Cardiovascular complications and anabolic steroids

Anabolic steroids have become a popular drug among athletes and are known to have a multitude of pathological effects when administered in suprapharmacological doses. Sudden death due to right heart failure subsequent to venous thrombus formation in an athlete abusing anabolic steroids has not been previously reported. We are now reporting on the role of testosterone in coagulation and hope this will further direct attention to its probable role in the myocardial infarctions and strokes that occur in athletes using anabolic steroids.

This report involves a 26-year-old competitive bodybuilder who suffered a sudden death due to right heart failure subsequent to a bilateral pulmonary embolism from deep venous thrombus of lower extremities. The 136 kg, 182 cm, male bodybuilder of very large muscular proportions (body mass index = 40.8 kg m\(^{-2}\)) collapsed suddenly while moving furniture. The patient was transferred by paramedics to a local community hospital where he never recovered. Autopsy was performed at the Medical Examiners office and the cause of death was ruled pulmonary embolism from deep venous thrombosis and anabolic steroid use and had been reported that androgen receptors exist in the vessel tissue, on cardiac atrial and ventricular cells of primates. To date, the function of these receptors is unknown.

Myocardial infarctions, stroke and other thrombotic complications have been reported in athletes abusing anabolic steroids\(^1\). Therefore, with the majority of anabolic steroid cases being related to myocardial infarctions and stroke, it seems that the common denominator in all these cases is thrombus formation. The role of androgens in the complex coagulation system is far from being understood; however, this case points at the role of androgens in thrombus formation and subsequent death.

Interestingly, there is the possibility of androgen regulation of certain plasma coagulation factors. Protein S is an anticoagulant produced in hepatocytes and leydig cells of the testes. Protein S functions as a cofactor with Protein C in the inactivation of Factors Va and VIIIa. In addition, Protein S deficiency leads to a predisposition for venous thrombus\(^2\). A portion of Protein S is structurally homologous to the steroid binding domain of sex hormone-binding globulin (SHBG). SHBG is a steroid-binding protein that binds dihydrotestosterone, testosterone and estradiol. SHBG is positively regulated by oestrogens and negatively regulated by androgens.

Thus, with the administration of anabolic steroids, SHBG levels drop dramatically allowing more free (unbound), biologically active steroids in the system. If Protein S is regulated by sex steroids, it is plausible that aggregating agents in females. Castration of males markedly reduces their platelet sensitivity to aggregation, whereas ovariectomy elevated the platelet sensitivity in female rats. In vitro, androgens at physiological concentrations consistently stimulate platelet aggregation\(^3\). Androgens and other sex steroids are known to be absorbed at platelet membranes modifying their surface properties, inducing potential and permeability changes\(^4\). Androgens may potentiate platelet aggregation through increased production of arachidonic acid, a precursor to the potent platelet aggregator thromboxane A\(_2\) or, in aortic smooth muscle, decreased production of prostacyclin\(^5\). Recently, testosterone was shown to increase thromboxane A\(_2\) receptor density and responsiveness in rat aortas and platelets\(^6\). In addition, it has also been reported that androgen receptors exist in the vascular tissue, on cardiac atrial and ventricular cells of primates. To date, the function of these receptors is unknown.

References


Delayed occurrence of complete atrioventricular block after radiofrequency ablation of atrioventricular node reentrant tachycardia. Follow-up

The ability to cure atrioventricular (AV) nodal reentry with radio-
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frequency catheter ablation with preservation of antegrade conduction represents a significant clinical advance[1-3]. We report the follow-up of a delayed recurrent complete AV block after slow pathway ablation.

A 63-year-old woman with a 15-year history of symptomatic paroxysmal supraventricular tachycardia (3 to 4 tachycardias per week) was being treated with antiarrhythmic medication before being referred for ablation. Incremental pacing and extrastimulation were performed to confirm the presence of typical atrioventricular nodal reentry. The following criteria were sought to support the diagnosis of AV nodal reentry tachycardia: (1) discontinuous AV nodal function curve, defined as an increase of >50 ms in the A1 H2 interval, and a decrement of 10 ms in the A1 A2 interval; (2) tachycardia induction associated with a critical degree of prolongation of the AH interval and (3) atrial septal activation during tachycardia that was coincident with or closely (<50 ms) followed ventricular activation. Ventricular premature beats introduced during His bundle refractoriness failed to advance the subsequent atrial activation during the tachycardia.

