenterally. The total cholesterol was 596 mg/dL (LDL, 513 mg/dL and HDL, 14 mg/dL) on admission, falling to a level of 283 mg/dL after 24 days without steroids. Cardiac catheterization demonstrated normal coronary arteries. Platelet aggregation levels, elevated at admission, were normal 12 days later.

Behrendt and Boffin [*13*] produced ultrastructural myocardial changes in rats after administering methandrostenolone (Dianabol) parenterally for three weeks. They liken their findings to those present in the early stages of cardiac failure.

Conclusions

Given the relative newness of steroid usage as an adjuvant to athletic performance, we think it is useful and prudent to document the pathologic and toxicological findings presented here, in the hope that other fatalities involving a history of steroid usage will be scrutinized with appropriate documentation in mind. While the use of anabolic androgenic steroids represents one explanation for a constellation of pathologic findings, unusual by their very nature in the face of a negative medical history and lack of restricted physical activity, there is, at this time, no proof of that association. Consequently, we would encourage the continued development of a suitable animal model to study the biology of steroid-related cardiac involvement. By these means, it may be possible to elucidate the pathogenesis, pathophysiology, and potential public health risk of this remarkable and, as yet, poorly understood, class of drugs of abuse.

Acknowledgment

The authors wish to thank Drs. Kurt B. Nolte and H. Wayne Carver II, of the Connecticut Medical Examiner's Office, for helping to facilitate the preparation of this paper.

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