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Sociedad Venezolana de Farmacología Clínica y Terapéutica
Caracas, Venezuela

Disponible en: http://www.redalyc.org/articulo.oa?id=55919726005
Pathology of the testicle after long-term anabolic steroid treatment in a thoroughbred horse from Venezuela

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Recibido: 20/01/2011 Aceptado: 30/03/2011

Abstract

The aim of this study was to describe a case of pathology of the testicle after long-term anabolic steroid treatment in a Thoroughbred horse. Were study an equine Thoroughbread male of 5 years old in the National Race Track “La Rinconada” Caracas-Venezuela. The equine present history of lameness chronic and subfertility. Necropsy was performed and samples of testicle tissue were collected. The tissue samples were fixed in formalin and processed by conventional H&E techniques. Additionally, the special staining procedure of Tricromico de Gomory and Blue VonKossa were also carried out. Samples of blood and urine were recollected for toxicological by competitive ELISA, specific Boldenone ELISA Kit (Cat.N.FA650 TECNA); Nandrolona: Nortestosterona ELISA Kit (BIO K 208 BIO Diagnostic) y Dexametasona: Dexamethasone ELISA Kit (101519 BIKITS).

Introduction

The disgenesis reproductive is a severed problem in horse’s athletes. Atrophy of seminiferous tubules and interstitial fibrosis are frequently observed in aged horses5,6,7,8. The testicular degeneration is caused by processes such as cryptorchidism and the inflammatory processes5. Many pathological changes such as degeneration of spermatogonia and Sertoli cell, and atrophy and fibrosis of seminiferous tubules with hyalinosis have been reported6. Investigations carriedout during the last years indicate that COX2 and PGs may play key roles in testicular patho-physiology7. Anabolic steroids (ABS) are illegally used for anabolic purposes in humans, horses and cattle4. The aim of this study was to describe a case of pathology of the testicle after long-term anabolic steroid treatment in a Thoroughbred horse.

Materials and Methods

Were study a equine Thoroughbred male of 5 years old in the National Race Track “La Rinconada” Caracas-Venezuela. The equine present history of lameness chronic and subfertility. Necropsy was performed and samples of testicle tissue were collected. For histological examination, samples were fixed in 10% formalin, embedded in paraffin and cut to 5-μm sections and processed by conventional H&E techniques1,2. Additionally, the special staining procedure of Tricromico de Gomory and Blue VonKossa were also carried out1,2. Samples of blood and urine were recollected for toxicological by competitive ELISA, specific Boldenone ELISA Kit (Cat.N.FA650 TECNA); Nandrolona: Nortestosterona ELISA Kit (BIO K 208 BIO Diagnostic) y Dexametasona: Dexamethasone ELISA Kit (101519 BIKITS)1.

Results

The right testis showed signs of cryptorchidism. The size of the left atrophied testis was 6 x 2.5 x 2 cm. The scrotum of this horse had swollen to a thickness of approximately 8 cm. and adhered to the tunica vaginalis. A sagittal section of the testes...
revealed fibrotic interstitium. Histologically the seminiferous tubules consisted of vacuolated, severed atrophied and scattered Leydig cells and fibrosis interstitium severed (Figure 1). Proliferated spindle cells with fibrogenesis positive a Tricromico de Gomory (+) and Vonkossa (-) (Figure 2). Leydig cells and collagen fibers had increased. There was no spermatic cell in the seminiferous tubules and only Sertoli cells with deposition of brown granules of lipofuscin remained. Toxicological studies in blood and urine samples allowed the detection of boldenona 18 ng/ml and dexamethasone generic 7ng/ml.

### Table 1.- Results of toxicological analysis by ELISA competitive of samples of blood and urine in an equine studied.

<table>
<thead>
<tr>
<th>Sample</th>
<th>Boldenona</th>
<th>Dexamethasone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood 60ml</td>
<td>16ng/ml</td>
<td>5ng/ml</td>
</tr>
<tr>
<td>Urine 300ml</td>
<td>18ng/ml</td>
<td>7ng/ml</td>
</tr>
</tbody>
</table>

Discussion

Testicular fibrosis is considered to be a pathway leading to hypospermatogenesis. The left testis frequently lags as much as 6 cm behind the right testis in descent, so is more likely to be locked in the abdominal cavity by fibrosis of the internal ring after birth.

Prostaglandins (PGs) are derived from arachidonic acid by the action of the constitutively expressed cyclooxygenase isoenzyme type 1 (COX1) and the inducible isoenzyme type 2 (COX2). Reasons for testicular expression of COX2 and consequences of its actions are not fully known, but cellular and ex-vivo studies provided insights. Mast cells and MAC appear to be involved. Both are significantly increased in the testis of infertile men and correlate with the degree of fibrosis of the tubular wall, a change typically associated with impaired spermatogenesis. The major mast cell product, tryptase, through protease-activated receptors (PAR2) increased the expression of COX2. Subsequently, one of the PGs produced, 15d-PGJ2, acting via the nuclear peroxisome proliferator-activated receptor γ (PPARγ) was found to induce human fibroblast proliferation. Studies have also shown that all components of the signalling pathway (tryptase-positivemast cells, COX2 and PPARγ) are present in testes of infertile men and could be responsible for human testicular fibrosis. Testosterone induces COX2 expression and PGF2α production, but COX2/PGF2α inhibits StAR and 17α-HSD expression and consequently, testosterone production thereby setting a brake on testicular steroidogenesis. This regulatory loop might be of relevance in physiological conditions and/or pathological states. These myoid cells transformed into myofibroblasts. The changes are interpreted as evidence of injured structure and function of the lamina propria and basal lamina and may explain the functional decline of the blood-testis barrier. Myoid cells may play an important role in the progression of testicular fibrosis. Plasma concentrations of endogenous 19-nortestosterone (nandrolone; NA) from racing and nonracing males were 50.2+/-5.5 and 71.8+/-4.6 pg/mL, respectively.
The lesions resembled the effects already observed after the administration of androgen hormones to cattle. Main findings were represented by prostate hypersecretion, increased rate of apoptotic cells and decreased rate of Ki67 positive cells in the germ cell line of treated animals, particularly in boldione group and finally some new features like hypertrophy of the prostate urothelial cells. To conclude we reported a case of pathology of the testicle after long-term anabolic steroid treatment in a Thoroughbread horse.

References


