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EDITORIAL

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Nosocomial sinusitis

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The four paired cavities of the paranasal sinuses are lined with ciliated, pseudostratified, columnar epithelium and have a narrow ostium that opens into the nasal cavity. In spite of the continuous contact with a heavily colonised area, the sinuses have no indigenous resident bacterial flora and are considered sterile. Similar to the tracheobronchial system, the sinuses are covered with a protective mucous layer which envelops bacteria and other irritants, covers the respiratory cilia and is moved continuously to the sinus ostia.

Sinusitis occurs when this transport mechanism fails, usually in response to an allergen or a microorganism. Other causes comprise mechanical obstruction, trauma and primary ciliary dysfunction (e.g. Kartagener syndrome). The mucociliary transport is impaired by hypersecretion of mucus and/or inflammatory mediators released in response to infecting organisms or allergens. Additionally, this causes mucosal oedema which may lead to ostial obstruction, congestion and thickening of the sinus walls. Secondary bacterial invasion from the nasal cavity cannot be cleared adequately, thus reinforcing this circulus vitiosus.

While acute and chronic sinusitis in non-hospitalised patients are common [1], the significance of nosocomial sinusitis (NS) in intensive care patients was not well recognised before the mid-1980s [2], although the first published cases dated back to 1974 [3]. Up to now, a wealth of studies covering the different aspects of NS has been published with reported frequencies of NS varying greatly from < 5 to 100%, depending on the diagnostic criteria applied and the population studied [4, 5]. Epidemiological studies have linked nasotracheal and nasogastric intubation to the occurrence of NS, as well as patients with head injuries and facial fractures exhibiting an increased risk of this disease [6, 7]. There is an ongoing discussion whether NS is an important reason for unexplained fever in patients in the intensive care unit (ICU) and if it may be a primary source of ventilator-associated pneumonia (VAP) [5,8].

Unlike community-acquired sinusitis, NS is difficult to diagnose by physical examination due to the paucity of clinical signs. Besides the non-specific signs of fever and leucocytosis, mucopurulent nasal discharge may lead to the first suspicion of NS, which usually is followed by radiological examination either by standard plain sinus radiograph or computed tomography (CT) [4]. Although normal radiographs at the bedside are more convenient and easier to perform, their diagnostic yield is worse than with CT, which may differentiate between mucosal thickening, air fluid level and total opacification. An alternative to the bedside radiograph is ultrasonography [9], with the major advantage of avoiding radiation exposure for the patient; however, special sonography equipment is necessary. If used for daily monitoring, the diagnostic ability to recognise an antral abnormality has proved to be good, but it is inadequate for differentiating the type of abnormality.

However, for a definite diagnosis these three indirect diagnostic methods are not sufficient because they do not distinguish between infectious and non-infectious sinusitis. The diagnostic gold standard for antral disease is direct examination by antroscopy and microbiological culture. Provided that an antral bacteriological culture is positive and without contaminants, infectious sinusitis may well be the definite and correct diagnosis. Although most probably the origin of the infecting bacte-

ria is the flora of the nasal cavity – either indigenous or nosocomially acquired – a single nasal swab or sampling of mucopurulent discharge is definitely inadequate. The standard diagnostic as well as therapeutic procedure is antral puncture with or without lavage, either transnasally via the inferior meatus or transorally through the canine fossa. But both methods bear a considerable risk of contamination with the local bacterial flora. Even thorough disinfection of the nasal cavity with povidone-iodine "sterilises" the puncture site in only 50 % of cases [6]. Therefore, Westergren et al. proposed a quite, radical approach by preparing a free bone area and then introducing a trocar through the canine fossa into the maxillary cavity [9]. By this procedure the authors lowered their contamination rate down to 5 %. In their study of 33 long-term ventilated patients with clinically suspected NS (purulent nasal discharge and radiographic sinusitis), Westergren et al. [9] established a definite diagnosis of infectious sinusitis in only 6 %, while in 79 % the diagnosis was non-infectious reactive inflammatory sinusitis. The remaining 15 % were without antral disease. The findings from culture of the antral secretions using the above procedure were a mixed anaerobic flora with a predominance of *Fusobacterium* spp. and *Prevotella* spp. in two cases, one pure culture of *Escherichia coli* and one sample yielding growth of *Lactobacillus* sp.

These data are strikingly different from results of studies such as the paper published in this issue by LeMoal and colleagues [10]. Although extremely well performed from the diagnostic microbiologist's viewpoint, as indicated by the high isolation and identification rate of strictly anaerobic bacteria, the investigation is hampered by the unmentioned problem of contamination during specimen collection (see above). Nevertheless, the spectrum of isolated bacteria parallels that found by Westergren et al., reflecting the local resident and transient oropharyngeal and nasal flora in artificially ventilated patients. Members of this group of flora, as LeMoal et al. also stated, may not only cause NS but should be regarded as primary candidates for VAP. Therefore, it seems a somewhat academic discussion whether NS can be the origin of VAP or if the oropharynx is the source of the microorganisms implicated in either disease.

The conclusions and pragmatic guidelines for daily routine practice in intensive care may be as follows:

Sinusitis is a frequent disease in long-term ventilated ICU patients; however, the rate of infectious sinusitis based on stringent diagnostic criteria using antroscopy and histopathological and microbiological examination of the antral mucosa may be only about 10 % in those patients with radiological sinusitis.

Major causes of NS are nasotracheal and nasogastric intubation and also head trauma and cranial or facial surgery.

The role of NS as the underlying cause of fever of unknown origin in long-term ventilated patients is difficult, and in most cases even impossible, to establish.

Diagnosis of NS could be based on routine ultrasonographic monitoring of the paranasal sinus every 1 or 2 days. Signs of new antral abnormalities should be followed by CT. Plain bedside radiography seems to be of little value.

If, together with purulent nasal discharge, CT confirms the diagnosis of NS, antral puncture with lavage is the most important therapeutic measure. Antroscopy is only indicated when CT detects an unusual form of sinusitis (e. g. mucocele, abscess formation or fungus ball).

The value of specimens for microbiological cultures must be questioned due to the very high contamination rate by nasal flora. Additionally, in long-term ventilated patients there should be enough information about the individual oropharyngeal or tracheal flora, which most probably is very similar to the "sinusitis flora". Nevertheless, if samples for microbiological cultures are taken, they must be classified as "high urgency" specimens, meaning immediate transportation to the laboratory and rapid and adequate processing. Results, which at least should be reported semiquantitavely, must be interpreted cautiously, bearing in mind contaminating microorganisms of the nasal flora.

Antibiotic therapy of NS should be performed in all patients with systemic signs of infection and should cover a broad range of aerobic and anaerobic bacteria. The initial empirical antibiotic therapy should consist of broad-spectrum antibiotics (e. g. penicillin/beta-lactamase-inhibitor combinations, or penems, or quinolones plus metronidazole, or third generation cephalosporins plus metronidazole) similar to the initial therapy of nosocomial pneumonia. The choice of the antibiotic must consider the local "hospital flora" and the antibiotic resistance situation, such as rates of methicillin-resistant *Staphylococcus aureus*, extended spectrum beta-lactamase-positive enterobacteria or quinolone-resistant *Pseudomonas aeruginosa*.

Finally, preventive measures, such as avoiding major risk factors (long-term nasopharyngeal or nasogastric intubation), strict adherence to well-known measures of hospital hygiene (hand disinfection, closed ventilation circuits using sterile water for humidification, use of aseptic solutions for mouth care, etc.) must be emphasised as an integral part of quality intensive care.

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