



Severe Multi-organ Involvement due to Polysubstance Abuse Including Anabolic Steroids

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ABSTRACT

Polysubstance abuse is an established rampant problem in the current young generation with advent of easy availability and uncontrolled use of emerging supplements for bodybuilding. Our case report depicts a rapidly progressive multi-organ dysfunction with acute presentation due to polysubstance abuse. This brawny physique young male was admitted to the intensive care unit (ICU) with unusual complaints of shooting pain, paresthesia, and weakness of both lower limbs but massively altered blood parameters. On careful and detailed history taking he was found to be known to consume cannabidiol oil, anabolic steroids, amino acid supplements, cocaine, and alcohol.

The key to this case was to keep an eye on suspicious ways of presentation, detailed history, and careful examination. Emphasis on questions the surrounding substance abuse should be an integral part of the history taking process. His organ failures resolved with appropriate treatment during ICU stay to be stepped down toward and later discharged home.

Keywords: Anabolic steroids, Drug abuse (including addiction), Drugs in sport, Toxicology.

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CASE DESCRIPTION

A 33-year-old male was brought in to the hospital following a collapse. He had a 1 day history of central back pain which started insidiously overnight, soon followed by shooting pain, weakness, and then paresthesia of both legs. He fell down after trying to mobilize and was noted by the ambulance to be sweating profusely with a blood pressure of 80/30 mm Hg. He recalled multiple episodes of vomiting in the past 2 days and not passed any urine since this morning despite having drunk 3 L of fluids. He had no comorbidities.

He is a smoker and initially denied any drug history, however, further questioning and collateral history revealed use of cannabidiol oil twice a week, cocaine use socially, binge drinking 2–3 times a week of 700 mL to more than 1 L of vodka each time. He “goes hard” at the gym 2–3 times a week with excessive use of creatine supplements and anabolic steroids. He has been using testosterone propionate and testosterone enanthate, however, denies any use for the past 6 months. His usual medications were promethazine, co-codamol, and sertraline, which there were concerns of him routinely taking excessive amounts of.

On examination, he had a capillary refill time of 4 seconds, tenderness on palpation of the right flank, and right iliac fossa. On neurology examination, he had global lower limb weakness with Medical Research Council’s power assessment varying between 2 and 4 out of 5 in a nonspecific pattern. Rest of his neurological examination of lower limbs was normal with preserved sensations and normal deep tendons reflexes. His respiratory and cardiovascular system examination did not reveal any major abnormality.

INVESTIGATIONS

His blood parameters on admission are shown in [Table 1](#).

He was in acute liver injury with high enzymes and normal protein levels. Other blood showed hyper-inflammatory response of troponin I, creatine kinase, and lactate dehydrogenase. His admission paracetamol level was less than 10 mg/L. Urine toxicology screen on admission positive for morphine, codeine, and cocaine

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metabolites. Urine creatinine 3.2 mmol/L and alcohol urine level <100 mL/L.

DIFFERENTIAL DIAGNOSIS

There were multiple differentials for this presentation and blood parameters like rhabdomyolysis induced renal failure, chronic renal failure, or acute on chronic liver failure. Also, alanine transaminase rise was given a differential of skeletal muscle breakdown after rigorous workout. The presentation of shooting pain, paresthesia, and weakness of both lower limbs raised suspicion of Guillain–Barré syndrome, multiple sclerosis, myopathies, space-occupying lesions in spinal cord but the neurological examination was normal except for inconsistent motor power which improved rapidly after admission to ICU.

TREATMENT

This patient was managed with advice from the regional liver unit. He was given N-acetylcysteine, antimicrobial cover with Piperacillin/Tazobactam and fluconazole. He was oliguric with worsening metabolic acidosis despite medical treatment and therefore needed admission to the ICU and underwent 48 hours continuous veno-venous hemofiltration. He was regularly monitored with Clinical

Table 1: Blood tests and further investigations on admission, compared to results on discharge 2 weeks following

	<i>Results on admission</i>	<i>Results on discharge</i>
White blood cell count	20 × 10 ⁹ /L	5.7 × 10 ⁹ /L
Hemoglobin level	148 g/L	147 g/L
Platelet count	155 × 10 ⁹ /L	378 × 10 ⁹ /L
Sodium	128 mmol/L	142 mmol/L
Potassium	6.5 mmol/L	4.5 mmol/L
Creatinine	448 µmol/L	125 µmol/L
Urea	10.0 mmol/L	5.1 mmol/L
eGFR	13 mL/min/1.73 m ²	58 mL/min/1.73 m ²
Phosphate	1.40 mmol/L	
Adjusted calcium	1.28 mmol/L	
Calcium total 3.16 × 2.19 × 2.15–2.51 mmol/L		
Calcium ionized 1.56 × 1.37 × 1.17–1.34 mmol/L		
Magnesium	0.62 mmol/L	
Alanine transaminase	9915 U/L	106 U/L
Aspartate transaminase	6888 U/L	59 U/L
Gamma-glutamyltransferase	167 U/L	
Bilirubin	23 µmol/L	10 µmol/L
Albumin level	40 g/L	39 g/L
INR	1.54	
Prothrombin time	18.3 seconds	
Troponin	6234 ng/L	
Creatine kinase	10,263 U/L	
Lactate dehydrogenase	421 U/L	
pH	7.195	
Base excess	−10.2 mmol/L	
Lactate	5.2 mmol/L	
Glucose	11.5 mmol/L	
Paracetamol level	<10 mg/L	
Urine toxicology	Positive for morphine, cocaine metabolites. Negative for marijuana	
Urine creatinine	3.2 mmol/L	
Urine alcohol level	<100 mL/L	
Electrocardiography	S–T depression in V4–V6	
Echocardiogram	No abnormalities	
Kidney and liver immunology and virology screen	No significant findings	
Kidney ultrasound	Poor corticomedullary differentiation with hyperechoic cortices consistent with AKI. Noted to have 5 cm depth right pleural effusion.	

eGFR, estimated glomerular filtration rate; INR, international normalized ratio

Institute Withdrawal Assessment for Alcohol score and managed for alcohol withdrawal accordingly.

normal (as shown in Table 1). He was followed by the renal team as an outpatient.

