Unusual Presentation of Cortical Venous Sinus Thrombosis in a Body Builder Using Anabolic Steroids: A Case Study

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Abstract: There are only few cases of patient developing cerebral venous thrombosis after androgen therapy. We represent here a 22 yr young man who developed cortical venous thrombosis after using androgens to increase muscle mass. He was hospitalised for headache and generalised tonic clonic seizures. He recovered well with low molecular weight heparin and antiepileptic medications. Brain MRI and MRV showed superior sagittal and transverse sinus thrombosis and extensive investigations did not reveal any known cause. There are many causes of venous sinus thrombosis but use of anabolic steroids occurs to be a rare cause. Conclusion: We suggested that androgen was the predisposing factor in our patient. Androgens may increase coagulation factors or platelet activity and cause venous thrombosis. As athletes may hide using androgens, it should be considered as a predisposing factor for thrombotic events in such patients. Careful detailed drug history should be taken in these patients.

Keywords: Cortical Venous Sinus Thrombosis, Headache, Young Body Builder, Anabolic Steroids, Hypercoagulability.

1. Introduction

Cerebral venous sinus thrombosis (CVST) is a disease with a wide spectrum of non specific clinical signs and symptoms, including headache, focal neurological deficits, seizures and coma.

While studying stroke in the young, Indian studies revealed that CVT constitutes 10- 15% of stroke in the young individuals.⁴, ⁵, ⁶

The clinical outcome is highly variable; patients may recover completely or may develop severe and lasting neurological deficits⁷.

There are various factors responsible for this disease but the most common predisposing factors are pregnancy, puerperium, contraceptive pills, coagulopathies and intracranial infections⁸.

There are very few studies done on patients with CVST after androgen therapy. We present here a young bodybuilder man who developed CVST with abusing androgens for increasing muscle mass.

2. Case Report

28 year male body builder by occupation presented in casualty with complaint of headache and generalised tonic clonic convulsion and confusion.

On clinical examination patient was well nourished .Patient was confused ,afebrile with pulse rate of 88/min regular in rhythm. Blood pressure was 130/80mm of Hg. Respiratory system, cardiovascular system and per abdomen examination was within normal limits. Central nervous system examination revealed Glass Gow Coma scale 13/15, all reflex were intact, normal tone and power both plantar were flexor; pupil were normal size and bilaterally reacting to light, fundus examination revealed early papilloedema..

However papilloedema resolved gradually by intravenous mannitol (0.5g/kg/day)8 hourly for 5 days. As patient was in postictal phase after regaining conciousness he gave history of anabolic steroid (decadurabolin-nandrolone decanoate)10
intramuscular injection per week 5 years back taken for body building purpose.

MR with MR venogram study revealed cortical venous thrombosis of sagittal and right sigmoid sinus. Details of investigation done during hospitalization are described in the Table 1 below. Patient was treated as a case of dural venous sinus thrombosis with low molecular weight heparin (0.6 subcutaneous twice a day for 5 days), overlapped with warfarin till desire INR was achieved and antiepileptic medication in the form of intravenous levitiracetam.

Patient made good recovery after LMWH and antiepileptic treatment and was discharged home after 15 days on oral warfarin therapy and antiepileptic medications and to be followed up after 15 days. INR repeated after 15 days of follow up was 2.8 & consecutively 3.2 on next visit.

3. Discussion

There are a limited case records of patients of cortical venous sinus thrombosis secondary to androgen induced therapy. The anabolic activity of testosterone and its derivatives is primarily manifested in its myotrophic actions which result in greater muscle mass and strength. This has led to widespread use of androgenic anabolic steroids by athletes at all levels.

Seizures – unifocal or multifocal, are present in 50% cases. They may be localized at the onset but may later become generalized. Rarely they may persist after an acute phase is over. Their early appearance is the hallmark of bad prognosis. 

Kinetin autism of short duration manifesting during recovery phase, have been reported. Aetiological causes of CSVT can be divided into 3 subtypes.


b) Changes in vessel wall: Malignancy Infections: local-chronic- otitis media, nasolabial and/or facial infections, pyogenic meningitis – Systemic, e.g., gram negative septicamia, fungal infections etc.

c) Changes in blood flow/viscosity: Malnutrition, Dehydration, Congestive heart failure, Hyperviscosity syndrome.

Nandrolone decanoate is a synthetic anabolic steroid (structure given below). 19-Norandrosterone was identified as a trace contaminant in commercial preparations of androstenedione, which until 2004 was available without a prescription as a dietary supplement in the U.S. In focus on homeostasis system the most important factors under testosterone regulation are fibrinogen, Plasminogen activator inhibitor-1 (PAI – 1) and platelet aggregability.

