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Acute suppurative left parotitis after myocardial revascularization surgery: a case report

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Summary

Introduction. Acute suppurative parotitis is a rare infectious process occurring in debilitated patients and/or after surgery. The parotid gland is most commonly affected by an inflammatory process. ***Case report.*** The authors describe acute suppurative left parotitis in the postoperative period after coronary artery bypass grafting in a 61-year-old patient. According to the best knowledge of the authors this is probably the first described case of this complication's occurrence in the postoperative course in the patient who underwent surgical treatment of ischemic heart disease. *Anestezjologia i Ratownictwo 2010; 4: 422-426.*

Keywords: acute suppurative parotitis, coronary artery bypass graft, ventilator-acquired pneumonia, multiorgan failure

Introduction

Acute suppurative parotitis is an infrequent disease entity occurring in debilitated patients or after surgery [1-3]. The parotid salivary gland is the gland most often affected by the inflammatory process [2,4-6]. Among predisposing risk factors, dehydration, malnutrition, oral cavity neoplasms, immunosuppression, sialadenitis and commonly prescribed drugs causing a decrease in salivary production seem to be the most important [1,4].

The oral cavity is colonized by 300-400 bacteria species [7]. The bacteria causing ASP (acute suppurative parotitis) most often are: *Staphylococcus aureus*, *Streptococcus* and – less frequently – anaerobic, Gram-negative bacteria: *Prevotella, Porphyromonas, Fusobacterium, Peptostreptococcus* [4]. Gram-negative bacteria often occur in immobilized, hospitalized patients [5].

Treatment is by application of wide-spectrum oral antibiotics [4]. The antibiotic therapy may change after obtaining results from culture of the parotid purulent secretion and determination of its antibiotic sensitivity.

Case report

The patient 61 years old, with long standing hypertension, experienced acute coronary syndrome with non-ST-segment elevation myocardial infarction (NSTEMI) in October, 2008, treated by right coronary angioplasty with in-stent implantation. Coronary angiography presented three-vessel coronary disease, and hypokinesis of the apical segment of the lateral wall with EF 50% in the echocardiographic evaluation. The patient was scheduled for elective myocardial revascularization surgery.

On 11 December 2008 in extracorporeal condi-

tions, 3 venous aortal-coronary bridges (Dg [diagonal coronary artery], PL [left posterolateral branch] jump Cx [circumflex coronary artery]) and a bridge from the internal thoracic aorta to the left coronary aorta LAD (left anterior descending coronary artery) were performed.

The mid- and postoperative course were complicated by anterolateral myocardial infarction (MI) with accompanying cardiac shock treated with catecholamines and intra-aortal balloon counterpulsation (IABP) for 48 hours. In echocardiographic examination EF decreased to 35%.

On the second day after the surgery circulatory system stabilization was achieved, as well as the patient's spontaneous, efficient respiration. On the fifth day after the surgery, WBC (white blood cells) increased to 23,0 x 10^9/L, CRP (C-reactive protein) was 344 mg/L, and antibiotic therapy was changed: cefotaxime (Tarcefoksym, IBA Bioton SA, Poland) was stopped, and meropenemum (Meronem, AstraZeneca, Poland) was included. The patient, periodically restless and disoriented, needed to be pharmacologically sedated. Neurological consultation did not present any deviations.

On the eleventh postoperative day, due to rapid deterioration of respiratory efficiency, spastic changes and ineffective expectoration, the patient was intubated and underwent MCV (mechanical controlled ventilation). On the twelfth day there was an increase of body temperature to 37.6°C, WBC 13,0 x 10^9/L, in the white blood picture 41% stab cells, diarrhoea. To meropenemum administration (3 x 0.5 i.v./daily), metronidazole (Metronidazol, Fresenius-Kabi, Poland) was added. On the sixteenth postoperative day were observed oedema of the left parotid, increased temperature of this site, and purulent secretion from the duct orifice in the oral cavity upon massage. The patient was without fever, leucocytosis 8,2 x 10^9/L. On the eighteenth day leucocytosis had grown to 12,6 x 10^9/L, in the white blood picture 58% stab cells, CRP 255 mg/L, without increased temperature. Parotid oedema remained, and attempts to take the respirator off were unsuccessful. On the nineteenth day the patient had a temperature of 38.9°C, as a result of obtaining a culture from tracheal-bronchial tree sampling on the sixteenth day – *Pseudomonas aeruginosa* was cultured, sensitive to meropenemum, and yeast-like fungi. Blood culture was negative. Due to the total clinical picture meropenemum was stopped (fourteenth day of therapy), and administration of ciprofloxacin (Cipronex, Polpharma SA, Poland) and netilmicin (Netromycin, IBA Bioton SA, Poland) was started according to the antibiogram; fluconazolum (Flumycon, Pliva, Poland) and metronidazole were maintained. Due to the growing left parotid oedema and development of inflammatory infiltration in the temple region, the patient was consulted by a maxillary and facial surgeon.

