Two cases of parotid gland infection with bacteremia due to meticillin-resistant
*Staphylococcus aureus*

D. A. Enoch,1 J. A. Karas,2,3 M. M. Emery3 and C. Borland4

1Clinical Microbiology and Public Health Laboratory, Health Protection Agency East of England, Addenbrooke’s Hospital, Hill’s Road, Cambridge, Cambridgeshire CB2 2QQ, UK
2Clinical Microbiology and Public Health Laboratory, Health Protection Agency East of England, Papworth Hospital, Papworth Everard, Cambridgeshire CB3 8RE, UK
3Department of Infection Control3 and Department of Medicine4, Hinchingbrooke Hospital, Hinchingbrooke Park, Huntingdon, Cambridgeshire PE29 6NT, UK

Parotid gland infection as a source of meticillin-resistant *Staphylococcus aureus* bacteremia has been rarely reported. It is predominantly a disease of the elderly and is associated with significant mortality. Two cases are described here that presented over a 6 month history at a district general hospital. Many cases may be preventable with adequate hydration and good oral hygiene, combined with effective infection control.

Introduction

*Staphylococcus aureus* has long been recognized as a common cause of suppurative parotid gland infection (Petersdorf *et al.*, 1958) and remains one of the most frequent causes to this day (Brook, 2003). We describe two cases of parotid gland infection complicated by bacteremia due to meticillin-resistant *Staphylococcus aureus* (MRSA) that occurred in a district general hospital over a 6 month period.

Case 1

An 88-year-old female patient was admitted from home with a history of a fall following a cerebrovascular accident. She had a past history of myeloproliferative disorder and had been admitted twice in the preceding year, during which she had received numerous antibiotics following an episode of febrile neutropenia. She also had a sacral pressure sore. She had never had a previous MRSA-positive sample. Ten days after admission she developed a swelling over the left parotid area and a temperature of 38.5 °C. She was dehydrated. Her medication included 40 mg furosemide and 10 mg cetirizine, once daily. Her white cell count was elevated at 28.1 × 10^9 cells l^-1 (normal range 4–11 × 10^9 cells l^-1) and her C-reactive protein was 118 mg l^-1 (normal range < 6 mg l^-1). Empirical flucloxacillin and metronidazole were commenced. Blood cultures taken at this time and a swab of expressed pus taken from the parotid gland opening grew MRSA so her antibiotics were changed to intravenous vancomycin, and she completed 14 days treatment. She died due to complications of her myeloproliferative disorder 7 weeks later.

Case 2

A 73-year-old female was admitted from a nursing home with pyrexia of 38.2 °C, swelling of the left parotid gland and dehydration. There was no evidence of a parotid abscess and no stones were palpable in the duct. She was edentulous and there was pus visible at the parotid duct opening. A clinical diagnosis of parotitis was made. A sacral pressure sore was present but showed no signs of cellulitis. She had a past medical history of type II diabetes mellitus, dementia and a left below knee amputation for gangrene of the foot. She lived in a nursing home and had four previous admissions to hospital in the preceding 12 months. During an admission 2 months previously she had developed a sacral pressure sore infection with MRSA and group G streptococcus. Her medication included 2.5 mg bendroflumethiazide and 10 mg amitriptyline once daily. Her white cell count (31.6 × 10^9 cells l^-1) and C-reactive protein (177 mg l^-1) were elevated. Vancomycin was started empirically because of her MRSA status, and was continued when blood cultures taken on admission grew MRSA within 24 h. A mouth swab taken on admission grew mixed oral flora. No other source for the bacteremia was found. She was discharged 19 days after admission and was well at follow up 12 months later.

Results and Discussion

MRSA is increasing in prevalence, and whilst community-acquired MRSA is increasingly recognized, acquisition is still predominantly hospital or healthcare associated. Many of the risk factors for acute suppurative parotitis and MRSA overlap, and include old age, multiple co-morbidities,
hospital admission and residence in a nursing home. Other risk factors for acute suppurative parotitis include dehydration and malnutrition, poor oral hygiene, sialectasis, dental infection, immunosuppression and medication (diuretics, antihistamines, anticholinergics) (Brook, 2003). These risk factors have not changed over time (Petersdorf et al., 1958). Both the cases described had multiple risk factors for parotitis.

Despite the frequent occurrence of patients with these risk factors, MRSA parotitis remains rare in the published literature, with only eight cases reported to our knowledge. The first case was described in 1990 and proved rapidly fatal (Rousseau, 1990), with seven cases being described subsequently (Manfredi et al., 1997; Cohen & Docktor, 1999; Chien et al., 2000; Molina et al., 2003; Mohammed & Hofstetter, 2004). Only three cases have described bacteraemia as a result of parotid gland infection with MRSA (Cohen & Docktor, 1999; Molina et al., 2003). A total of 2 cases from 76 (2.3%) MRSA bacteraemias over a 4 year period at Hinchingbrooke Hospital were due to parotitis (Karas et al., 2006). The risk factors, clinical details and outcome of the published cases of MRSA parotitis are presented in Table 1. The median age of the ten cases was 82 years (range 58–97 years), with six cases occurring in women. Two cases were bilateral. Most of the patients were elderly, with high peripheral white cell counts (13–36.8 × 10⁹ cells l⁻¹). Local risk factors were not mentioned for four patients. The mortality rate was 25% (outcome not recorded in two