Radiofrequency energy was delivered by a generator, HAT 200 (Dr Osypka, GmBh Medizintechnik). An electrode catheter with a large distal electrode was used to ablate the posterior site of the perinodal region. The target site was identified near the ostium of the coronary sinus where a discrete slow potential was recorded. Energy was delivered and adjusted automatically, corresponding to the set temperature of 70 °C for 30 s. During the application a junctional bradycardia was recorded. Immediately after application there was a sinus bradycardia and an increase in PR interval (0-24 s) followed by the abrupt occurrence of a 5 s transient complete AV block. The degree of AV block decreased progressively and a first-degree AV block was noted 22 s later. At the end of the procedure, the PR interval was 0-26 s; the cycle length during atrial pacing, at which the Wenckebach block occurred, was 400 ms. There was also a complete VA block. Tachycardia was not inducible.

One hour later, the PR interval was normal (0-16 s) (Fig. 1) and remained normal for 3 days without occurrence of AV block during continuous ECG monitoring. On the 4th day a complete AV block was recorded. The clinical tolerance was excellent and continuous monitoring was performed until the regression of the AV block, which was total 4 days later. There was no pacemaker implantation and the patient was discharged without medication. Five months later the patient remained free of tachycardia. Her ECG and 24 h Holter monitoring were normal.

Transient AV block occurring during catheter modification of the AV node complicated 0 to 5% of the slow pathway ablation and 5 to 10% of the fast pathway ablation[4], especially during catheter modification of the AV node using direct current energy, but no patient was reported to develop late complete AV block. Using radiofrequency energy Wathen et al.[5] reported six transient AV blocks out of 25 slow pathway ablations without late complete AV block during follow-up. Several other authors reported several cases of late occurrence (20 h up to 1 month) of late complete AV block after fast pathway ablation. Recently, Fenelon et al.[4] were the first to report the impairment in AV conduction 3 months after a successful slow pathway ablation.

In conclusion, careful follow-up of the patients who present with a transient complete AV block after slow pathway ablation is required but the significance of late recurrence of the AV block remains unknown. Regression of this block might be expected.

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Spontaneous restoration of sinus rhythm after cardiac surgery

Chronic atrial fibrillation is the most frequent arrhythmia in rheumatic valvular heart disease. This arrhythmia increases the risk of thromboembolism and impairs cardiac performance. Unless sophisticated surgical techniques are used to decrease atrial size[1,2], mitral valve substitution rarely eliminates chronic atrial fibrillation in patients with enlarged atria[3]. In addition, postoperative treatment with both quinidine[4] and direct-current shocking[5] have limited benefits when patients with mitral valve disease have long lasting atrial fibrillation and large atrial size.

A 53-year-old woman was admitted to our department with chronic heart failure in NYHA functional class III. Chronic rheumatic heart disease was diagnosed at the age of 35 and treatment with digoxin and diuretics was begun. The patient did fairly well until the age of 51 when atrial fibrillation was demonstrated on the Holter electrocardiogram and warfarin and was seen monthly. This report is the first to show spontaneous and stable conversion of atrial fibrillation to sinus rhythm in subjects with long-lasting atrial fibrillation. Six months after surgery, on a routine visit, the baseline electrocardiogram showed sinus rhythm with a heart rate of 88 beats.min⁻¹. Echocardiography showed the dimension of the left atrium to be increased from 50 mm to 680 mm (94° 13° 19° 19°). Transient atrioventricular block during radiofrequency ablation of atrioventricular node reentrant tachycardia, Eur J Cardiol 1997; 95: 75.

References


Pacemaker twiddler’s syndrome: delayed transection of permanent ventricular lead

A 74-year-old woman with a permanent ventricular-inhibited pacemaker implanted 1 year earlier was admitted because of dizziness and local muscle stimulation. A year before, she had undergone successful repair of a dissecting aneurysm of the ascending aorta. Appropriate placement of the lead in the right ventricle was confirmed by a chest radiograph. The implantation site was well healed without fluid accumulation.

On admission, the electrocardiogram demonstrated loss of ventricular capture. The chest X-ray revealed twisting and knotting with transection of the lead within the subcutaneous pocket and apical dislodgement. The lead impedance was found to be increased from 509 to 680 ohms. At reoperation, a tightly fitting fibrous capsule was found without fluid build-up. The terminal pin was retracted and removed from the header. Successful reimplantation of a bipolar silicone lead was performed after puncture of the left subclavian vein.