Bodybuilders pneumonia

Dear Editor,

A 22-year-old male, previously healthy, nonsmoker engineering student, presented with shortness of breath, anorexia, and generalized weakness over the past 2 weeks. There was no history of fever, sore throat, cough, or coryza before this illness. He was evaluated at a peripheral hospital, and a chest X-ray (CXR) was ordered. In view of bilateral pulmonary infiltrates, a diagnosis of atypical pneumonia was entertained, and he was started on oral azithromycin and intravenous ceftriaxone. In view of worsening breathlessness, nausea, and vomiting, he was referred to our center. On examination, he was in respiratory distress with respiratory rate of 34/min. Blood pressure was 80/40 mmHg. He was maintaining a saturation of 60% on a rebreathing mask with 10 L oxygen. Arterial blood gas analysis on the same oxygenation revealed a PO_2 of 58 mmHg, PCO_2 of 32 mmHg, pH of 7.48, and bicarbonate 18 mmol/L. In view of hemodynamic instability and respiratory failure, he was intubated and mechanically ventilated. Although general physical examination revealed a well-built muscular young man, note was made of confluent acneiform rashes on the chest and back of the patient. CXR revealed extensive bilateral consolidation. Laboratory investigations revealed hemoglobin of 10 g/dl, total leukocyte count of 34,000 cumm with 93% neutrophils and toxic granules. Platelet count was 5.2 lakh/cumm, serum urea was 92 mg/dl, creatinine was 1.9 mg/dl, serum bilirubin was 2.1 mg/dl, indirect fraction being 1.8 mg/dl, aspartate aminotransferase – 543 U/L, and alanine aminotransferase - 982 U/L. Lactate dehydrogenase levels were elevated at 1222 U/L. Urine routine and microscopic examination was normal. Bronchoscopy was done via the endotracheal tube, which revealed diffuse hyperemia of the bronchial mucosa. Sequential bronchoalveolar lavage (BAL) from the right middle lobe showed progressively hemorrhagic return, suggestive of diffuse alveolar hemorrhage. The microscopy of the BAL revealed fresh red blood cells along with copious hemosiderin-laden macrophages. Blood and BAL pyogenic cultures did not reveal the growth of any pyogenic organisms. Urinary antigen for *Streptococcus pneumoniae* and *Legionella* was negative. Throat swab for H1N1 and influenza A also tested negative. IgM scrub typhus and Leptospira antibodies were negative. HIV testing by ELISA was negative. Random blood sugar levels were within range. Ultrasound abdomen revealed hepatomegaly. Two-dimensional echo was normal and did not reveal any evidence of infective endocarditis. Computed tomography (CT) chest was suggestive of bilateral consolidation with surrounding ground glass opacities [Figure 1].

A working diagnosis of severe community-acquired pneumonia (CAP) was made, and the patient was treated with intravenous cefoperazone with sulbactam, doxycycline, and methyl prednisolone[1] as per standard recommendations. He was ventilated with the standard low tidal volume acute respiratory distress syndrome protocol. The CT, CXR, and the clinical profile of the patient matched with severe CAP, but there were no evident risk factors for the development of severe CAP in this otherwise healthy young individual. The presence of the red acneiform rash on the trunk also led us to think that the whole episode was linked to an infection which we were unable to pick up with the battery of tests which had been done. In view of diffuse alveolar hemorrhage (DAH) and the rashes, a possibility of vasculitis was also entertained. However, as there was no renal dysfunction, no past history suggestive of any vasculitis, this diagnosis seemed unlikely. Antineutrophil cytoplasmic antibodies testing by ELISA also turned to be negative. Over the next 5 days, the patient made a gradual recovery and was extubated. However, the reason for developing severe CAP without any fever or other premonitory symptoms was unclear.

After extubation, a more detailed history was taken from the patient himself. The patient was an amateur body builder and wanted to enter a body building contest. At his local gymnasium, he had been advised by his peers to take a concoction of oral and intramuscular drugs to enhance his muscle power and bulk. Before this illness, he had started taking this concoction for around 15 days. His family was asked to procure the drugs that he was taking. They comprised oral stanozolol which is an anabolic-androgenic steroids (AAS) with creatine, which is commonly used to enhance muscle mass, along with intramuscular injection of nandrolone decanoate (Deca-Durabolin) and testosterone cypionate (depot testosterone) in the buttocks every day. Before this use for the past 15 days, the patient denied any history of using these drugs. The doses of the nandrolone decanoate, being used were extremely high (1000 mg/day).

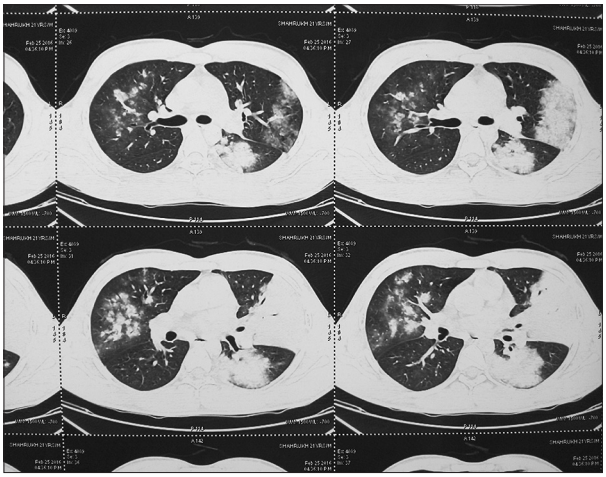

Figure 1: Computed tomography chest, lung window, axial cuts showing bilateral consolidation with surrounding ground glass opacities

In retrospect, the red acneiform rash present on the trunk was probably due to the effect of the anabolic steroids and not due to an infective etiology. Such skin lesions have been well described with the use of these agents.[2]

AAS are used medically to treat conditions such as hypogonadism, osteoporosis, and cachexia secondary to various diseases.[3] A number of adverse effects have been attributed to these agents, the primary being, skin rashes, virilization, cardiovascular-metabolic diseases, and hepatic and renal dysfunction.[4] Direct pulmonary damage due to these drugs is not reported. The effect of AAS in modulating the immune system is poorly understood. Furthermore, we could not find in the literature any pulmonary and infective complications of these agents, especially when taken at very high doses as was the case in our patient.

Our patient probably developed severe CAP as a result of heavy doses of the AAS he used for 15 days. Whether the DAH which then ensued was because of pneumonia or as a direct effect of the AAS is not clear but likely to be drug effect.

Whenever we encounter a patient with severe CAP, it is imperative that we try and find out the cause and risk factor of the same. Our patient, who was otherwise healthy and immunocompetent, developed severe life-threatening CAP with DAH after using heavy dose of AAS. Many young people these days are regular "gymmers" and frequently use substances which aid in body building. The side effects of these drugs must be made known to the public at large. The primary care physician and the intensivist involved in the management of young patients must take the history of the use of such agents.

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Conflicts of interest

There are no conflicts of interest.

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