

Ischemic Stroke in a Young Man Bodybuilder

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Abstract

Ischemic stroke among young people is an infrequent and rare entity. Despite the existence of a long list of etiologies, the progressive imposition of an ideal body image in society represents another cause of ischemic stroke. To this end, bodybuilding practices have been imposed on a large part of the adolescent sector with intense exercise routines accompanied by supplementation with anabolic-androgenic steroids (AAS). Abusers typically use up to 15 times the recommended medical doses of anabolic steroids. Nowadays, AAS are being used worldwide by millions of men, including those with no athletic ambitions, wishing to increase and improve their physical strength and appearance. AAS increase the risk of ischemic cerebral events through the interaction of certain variables whose common denominator is blood hypercoagulability. Consequently, in the presence of signs and symptoms of a stroke in a young person, the consumption of anabolic substances should be considered as a possible etiology.

Keywords: Ischemic stroke; Bodybuilder; Anabolic-androgenic steroid; AAS.

Introduction

Bodybuilding involves the consumption of a wide range of substances that contain unspecified components. A vast variety of compounds, mainly AAS, have been individualized. To achieve maximum performance, not only AAS but also diuretics, beta agonists, growth hormone, insulin and distilled water are used. AAS use is associated

with increased blood coagulability along several other factors. An important aspect to emphasize is that AAS consumption is mostly illegal, and the commercialization routes involved can be extremely dangerous. There are even reports of anabolic steroids for veterinary use, such as the current case. A supplementary practice to physical activities is chiropractic adjustment. This variable

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acquires relevance since it brings in the possibility of endothelial laceration. This, added to an increase in blood coagulability, strongly predisposes the development of thrombi and potentially systemic embolism, including cerebral circulation.

Clinical case

A 22-year-old patient presented to the emergency room because of headaches of moderate intensity for the last 12 hours. These were predominant on the right hemicranium. Progressive deterioration of the sensorium and left hemiparesis 1/5 at the time of admission were evidenced. Physical examination revealed severe acne and noticeable development of muscle mass. A computed tomography of the brain Figure 1 was performed and revealed a right sylvian infarction. Subsequently, a carotid doppler ultrasonography was performed, where an increase in the resistance index at the level of the right internal carotid artery, with signs of narrowing of the lumen were reported. Consequently, a cerebral angiography was performed, showing complete obstruction of

the right cervical internal carotid artery at the postbulbar level Figure 2 with neovascularization of the homonymous hemisphere through the anterior and posterior communicating artery of the circle of Willis Figure 3. The patient was not hypertensive, diabetic, smoked, nor reported any other history. For the past five years he has been consuming anabolic steroids, including those for veterinary medicine used on horses. Likewise, the patient also reported chiropractic adjustment on the spine as a complementary practice after training.

To study this case a complete immunological laboratory, anticardiolipin and lupus antibodies were requested, all of which came back negative. Urine screening for cocaine was performed, since its presence was detected in certain illegal formulas, while investigating the composition of veterinary anabolics used in large animals. This patient urinary sample tested positive for cocaine. Ischemic stroke was diagnosed in this young patient, in association with the consumption of AAS and cervical chiropractic adjustments.

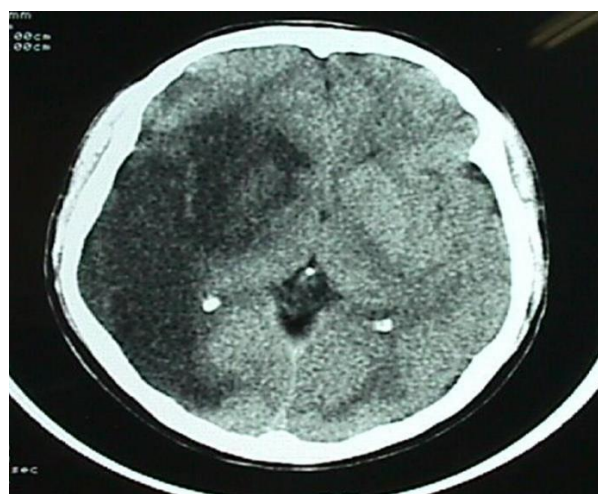


Figure 1: Computed tomography of the brain showing a hypodense image with loss of differentiation between gray and white matter that encompasses the entire right sylvian area. Mild edema with minimal mass effect is noted.