### Table 1. MRSA parotitis cases described in the literature

<table>
<thead>
<tr>
<th>Study</th>
<th>Age (years) and sex</th>
<th>Risk factor</th>
<th>Clinical presentation*</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rousseau (1990)</td>
<td>85, male</td>
<td>Cerebrovascular accident, ischaemic heart disease, furosemide, dry mucosa, poor oral intake†</td>
<td>T 38.9 °C, bilateral firm erythematous swelling, WCC 13·0 × 10⁹ cells l⁻¹</td>
<td>Nafcillin</td>
<td>Died day 1</td>
</tr>
<tr>
<td>Manfredi et al. (1997)</td>
<td>72, male</td>
<td>Pancreatitis, central line, dry mucosa, poor dentition†</td>
<td>'Hyperpyrexial', bilateral firm swelling, tender, trismus. WCC 20·0 × 10⁹ cells l⁻¹</td>
<td>Teicoplanin</td>
<td>Fistula formation and CNVII palsy, died day 12</td>
</tr>
<tr>
<td>Cohen &amp; Docktor (1999)</td>
<td>85, female</td>
<td>Type II diabetes mellitus, congestive cardiac failure, dry mucosa, multiple medication (not stated)†</td>
<td>T 39·5 °C, left swelling, tender, erythematous, WCC 36·8 × 10⁹ cells l⁻¹, bacteraemia</td>
<td>Cefazolin then vancomycin</td>
<td>Survived</td>
</tr>
<tr>
<td>Chien et al. (2000)</td>
<td>58, female</td>
<td>Previous MRSA, multiple hospital admissions, medical problems</td>
<td>Left swelling, no further details</td>
<td>Vancomycin then linezolid</td>
<td>Survived</td>
</tr>
<tr>
<td>Molina et al. (2003)</td>
<td>97, female</td>
<td>Dementia, nursing-home resident, dehydrated†</td>
<td>T 37·8 °C, drowsy, right parotid swelling and pain, WCC 31·0 × 10⁹ cells l⁻¹, bacteraemia</td>
<td>Vancomycin</td>
<td>Survived</td>
</tr>
<tr>
<td></td>
<td>91, female</td>
<td>Dementia, nursing-home resident†</td>
<td>T 37·5 °C, right parotid swelling, WCC 29·0 × 10⁹ cells l⁻¹, bacteraemia</td>
<td>Vancomycin</td>
<td>Survived</td>
</tr>
<tr>
<td>Mohammed &amp; Hofstetter (2004)</td>
<td>79, male</td>
<td>Cerebrovascular accident, dry mucosa, poor dentition†</td>
<td>T 40·4 °C, swollen right parotid, WCC 15·6 × 10⁹ cells l⁻¹</td>
<td>Cefazolin then vancomycin</td>
<td>Outcome not recorded</td>
</tr>
<tr>
<td></td>
<td>73, male</td>
<td>None recorded†</td>
<td>T 40·3 °C, painful right swelling, WCC 24·0 × 10⁹ cells l⁻¹</td>
<td>Vancomycin</td>
<td>Outcome not recorded</td>
</tr>
<tr>
<td>This study</td>
<td>88, female</td>
<td>Myeloproliferative disorder, type II diabetes mellitus, no previous MRSA result</td>
<td>T 38·5 °C, left painful swelling parotid, WCC 28·1 × 10⁹ cells l⁻¹, bacteraemia</td>
<td>Flucloxacillin then vancomycin</td>
<td>Died 7 weeks later due to myeloproliferative disease, Survived</td>
</tr>
<tr>
<td></td>
<td>73, female</td>
<td>Dementia, type II diabetes mellitus, previous MRSA, nursing-home resident</td>
<td>T 38·2 °C, left swelling, WCC 31·6 × 10⁹ cells l⁻¹, bacteraemia</td>
<td>Vancomycin</td>
<td>Survived</td>
</tr>
</tbody>
</table>

*T, temperature; WCC, white cell count.
†MRSA status prior to episode not recorded.
††CNVII, VIIth cranial nerve.
cases). Previous infection or colonization with MRSA was not recorded in the other studies. Knowledge of such prior infection enabled vancomycin to be given empirically in case 2 described above. Most of the cases had several risk factors.

We believe that prompt management of cases is essential, involving culture of parotid drainage fluid (via pus expression or needle aspiration) and blood cultures. Empirical antibiotics should cover *S. aureus* (including MRSA if risk factors exist) and anaerobes, pending susceptibility results. Drainage is usually only required if an abscess forms. MRSA parotitis is largely a disease of the elderly with a high mortality. Adequate hydration and good oral hygiene with effective infection control should prevent future cases.

References