OUTCOME AND FOLLOW-UP

On discharge, 2 weeks after admission, his muscle power returned was normal with some mild right ankle pain. His renal function improved, trending upwards with liver function tests returning to

DISCUSSION

Anabolic steroids are synthetic compounds that bind to androgen receptors and exert anabolic effects and increase muscle mass.¹ In addition, they are effective as performance-supporting agents

and have been “off label” misused by athletes for many years. Many factors determine the severity and frequency of the anabolic steroids side effects. These factors are drug formula, administration route, dose, period of use, and patient response.¹ Users who are targeting appearance purposes may use anabolic steroids at higher dosage with no cycling off which causes substance dependence disorder. One review has demonstrated 19 deaths in published case reports from 1990 to 2012 related to misuse of synthetic anabolic steroids; however, many of these also used other drugs, making it difficult to confirm that anabolic steroids use has caused these deaths.²

Exaggerated muscle load together with anabolic steroids abuse may result in rupture of muscles tendons especially in the points of insertions due to alterations in collagen fibers structure which show up as an increase in creatine kinase which leads towards rhabdomyolysis, similar to our case presentation.³ Anabolic steroids have been involved in different forms of hepatic injury mainly an initial temporary increase in liver enzymes.⁴

The cannabis group has two well-described cannabinoids—tetrahydrocannabinol, which accounts for most of the neurological intoxication and cannabidiol, which our presenting patient took in large amounts, is not a psychoactive component but a known CYP450 enzyme family inhibitor which is important in this context as it can increase the concentrations of co-administered metabolized drugs.⁵

Regarding heroin, different central nervous system (CNS) diseases have been documented in heroin addiction such as stroke, polyradiculoneuropathy, and acute inflammatory demyelinating mononeuropathy.⁶ In most cases reports of heroin—induced myelopathy, the patients showed a similar presentation to our patient, that is, with spinal shock in a similar fashion as seen in traumatic spinal cord injury with flaccid paralysis but with absent deep tendon reflexes, urinary retention, and diminished rectal tone.⁷

In the literature review, we found some studies that reported multi-organ failure related to polysubstance abuse, in particular synthetic anabolic steroids, similar to our case presentation. Bass and Linz reported a patient on regular cannabidiol gummies developed neurological manifestations with low glasgow coma scale (GCS) and later cardiovascular and respiratory compromise which needed IV fluids and oxygen support. This case may correlate to the neurological presentation in our case report.⁸

Acute kidney injury (AKI) occurs in 33% of episodes of rhabdomyolysis and is a common presentation amongst fitness enthusiasts. However, in published reports, the CK levels were substantially higher than in our case.^{9,10} Creatine supplements increase the risk of dehydration from intravascular volume depletion which is a risk factor for rhabdomyolysis.

Another interesting case about anabolic steroids in a middle-aged patient published by Unai et al. demonstrated a similar presentation to our patient who developed AKI with supraventricular tachycardia (SVT) and acute respiratory distress syndrome (ARDS) secondary to anabolic steroids abuse for weightlifting enhancement that required extra-corporeal membrane oxygenation (ECMO) support with continuous veno-venous hemofiltration (CVVH) and catheter ablation for SVT treatment.¹¹

Garg published a case series which demonstrated the first patient with possible rhabdomyolysis secondary to vigorous gymnastics exercise who developed AKI; second patient, a weightlifter, presented with acute pancreatitis due to anabolic steroids and protein supplements abuse and the third patient

with abnormal jerky movements and muscle spasms that led to respiratory failure requiring ventilation for 6 weeks. These presentations may explain the detrimental liver and renal functions in our case.¹⁰

LEARNING POINTS/TAKE HOME MESSAGES

- Polysubstance abuse is becoming a very rampant presenting scenario nowadays.
- A thorough and detailed history taking and careful clinical examination are the basic keys for narrowing down on the differential diagnosis.
- This kind of hyper-inflammatory response due to polysubstance abuse should ideally be managed in ICU with appropriate organ support to improve outcome.
- Amino acid as well as anabolic supplements may lead to abnormal functioning of many organs, which could be fatal in some instances. This mandates worldwide and concerted efforts to educate the public, especially the youth, about the dangers of these increasingly abused drugs.

PATIENT'S PERSPECTIVE

I recall getting admitted to ICU for my kidney problems for few days and remember feeling anxious and irritated. I workout very heavily and also take a lot of steroids, creatinine supplements, and banana in protein shakes. This event happened after my 3 day vigorous workout regime and I was feeling very lethargic and fatigued, I did not drink enough water the following day and started having tummy ache and vomiting. In the next 2 days, I started having leg cramps and my urine was becoming very dark and less in amount. The moment my legs started becoming numb, I was very dizzy. From that time, I cannot remember that much till admission to ICU. I am in a good shape now and resolve to stop taking heavy supplements for bodybuilding. I am thankful to the hospital team for taking care of me during this period. I was happy that I came through it.

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