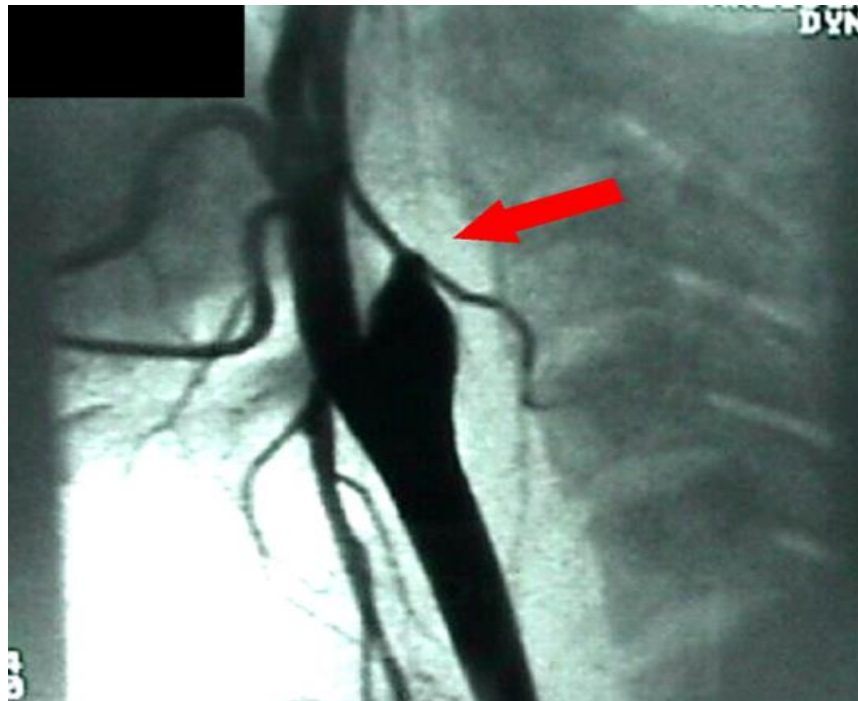


Figure 2: Cerebral angiography showing a complete obstruction of the postbulbar region of the right internal carotid artery.



Figure 3: Cerebral angiography showing neovascularization of the homonymous hemisphere compensating blood flow deprivation.

Discussion

The presence of a stroke in a young patient makes it necessary to list a variety of

etiologies, beyond those usually considered in older adults. Over 100 causes of this association have been described [1-4] and are summarized in Table 1.

Vascular
Cervicocephalic dissection Inflammatory vasculitis Infectious vasculitis Posterior reversible encephalopathy syndrome Moyamoya syndrome
Hematology
Prothrombotic disorders Hyper viscosity Coagulopathies Anemia Embolism Thrombosis
Cardiac
Valvular heart disease Cardiomyopathies Chagas diseases Anatomical defects
Others
Infective endocarditis with septic embolism Migraine Trauma Drugs Hypertension Fat embolism Neuroleptic malignant syndrome Neoplasia Lung diseases

Table 1: Stroke etiologies categorized by systems.

In the present case, the existence of previous pathological antecedents, the confirmation of the absence of the pathologies indicated in Table 1 and the clear exposure to the consumption of AAS and practice of chiropractic sessions, allows us to establish a cause-effect relationship between the development of ischemic stroke and the two variables recently mentioned. The first reported cases of stroke due to the use of anabolics by Mochizuki and Richter [5] and, from that date, the publications on the subject grew rapidly. AAS are widely used as

performance-enhancing drugs among young athletes as well as by bodybuilders to enhance the development of muscle mass [6-12].

Commonly abused AAS can be administered orally (oxymetholone, oxandrolone, methandrostenolone, stanozolol) or intravenously (nandrolone decanoate, nandrolone phenylpropionate, testosterone cypionate, and tetrahydrogestrinone). The use of AAS is associated with a wide list of side effects [6,13-16] and affects various organ systems Table 2.

Hormone system
Men <ul style="list-style-type: none"> • Infertility • Gynecomastia • Testicular atrophy • Baldness Woman <ul style="list-style-type: none"> • Clitoral hypertrophy • Hirsutism • Baldness
Musculoskeletal system
<ul style="list-style-type: none"> • Epiphyseal consolidation • Tendon ruptura
Cardiovascular system
<ul style="list-style-type: none"> • Prothrombotic disorders • Increase of LDL colessterol • Decrease of HDL choleterol • Hypertension • Infarction • Left ventricular hypertrophy
Liver
<ul style="list-style-type: none"> • Hepatocellular carcinoma • Peliosis hepatis
Kidney
<ul style="list-style-type: none"> • Renal failure
Skin
<ul style="list-style-type: none"> • Severe acne • Seborrheic dermatitis • Jaundice • Edema
Infections
<ul style="list-style-type: none"> • VIH/SIDA • Hepatitis
Psychiatric disorders
<ul style="list-style-type: none"> • Aggression • Mania • Delusion

Table 2: Collateral effects associated with AAS use.

It is necessary to emphasize that it has been widely demonstrated that there is a close relationship between the formation of thrombi and the consumption of this type of drugs. Experimental evidence suggests that

testosterone stimulates thrombus formation through at least three mechanisms:

- suppression of prostacyclin production (an inhibitor of platelet

aggregation) in arterial smooth muscle cells [17].

- increase in the platelet receptors density for thromboxane A₂ [18].
- increased fibrinogen [19].