The current data indicate that testosterone lowers fibrinogen and PAI – 1, however these anticoagulatory and profibrinolytic may be opposed by proaggregatory effects on platelets because high dosages of androgens were found to decrease cyclooxygenase activity and thereby increase platelet functions.

Superior sagittal sinus is the commonest sinus to be involved in aseptic CVT. Involvement of superior sagittal sinus accounts for about 72% of the affected cases, lateral sinus(combined) is involved in about 70% of cases, and straight sinus being involved in about 13% of individuals.

Pathological findings observed in central nervous system as a result of CVT are determined by a) Underlying Disease pathology b) nature of sinus/ cerebral vein involved; c) interval between the onset and pathological examination.

Cortical vein thrombosis usually presents as a cord like swelling with minimal or absent haemorrhagic infarction of the brain. This discrepancy has been explained on the presence of frequent intercommunications between various cortical veins and sinuses.

In case of superior sagittal sinus thrombosis, sinus is distended and appears blue. Cortical veins are also swollen and may rupture at some places giving rise to haemorrhagic infarction and even intercerebral haemorrhage.

In an occasional case, haemorrhagic infarction may appear on the other side due to occlusion of opposite cortical vein (parasagittal). In deep cerebral vein thrombosis, white matter may be involved, eg., basal ganglia, thalamus, etc. As time passes, thrombosis gets recanalised, organized and may even disappear in majority of cases.

4. Structure of Nandrolone Decanoate

![Structure of Nandrolone Decanoate](image)

<table>
<thead>
<tr>
<th>Haemoglobin (gm%)</th>
<th>Total leucocyte count (cu.mm)</th>
<th>PLATELET(cu.mm)</th>
<th>BLOOD UREA(mG/dL)</th>
<th>CREATININE(mG/dL)</th>
<th>SODIUM(Meq/L)</th>
<th>POTASSIUM(Meq/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>13/7/2015</td>
<td>15.5</td>
<td>11410</td>
<td>197000</td>
<td>18</td>
<td>1.2</td>
<td>138</td>
</tr>
<tr>
<td>14/7/2015</td>
<td>15/7/2015</td>
<td>17/7/2015</td>
<td>19/7/2015</td>
<td>26/7/2015</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 1
Photographic Images of the Patient Suffering From CSVT with Anabolic Steroid Use

Table 2 (MRI & MRV)

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>INR</td>
<td>1.01, 1.02, 1.33, 2.8, 3.2</td>
</tr>
<tr>
<td>Antithrombin 3</td>
<td>110</td>
</tr>
<tr>
<td>Homocystine</td>
<td>29.4</td>
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<tr>
<td>Protein-C</td>
<td>28</td>
</tr>
<tr>
<td>Protein-S</td>
<td>100</td>
</tr>
<tr>
<td>APA IgG (U/ml)</td>
<td>2.15</td>
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<tr>
<td>APA IgM (U/ml)</td>
<td>4.090</td>
</tr>
<tr>
<td>ANA</td>
<td>0.3</td>
</tr>
<tr>
<td>Total Bilirubin (IU/ml)</td>
<td>1.7</td>
</tr>
<tr>
<td>Direct Bilirubin(IU/ml)</td>
<td>0.3</td>
</tr>
<tr>
<td>SGOT (IU/ml)</td>
<td>381</td>
</tr>
<tr>
<td>SGPT (IU/ml)</td>
<td>84</td>
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<tr>
<td>Alkaline Phosphatase (IU/ml)</td>
<td>84</td>
</tr>
<tr>
<td>Total Protein (gms/dl)</td>
<td>6.3</td>
</tr>
<tr>
<td>Total Albumin(gms/dl)</td>
<td>3.5</td>
</tr>
<tr>
<td>Hba1c</td>
<td>6.8</td>
</tr>
<tr>
<td>Fasting BSL (mg/dl)</td>
<td>131</td>
</tr>
<tr>
<td>Postprandial BSL(mg/dl)</td>
<td>113</td>
</tr>
</tbody>
</table>

Figure 1 & 2: Shows multiple areas of restricted diffusion seen in bilateral posterior and multiple foci of blooming are seen in these images. Figure 3 & 4: Shows that there is loss of flow that is seen in right sigmoid sinus and superior sagittal sinus, hypoplastic right transverse sinus can also be seen.
References


