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Unusual association of diseases/symptoms

The development of focal segmental glomerulosclerosis secondary to anabolic steroid abuse

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Summary

The authors present the case of a patient who presented to the nephrology department of a district general hospital with end-stage renal failure. He presented with malignant hypertension and symptoms and signs of uraemia. He also gave a history of prior abuse of anabolic steroids over a number of years. Renal biopsy was performed and the findings were in keeping with a diagnosis of advanced focal segmental glomerulosclerosis (FSGS). The patient went on to require renal replacement therapy within weeks of presentation. The authors suggest that anabolic steroid abuse is a direct cause of FSGS. People with raised body mass index are known to be at increased risk of developing this condition, due to increased haemodynamic stress on the glomeruli, with subsequent development of sclerosis. However, the authors believe that anabolic steroid abuse may be an independent risk factor, and that anabolic steroids have a direct nephrotoxic effect that leads to a more advanced initial presentation with rapid decline in renal function.

BACKGROUND

Anabolic steroid abuse is becoming increasingly common, with one study in Sweden finding that over 75% of bodybuilders admitted to regular use.[1] A number of adverse health effects secondary to taking anabolic steroids are well-recognised. However, the link with renal injury, and in particular focal segmental glomerulosclerosis (FSGS), is less well-recognised.

FSGS is a condition in which there is focal scarring of some glomeruli within the kidneys. It is observed in approximately 10% of renal biopsies that are performed for the evaluation of proteinuria. The majority of FSGS is primary. Secondary FSGS is associated with hyperfiltration from increased body mass or reduced renal mass, illicit drug use and HIV infection. Most patients with FSGS progress to end-stage renal failure (ESRF) within 6 to 8 years

CASE PRESENTATION

The patient was a 38-year-old male security guard who complained of a 4-day history of nausea and vomiting associated with abdominal pain and breathlessness on exertion. He also described generally feeling unwell over the preceding month. On further questioning, the patient, who had an interest in bodybuilding, admitted to regular use of anabolic steroids since the age of 18, claiming to have stopped for a year or so prior to presentation. This consisted of both oral and intramuscular routes of administration, at a range of doses. On examination, the patient was noted to have an extremely muscular physique with a body mass index (BMI) of 34.2. His blood pressure on admission was 225/140 mm Hg, and he was tachycardic with a heart rate of 110 beats per min. Examination of the chest and abdomen was unremarkable.

INVESTIGATIONS

The patient was found on admission to have a creatinine of 1797 µmol/l, with a urea of 55.2 mmol/l. He was also severely anaemic with a Hb of 6.0 g/l. Renal ultrasound scanning showed shrunken kidneys with hyperechogenicity of the parenchyma, indicative of intrinsic renal parenchymal disease.

Renal biopsy showed marked proliferation of both mesangium and cells in keeping with a diagnosis of FSGS. There were no features of collapsing FSGS. The autoimmune screen was negative and virology for hepatitis B and C and HIV was also negative.

DIFFERENTIAL DIAGNOSIS

Another factor that could account or contribute to the development of renal impairment in these patients is their high protein diets. As with many people who abuse anabolic steroids, the patient admitted to a diet that was extremely high in protein, in order to achieve greater muscle bulk. The natural response to an increase in nitrogenous waste is increased renal blood flow and chronic hyperfiltration may accelerate progression to glomerulosclerosis.

TREATMENT

The patient was initially stabilised with a labetalol infusion for blood pressure control, and also required urgent blood transfusion. He was started on haemodialysis within a period of 1 week from presentation. He then moved on to continuous ambulatory peritoneal dialysis (CAPD) after the initial presentation.

OUTCOME AND FOLLOW-UP

The patient remains stable on CAPD, and no longer takes anabolic steroids. He is currently on the waiting list for renal transplantation.

DISCUSSION

We suggest that this patient developed secondary FSGS as a direct result of abusing anabolic steroids and subsequently progressed to ESRF. There is little in the literature of the use of anabolic steroids and the development of FSGS, with only one study by Herlitz *et al* identified, looking at this association.[2] However it is known that obesity is a cause of secondary FSGS and we postulate that those with a raised BMI due to increased lean muscle mass are also at increased risk. It is thought that in obese patients, FSGS develops as an adaptive process, with a greater body mass leading to glomerular hyperfiltration, which in time leads to mechanical strain and scarring.

However in patients known to abuse anabolic steroids we believe there is an additional process occurring. From the study by Herlitz *et al*., the patients with FSGS thought to be due to anabolic steroid use, had a higher incidence of nephrotic syndrome at presentation and more advanced histological evidence of glomerulosclerosis, than other patients with secondary FSGS. This is in keeping with the advanced biopsy findings in our patient, and the rapid progression to ESRF requiring dialysis.

We believe that anabolic steroids have a direct nephrotoxic effect, in addition to the mechanical stresses that occur in patients with a raised BMI, and that these factors contribute to the development of FSGS.

Learning points

- Anabolic steroid abuse is an increasingly common problem.
- There may be a direct causative relationship behind the abuse of anabolic steroids and development of FSGS and ESRF.
- The mechanism behind this may involve direct nephrotoxicity, as well as increased stresses placed on glomeruli due to hyperfiltration.

Competing interests None.

Patient consent Obtained.

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