Testosterone is converted by aromatization to estradiol, which could explain the thrombotic tendency observed with estrogens and gynecomastia as a commonly reported effect in AAS users.

Cases of cerebral infarction during the course of hormone replacement therapy in the context of hypogonadism [20] such as the development of superior sagittal sinus thrombosis secondary to androgen therapy as a treatment for aplastic anemia [21] and other reported cases of ischemic stroke in young patients who consume significant amounts of anabolic drugs in bodybuilding practices [5] support this statement.

Another aspect to note is that AAS abuse may cause cardiac ischemia by exaggerating oxygen demand at peak exercise, potentially precipitated by accelerated atherosclerosis from lipoprotein abnormalities over years of abuse [22].

The analysis of cocaine's physiopathology and action in the context of this clinical picture is of particular interest [23] Table 3. Cocaine induces a transient erythrocytosis, which can increase blood viscosity and decrease tissue oxygenation due to vasoconstriction. An increase in Von Willebrand factor, without compensatory changes in endogenous fibrinolysis, can stimulate platelet adhesion and aggregation along with intravascular thrombosis [23-25].

Sequential changes in blood counts secondary to intranasal and intravenous cocaine administration sufficient to produce changes in blood pressure and heart rate in humans have been compared. The increase in hemoglobin, hematocrit and white blood cell counts was quantitatively similar to the administration of two units of red blood cells [26], the use of erythropoietin for six weeks at a dose of 20 U/kg [27] or the chewing of cocaine leaves during exercise [28].

The erythrocytosis noted is similar to other rheological effects observed with the use of red blood cell transfusion doping or the use of erythropoietin, factors that constitute a substantial risk for the development of intravascular thrombosis during exercise [29,30]. Changes in blood viscosity may contribute to different cardiovascular events due to exercise [25].

Cocaine administration was shown to produce splenic vasoconstriction, with a flow reduction of 20% [31]. This contributes to the rapid expansion of circulating erythrocyte pool occurring in contrast to the gradual splenic emptying during exercise, which may play an important role in reported cases of intracapsular bleeding [32] and infarction [33] due to cocaine use.

Coronary vasospasm [34] and decreased cerebral circulation [35] induced by cocaine are linked to a significant increase in serum von Willebrand factor concentrations without modifications in fibrinolytic activity, fibrinogen, Ag TPA and Ag PAI-1, indicating the loss of the compensatory endogenous fibrinolysis increase [26].

Short-term effects	
Effect	Pathophysiology
Vascular effects	
Vasoconstriction	<ul style="list-style-type: none"> • Increased alfa-aderenergic stimulation • Increased endothelin synthesis • Decreased oxide nitric synthesis
Thrombosis and atherosclerosis	<ul style="list-style-type: none"> • Increased plasminogen activating factor inhibitor • Increased platelet activation and aggregability • Increased endothelial permeability • Induction of erythrocytosis • Increased von Willebrand factor
Cardiac effects	
Increased myocardial oxygen consumption with limited supply	<ul style="list-style-type: none"> • Increased heart rate • Increased blood pressure • Increased myocardial contractility
Left ventricular systolic dysfunction Left ventricular diastolic dysfunction	<ul style="list-style-type: none"> • Direct toxic effects of the drug • Altered intramyocyte calcium handling • Acid-base balance disorders Adulterants and infectious agents together with the drug
Long-term effects	
Left ventricular hypertrophy Left ventricular systolic dysfunction Dilated cardiomyopathy Myocardial depression	<ul style="list-style-type: none"> • Ischemia and/or infarction • Repetitive sympathetic stimulation bymnorepinephrine reuptake inhibition • Altered production of cytokines in endothelium and circulating erythrocytes • Changes in composition of myocardial collagen and myosin • Myocyte apoptosis

Table 3: Short and long-term effects of cocaine on cardiovascular parameters.

Aside from the factors recently analyzed, the role played by chiropractic sessions should be considered. Animal studies have shown that endothelial laceration secondary to trauma, as well as to experimental vessel ligations, is accompanied in 70 to 90% by arterial thrombi as a result of exposure of sub-endothelial thrombogenic factors to blood coagulation factors [37].

Individual effects of endothelial laceration with exposure of the underlying collagen, the increased coagulability secondary to the inhibition of antithrombotic substances induced by the consumption of anabolic substances, plus the increase of prothrombotic components without compensatory increase of fibrinolysis induced by cocaine, all together explain the

development of ischemic stroke in the patient of this presentation.

Conclusion

AAS use today has spread beyond bodybuilding practices. Although commercial availability of this type of drug is widely accepted, due to its cost, many adolescents obtain it from dubious sources. In particular case, the patient consumed AAS for human and veterinary use. The combination

of the prothrombotic effects of AAS, changes in cardiovascular parameters caused by cocaine and endothelial laceration due to chiropractic sessions led to the development of a sylvian ischemic stroke.

This case should alert physicians to consider these variables, usually not so specified in literature, as another possible etiology of stroke in young patients.

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