Official Case Reports Journal of the Asian Pacific Society of Respirology
Respirology Case Reports
OPEN ACCESS
APSR

ECMO-dependent respiratory failure after snorting speed associated with anti-GBM antibodies

Nicholas De Rosa & Allan Glanville

Department of Thoracic Medicine, St. Vincent's Hospital, Sydney, Australia.

Keywords
Amphetamine, anti-GBM antibody, respiratory failure, speed.

Correspondence
Nicholas De Rosa, Department of Thoracic Medicine, St. Vincent's Hospital, PO Box 304, Sans Souci, NSW 2219, Australia. E-mail: derosa.nick@gmail.com

Received: 27 July 2015; Revised: 17 August 2015; Accepted: 01 September 2015.

Respirology Case Reports **2015; 3(4): 138–140**

doi: 10.1002/rcr2.128

Abstract

A previously well 20-year-old man with a history of nasal inhalation of "speed" was retrieved on extracorporeal membrane oxygenation for respiratory failure. Anti-glomerular basement membrane (anti-GBM) antibody was positive in the absence of renal disease. We postulate a hitherto unreported causal link between snorting "speed" and lung disease associated with anti-GBM antibody formation.

Introduction

Acute respiratory failure is a common complication of substance abuse. Mechanisms for drug-induced respiratory failure include pump failure (secondary to central nervous system depression or inadequate respiratory muscle function) and pulmonary pathology due to parenchymal, airway, or pulmonary vascular insults [1].

Immunologically mediated causes of parenchymal lung disease secondary to substance abuse are recognized but uncommon. Eosinophilic hypersensitivity pneumonitis ("crack lung") is a well-described reaction to cocaine smoking [1, 2]. Crystal amphetamine smoking has also been associated with acute eosinophilic pneumonia and alveolar damage [3].

We present the case of a 20-year-old man initially presenting with respiratory failure and anti-glomerular basement membrane (anti-GBM) antibody positivity. There was a history of intranasal use of a powder presumed by the patient to be "speed."

Case Report

The patient presented with a 1-week history of worsening dyspnea and 2 days of productive cough with hemoptysis. Despite oral antibiotics, his dyspnea progressed and he was admitted to a local tertiary hospital with type 2 respiratory failure requiring intubation and mechanical ventilation. Intravenous antibiotic and antiviral therapy were commenced; however, his respiratory failure worsened and he was retrieved and transferred to our institution on veno-venous extracorporeal membrane oxygenation (VV-ECMO).

He gave a history of nasal inhalation of "speed" 5 months previously but no other illicit substance use. He denied any interim symptoms. Past medical history included a 5-year history of untreated seronegative arthropathy affecting the hands and lower limbs, depression, and childhood asthma. Medications prior to presentation included duloxetine and multivitamins. He had smoked one to two cigarettes per day for the preceding 6 months, but there was no history of occupational exposure to dusts, gases, or petrochemicals.

Prior to retrieval, computed tomography (CT) pulmonary angiography was negative for pulmonary embolism but revealed extensive bilateral nodular lung infiltrate with tree-in-bud appearance, consistent with panbronchiolitis (Fig. 1). Chest X-ray showed significant interstitial markings throughout both lung fields (Fig. 2). Anti-GBM antibodies were detected by immunofluorescence, although the enzyme-linked immunosorbent assay result was below the cut-off for positivity (weak signal).

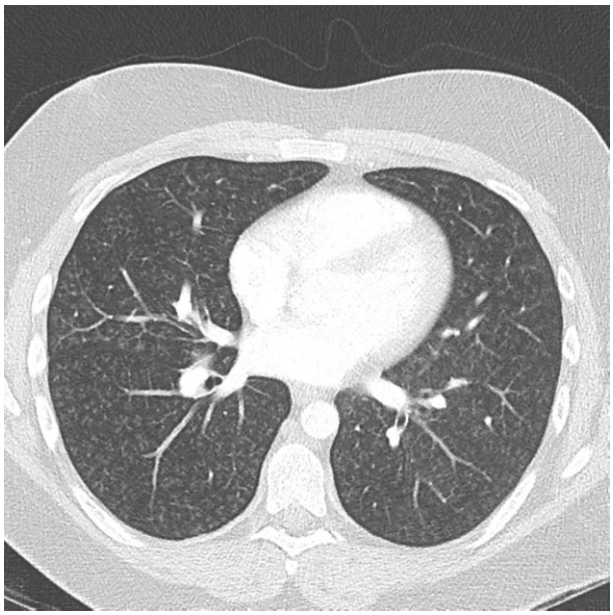

Figure 1. Computed tomography scan of the chest demonstrates widespread bilateral lung infiltrate, with centrilobular small nodules and tree-in-bud appearance peripherally, consistent with panbronchiolitis.

