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CASE REPORT

Lung and pharyngeal abscess caused by enterotoxin G- and I-producing *Staphylococcus aureus*

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Virulence factors

Summary We report a particularly serious case of extensive meticillin sensitive Staphylococcal lung and pharyngeal abscess. Our patient had no significant risk factors for severe infection. The detection of enterotoxin G and I here suggest that when present together, these toxins work synergistically to produce a more virulent strain of *Staphylococcus aureus*.

Introduction

Staphylococcus aureus is a potent pathogen that can cause a variety of diseases with different degrees of severity.[1] It has multiple virulence factors that contribute to its pathogenicity and several of these can be employed by a particular strain at any one time.[2] One of the important virulence factors is toxin production, including super-antigens, which

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can produce overwhelming infection.[3] Host factors also contribute to susceptibility.

Our patient presented with extensive abscesses in the pharynx and lung following a productive cough. His only history of note was diabetes mellitus. Enterotoxin G- and I-producing *S. aureus* was detected in blood cultures and aspirates from the pharyngeal abscess. We suggest that the additive effect of these toxins, as noted in other publications, could have led to the severity of disease seen here.

Case report

A 51-year-old male patient with a history of diet controlled diabetes mellitus was admitted with increasing erythema and swelling of the right side of his neck. He had a four-week history of anorexia, unstable blood glucose levels, and a three week history of sore throat, a cough productive of brown sputum and right shoulder pain.

He had never smoked, drank alcohol only occasionally and had no other medical problems. He kept a dog and three cats, but there was no history of bites or trauma. He worked as a videographer and had a normal exercise tolerance. There was no history of recent travel and he denied any previous intravenous drug use. He had no gastrointestinal symptoms or evidence of preceding influenza infection.

On examination he was hypoxic with oxygen saturations of 94% on air, and tachypnoeic at a rate of 22. Chest auscultation was normal. He had a fluctuant, erythematous swelling in the right pharyngeal region. No other skin lesions were evident. He was normotensive, and tachycardic at 124 beats/min in sinus rhythm. He had no murmurs. His blood sugar was 20.9. A nasendoscopy was normal. His initial blood tests revealed a raised White Cell Count of 13.8×10^9/L, Neutrophil count of 12×10^9/L, CRP of 400 mg/L. His chest radiograph showed a small right-sided cavitating lesion.

A CT scan of his chest and neck was carried out. This showed widespread infection: a 5 cm right-sided parapharyngeal abscess communicating with the anterior mediastinum and with an $8 \times 2.5 \times 12$ cm abscess underlying the pectoralis major and minor. It had eroded through the first right costal cartilage into a 5 cm apical lung abscess. There were a further 8 cavitating nodules in the left lung and a few mediastinal lymph nodes between 9 and 14 mm in size (Fig. 1).

His blood cultures grew a fully sensitive *S. aureus*, as did a swab and aspirate from the abscess in the neck. Staphylococcal enterotoxin G and I (SEG and SEI) were detected by PCR carried out on his blood cultures. SEB, SEC and the Panton Valentine Leucocidin (PVL) toxin were not detected. A transthoracic echocardiogram showed no evidence of endocarditis. This extensive infection and associated sepsis was treated with 2 g intravenous Flucloxacillin six hourly. Because of initial poor response, linezolid was added for 2 weeks, providing excellent tissue penetration. Flucloxacillin was subsequently given orally for a further six weeks. On follow up 2 weeks after cessation of antibiotics he had improved clinically, biochemically and radiologically. The benefit of using Linezolid in this case is its ability to inhibit the secretion of *S. aureus* virulence factors including staphylococcal toxins through its inhibition of bacterial protein synthesis.[4]

On balance it was decided that the initial diagnosis was a severe infection due to a fully sensitive *S. aureus* cavitating pneumonia leading to a right-sided lung abscess, which then spread into a right-sided pharyngeal abscess. However, this could have begun with a pharyngeal abscess which spread to his lungs.

Discussion

S. aureus is commonly present as a skin commensal and also in the nose and throat. It is a frequent and potent pathogen, causing a variety of diseases including food poisoning, skin abscesses, wound infections, osteomyelitis, pneumonia, endocarditis, toxic shock syndrome (TSS) and staphylococcal shock syndrome (SSS).[5]

The organism causes infection by toxin production, direct invasion or haematogenous spread. Staphylococcal pneumonia can either be primary, due to direct pulmonary

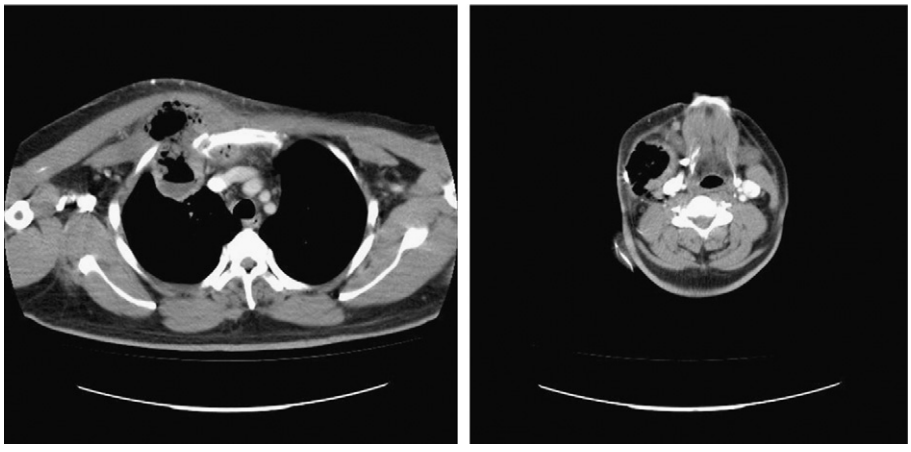

Figure 1 CT scan of the chest and neck showed a 5 cm right-sided parapharyngeal abscess communicating with the anterior mediastinum and with an $8 \times 2.5 \times 12$ cm abscess underlying the pectoralis major and minor. The chest wall abscess had eroded through the first right costal cartilage, into a 5 cm apical lung abscess.