Upon extra-oral examination a significant oedema of the left cheek spread to the left submandibular region, skin hyperaemia and pain on palpation were noted. Additionally, in the left temple and partially frontal region, a visible, "cake-like" infiltration was observed. Submandibular lymph nodes of group B on the left side were impalpable.

Intra-oral examination presented oedema and hyperaemia around the exiting duct's orifice of the left parotid. The remaining segments did not present any alterations.

In the first stage the left exiting duct's orifice was patented, achieving a significant amount of purulent secretion for the bacteriological test. The exiting duct was multiply irrigated with chlorhexidine solution and metronidazole – cold boric acid compresses recommended. In the second stage (after several hours) as a result of the lack of any improvement in the local state, under general anaesthesia, an incision of an abscess in the left submandibular and temple region was performed; tissues in the direction of the left cheek, temple and frontal region were delaminated. An ichor-like secretion was obtained, characteristic for soft tissue necrosis in phlegmon. The above described spaces were irrigated and drained (photos 1 and 2).

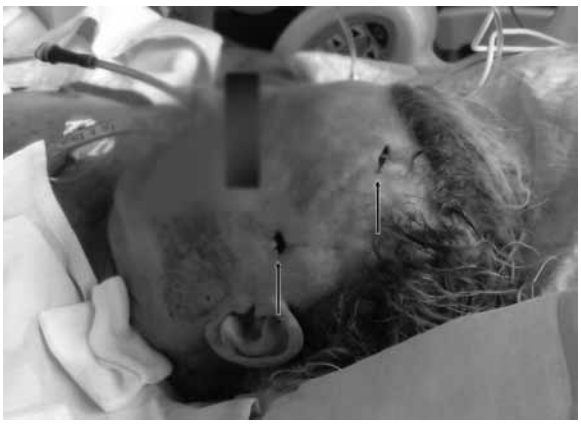

Photo 1. The places of incision and drainage of the abscess: left frontal and temporal regions

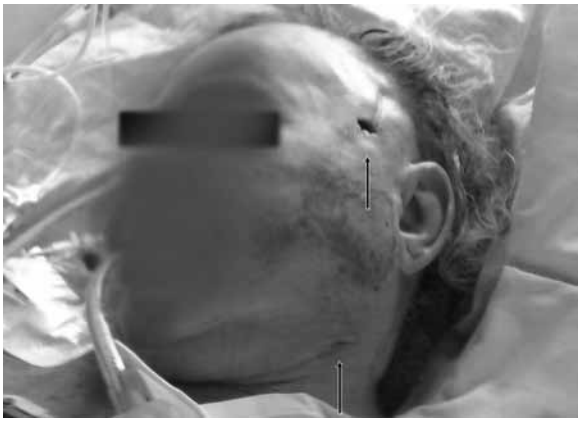

Photo 2. The places of incision and drainage of the abscess: left temporal and submandibular regions

The patient's local condition after the surgery improved slightly – left parotid oedema decreased during salivary gland massage, while subsequent maxillary and facial surgeon consultations on the 22nd and 23rd day revealed a smaller amount of purulent secretion. On the 24th day after the surgery the patient's local condition improved, no facial inflammatory infiltrations were observed, the left salivary gland secreted proper saliva, and all drains were removed. The patient's general condition improved as well. Leucocytosis drop to 6,0 x 10^9/L, lack of stab cells in smear, decrease in CRP to 50 mg/L, and normothermia were noted. In the smear from the salivary gland *Pseudomonas aeruginosa*, sensitive to the applied treatment, was cultured and coagulase-negative, methicillin-resistant staphylococci were found. Vancomycin (Edicin, Lek Pharmaceuticals, Slovenia) was included according to the antibiogram. On the 28th day bronchofibroscopy and bronchial tree toilet were performed, only to reveal buccal membrane inflammation and a remarkable amount of remaining purulent secretion. Percutaneous tracheotomy was applied. From the bronchial tree culture *Acinetobacter baumannii* was cultured – meropenemum was administered according to the antibiogram, while vancomycin and fluconazolum were maintained. Blood culture was negative.

On the third day after the surgery the patient's state rapidly deteriorated. In X-ray pulmonary oedema and right-sided inflammatory changes were noted, as well as circulatory destabilization requiring application of catecholamines, hypoalbuminaemia, generalized oedemas, temperature to 38.4°C, WBC 4,5 x 10^9/L. In the further course there were multiorgan failure symptoms. The patient died on the 35th day after the surgery. We do not have a reliable confirmation of the described cause of death because the patient's family applied to the hospital's authorities for a discharge from the autopsy.

