Venous Sinus Thrombosis Associated With Androgens in a Healthy Young Man

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**Background** Cerebral venous sinus thrombosis is rare and can be promoted by various conditions. We report the case of cerebral venous thrombosis in a patient using androgens.

**Case Description** A 31-year-old man using androgens for bodybuilding was admitted for headache and vomiting. He had cerebral venous sinus thrombosis, but extensive examinations did not reveal any known cause.

**Conclusions** We suggest that androgens may promote cerebral venous thrombosis. The mechanisms of venous thrombosis related to androgens may be platelet activation or an increase in coagulation factors. Because androgen use may be frequent and hidden in athletes, it may be an underestimated cause of cerebral venous sinus thrombosis in the young.

**Key Words** • drug abuse • platelet activation • sinus thrombosis • young adults

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Case Report

A 31-year-old Portuguese man was hospitalized 2 hours after sudden onset of severe headache and vomiting, which occurred during a bodybuilding workout. At admission, he was oriented but drowsy, had a right central facial paresis, and his visual acuity was diminished due to papilledema. Noncontrast computed tomography revealed a dense triangle sign of the superior sagittal sinus, and a hyperdensity of the straight sinus (Fig 1) and of the left transverse sinus. With contrast injection, the delta sign was present in the superior sagittal sinus, and a hyperdensity of the straight sinus (Fig 2), and in the left transverse sinus (Fig 2), and in the upper part of the jugular vein in T1- and T2-weighted images. At admission, serum luteinizing hormone was less than 0.65 mIU/mL (normal, 1 to 12 mIU/mL), follicle-stimulating hormone was less than 0.5 mIU/mL (normal, 1 to 8 mIU/mL), and plasma testosterone was 0.9 nmol/L (normal, 13 to 19 nmol/L). Chest x-ray, hemogram, coagulation studies, serum electrophoresis, erythrocyte sedimentation rate, ionogram, and cerebrospinal fluid were normal. There was no protein S, protein C, or antithrombin III deficiency. Homocystinemia, autoantibodies, and cryoglobulins were absent. Infection, cardiac disease, sarcoidosis, and neoplasia were not found. The patient's human leukocyte antigen type was HLA-B35. He had no personal or familial history of thromboembolic event, or personal history of any disease. He admitted that a physician had been giving him intramuscular injections of androgens: 25 mg testosterone twice a month, and 100 mg metolone (Primabolan) and 75 mg trembolone (Parabolan) weekly for 5 years. Anticoagulation therapy was initiated the first day of admission with intravenous heparin for 12 days and oral anticoagulation thereafter for 6 months. The patient completely recovered within 15 days. After 3 months, luteinizing hormone, follicle-stimulating hormone, and testosterone had returned to normal. He went back to bodybuilding training but stopped androgen administration. No recurrence of CVST occurred during the following year.

**Discussion** Although there is no evidence that androgens are thrombogenic in humans, arterial stroke and deep vein thrombosis have been linked with androgen abuse in athletes and with androgen treatments in patients suffering from aplastic anemia or hypogonadism. Animals pretreated with androgens have higher mortality rates, greater clot size, and lower arterial occlusion times than untreated controls in response to thrombotic stimuli. These effects may be mediated through platelet aggregation because androgens potentiate platelet aggregation both in vitro and in vivo through either increased production of thromboxane A2 or decreased production of prostacyclin. In athletes, androgen use increases platelet sensitivity to collagen. Androgens may also predispose to thrombosis by increasing collagen and other fibrous proteins in arterial vascular tissues and skin. Moreover, as danazol is used in
hemophilia to increase factors VIII and IX, androgens may raise the levels of coagulation factors.\textsuperscript{11}

Despite extensive evaluation, we failed to disclose a known cause of CVST in our patient. Therefore, we hypothesize that CVST may be caused by androgens. As far as we know, no case of CVST has been reported in a patient receiving androgens without underlying disease. Because the use of androgens may be frequent and hidden in athletes,\textsuperscript{6} the presence of central neurological symptoms in such a population requires the consideration of CVST, which may have a severe course without treatment. Moreover, androgen use may be an under-diagnosed cause of cerebral venous thrombosis in the young.

**References**


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