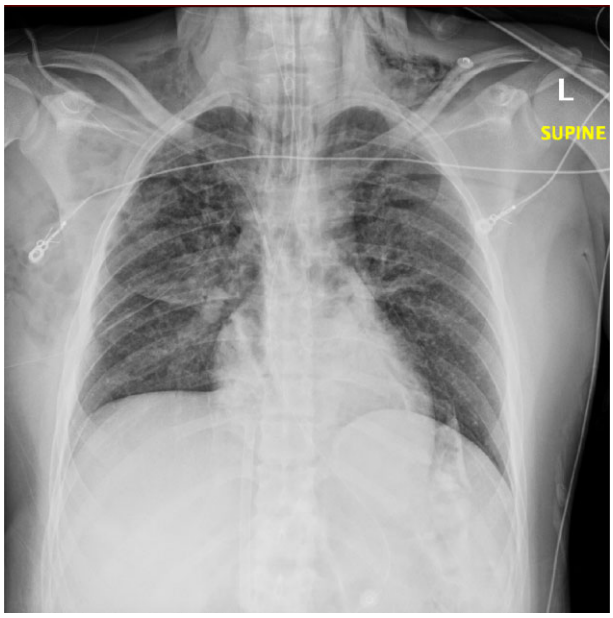

Figure 2. Widespread bilateral interstitial lung markings on chest X-ray.

Repeat blood cultures, sputum microbiology, and nasopharyngeal swab for respiratory viruses were negative. Bronchoscopy revealed mild erythema of the upper airways and bronchoalveolar lavage was negative for bacterial, fungal, and viral microbiology and cytology. Human immunodeficiency virus serology was negative. Anti-GBM antibody remained positive on triplicate testing. Renal function as determined by serum urea and creatinine concentrations remained stable throughout admission.

In view of refractory respiratory failure and critical illness, open lung biopsy was considered to have an unfavorable risk–benefit ratio. Hence, he was treated with three doses of methylprednisolone 1 g intravenously, followed by weaning high dose of methylprednisolone and prednisolone, one dose of immunoglobulin 60 g intravenously, a single dose of cyclophosphamide 1 g intravenously, adjuvant mesna, and one round of plasmapheresis. Following immunosuppressive treatment, anti-GBM antibody was negative 19 days after admission.

Complications included pneumothorax secondary to barotrauma, requiring intercostal catheter insertion, subcutaneous emphysema, pneumomediastinum, coagulopathy with hypofibrinogenemia, and hemothorax requiring blood product transfusion. Intravenous piperacillin/tazobactam and azithromycin were commenced for pneumonic changes on repeat CT scan of the chest in the absence of positive cultures. He developed a micropapular, erythematous eruption on the proximal limbs that resolved spontaneously, possibly drug induced. He was diagnosed with a superficial (long saphenous) vein thrombosis and warfarin was started.

After clinical and radiological improvement, he was decannulated from VV-ECMO on day 9 of admission and was discharged home on the 20th day. Following discharge, there was gradual improvement in his physical state and he returned to work after 2 months.

Discussion

We describe a case of non-infectious diffuse lung infiltration in the setting of anti-GBM antibody positivity and the absence of renal dysfunction. We postulate that the pharmacological component of "speed," being amphetamine, methamphetamine, or a structural analogue, was antigenic for anti-GBM antibody production, culminating in immunological respiratory failure. History and extensive investigation revealed no other likely cause.

A case of anti-GBM antibody-mediated glomerulonephritis following intranasal cocaine use has been reported previously, but there are no reports of amphetamine use associated with the development of anti-GBM antibodies [4]. Because of the inherent impurity of a non-proprietary product, there is the possibility that the "speed" inhaled by the patient presented in our case report may have contained cocaine.

Our case illustrates a possible causal association between the use of "speed" and anti-GBM-associated lung disease,

highlighting the complex relationship between autoimmune disease and environmental exposure.

Disclosure Statements

No conflict of interest declared.

Appropriate written informed consent was obtained for publication of this case report and accompanying images.

Acknowledgments

The authors would like to thank the other members of the lung transplant, thoracic medicine, and intensive care unit teams who contributed to the patient's care.

References

1. Wilson KC, and Saukkonen JJ. 2004. Acute respiratory failure from abused substances. J. Intensive Care Med. 19:183–193.
2. Shah R, Patel A, Mousa O, et al. 2015. Crack lung: cocaine-induced lung injury. QJM 108(9):749.
3. Lin SS, Chen YC, Chang YL, et al. 2014. Crystal amphetamine smoking-induced acute eosinophilic pneumonia and diffuse alveolar damage: a case report and literature review. Chin. J. Physiol. 57(5):295–298.
4. Peces R, Navascues RA, Baltar J, et al. 1999. Antiglomerular basement membrane antibody-mediated glomerulonephritis after intranasal cocaine use. Nephron 81(4):434–438.