Discussion

Surgical treatment of ischaemic heart disease involves the possibility of many complications which can occur in the postoperative period. They may, to a variable degree, affect the treatment result. Some of them result from the progression of ischaemic heart disease (myocardial infarction episode, impaired left ventricular ejection fraction (EF), arrhythmia, especially atrial fibrillation [AF]) [8], current treatment methods, including invasive cardiological methods (stents), drugs taken, and anti-platelet treatment, for example. Other complications can be caused by accompanying diseases: diabetes, hypertension [9], respiratory system diseases, especially chronic obstructive pulmonary disease (COPD), diseases of kidneys, liver, digestive system, central nervous system, and active inflammatory processes [10]. The operation mode is also very significant for the result of ischaemic heart disease treatment: emergent or elective, then age, sex, past myocardial infarctions and other surgical procedures [10].

During extracorporeal circulation, many cells responsible for the so-called immunological response start to activate. This phenomenon is called the generalized inflammatory reaction. Pro-inflammatory cytokines TNFα (tumor necrosis factor alpha), interleukins 1 and 6, and growth factors TGFα (transforming growth factor alpha) and IGF1 (insulin-like growth factor 1) take part in its appearance [10,11].

The inflammatory process is usually a result of application of the extracorporeal circulation technique; its intensity is not great and spontaneously subsides. In some situations, the inflammatory process can become more intensive (systemic inflammatory response syndrome, SIRS), turning into septic syndrome, which can lead to multiorgan failure syndrome (MF) occurrence [11,12].

This refers to patients operated on with an active inflammatory process. The oral cavity is colonized by 300-400 different species of bacteria [7]. The most

common chronic bacterial infections in people are: periodontitis, teeth diseases and their inflammatory complications in the range of soft tissues and maxillary bones. Acute sialadenitis is a rare, potentially serious disease entity. This disease most often affects a parotid gland [2,4-6], and is present in debilitated patients with a weakened anti-inflammatory response and/or after serious surgical procedures [1-3]. The most frequent bacteria causing sialadenitis are: S. *aureus*, *Streptococci*, and, less often, anaerobic Gram-negative bacteria, which are more frequent in hospitalized patients [4,5].

High susceptibility of patients to infections at intensive therapy units results from three basic causes: (a) Primary serious condition of hospitalized patients, (b) prolonged mechanical ventilation, long-term maintenance of a central venous catheter, and (c) specificity of hospital ward bacterial flora, necessity of wide-spectrum antibiotic therapy, hospital staff superinfection.

Both intubation and tracheostomy deprive patients of the possibility of cleaning, hydration and warming of the inhaled air, which, together with the inability of physiological expectoration, can create perfect conditions for development of infection, and the damaged mucosa becomes a place of Gram-negative bacteria binding.

Bronchial secretion evacuation of patients intubated with the aid of catheters means that the catheter must cover the zone of the tracheostomy/intubation tube potentially colonized by bacteria, and this is connected with a possibility of mechanical lesions to the bronchial tree mucosa – which, again, makes bacterial colonization easier [13).

Hospital-acquired pneumonia occurring up to 7 days after hospitalization is described as early. Late-onset hospital-acquired pneumonia, most often connected with seriousness of the general condition, and especially ventilatory-acquired pneumonia (VAP), is characterized by a high mortality rate at the level of 13-55% [10,13].

The percentage distribution of bacteria in VAP [14] shows a correlation between the duration of the infection period and the change of bacterial flora isolated from the respiratory tracts. On the first days of VAP, Gram-positive *cocci* are causative factors; on the 5^{th}-6^{th} day of infection the amounts of Gram (+) and Gram (-) bacterial strains are comparable; and after the 7^{th} day, Gram (-) rod strains, such as *Pseudomonas aeruginosa* or *Acinetobacter sp*., are in the majority – treatment of the latter is especially difficult.

In conclusions, acute suppurative left parotitis in postoperative period after coronary artery bypass grafting might especially occur in debilitated patients with a weakened anti-inflammatory response, usually after serious surgical procedures. *Staphylococcus aureus*, *Streptococci* and the most rare anaerobic Gram-negative bacteria are the most frequent factors that might cause sialadenitis. High susceptibility of patients to infections at ICUs mostly results from the three basic causes: (1) primary serious condition at admission, (2) prolonged mechanical ventilation, long-term maintaining of a central venous catheter, (3) specificity of hospital ward bacterial flora, necessity of wide-spectrum antibioticotherapy, and hospital staff superinfection.

Competing interests

The authors declare that they have no competing interests.

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