SPONTANEOUS CORNEAL PERFORATION AND ENDOPHTHALMITIS IN PSEUDOMONAS AERUGINOSA INFECTION IN A VENTILATED PATIENT: A CASE REPORT

WYNANTS S., KOPPEN C.*, TASSIGNON M.J.**

SUMMARY

We report a case of Pseudomonas keratitis and endophthalmitis after inoculation from the respiratory tract in a mechanically ventilated patient. In these (semi)comatose and more vulnerable patients, colonisation of the upper respiratory tract by Pseudomonas occurs frequently, and this can lead to inoculation of the eyes. Emphasis lies on careful prevention of ocular inoculation and aggressive therapy as soon as keratitis is noticed.

SAMENVATTING

We stellen een casus voor van Pseudomonas keratitis en endoftalmitis na besmetting vanuit de bovenste luchtwegen bij een kunstmatig beademde patiënt. Bij deze patiënten treedt vaak een colonisatie van de luchtwegen op door Pseudomonas aeruginosa. In combinatie met de verminderde oculaire afweermechanismen bij semi-comateuze patiënten, kan dit leiden tot een agressief evoluerende keratitis.

RÉSUMÉ

Nous présentons un cas de kératite et d'endophtalmie à Pseudomonas dûe à une inoculation par les voies respiratoires supérieures chez un patient ventilé mécaniquement. Chez ces patients la colonisation des voies respiratoires par des pathogènes résistants est bien connue. Ils peuvent être responsable d'une inoculation des yeux chez des patients présentants une cornée compromise. Il est très important de reconnaître la pathologie très tôt vu le danger d'une progression rapide et d'instituer immédiatement une thérapie agressive dès l'apparition d'une kératite.

MOTS CLÉS

Pseudomonas aéruginé, kératite, endophtalmie, ventilation mécanique.

KEY WORDS

Pseudomonas aeruginosa, keratitis, endophthalmitis, mechanically ventilated patients.

.....

* *Department of Ophthalmology, University Hospital Antwerp, Belgium.*

received: 31.01.00
accepted: 10.04.00

INTRODUCTION:

The development of corneal ulcers by pathogens of the respiratory tract as a complication of mechanical ventilation has been described since the early 1970's (7).
Pseudomonas aeruginosa can cause very virulent corneal infections which can quickly lead to hypopion formation and destruction of the corneal stroma. Perforation of the eye can occur within 48 hrs (1,3,9,17,23).
We present a case of Pseudomonas aeruginosa keratitis in a mechanically ventilated patient, leading to ulceration, perforation, and finally evisceration.

CASE REPORT

An ophthalmological consultation was requested for a 49-year-old polytraumatized, comatose man, mechanically ventilated in the intensive care unit for two months, because of multiple fractures, multiple organ failures, a torn liver, a splenoraphy and sepsis. The reason for the ophthalmological referral was the sudden appearance of a red eye with discharge on the left side.
It was known that the patient's upper respiratory tract was colonised with Pseudomonas aeruginosa. He had been treated with fusidine acid one month earlier for a corneal erosion in the left eye. The treatment had been stopped and replaced by an eye gel lubricant one week before the requested examination.
At the time of examination, the eyelids were swollen, sticking to each other and a purulent discharge was present. While opening the eyelids for further evaluation, the cornea perforated spontaneously, a drop of anterior chamber fluid squirted out of the eye and immediately hereafter the intraocular lens dropped onto the cheek, followed by vitreum. An evisceration was performed the next morning.
Cultures of the cornea and vitreum revealed the presence of Pseudomonas aeruginosa as was found in the patient's sputum.
The patient was closely monitored ophthalmologically: his right eye presented repetitively with sticky eyelids and mucous discharge, white dots and strands on the corneal surface. Cultures of this right eye remained negative however, and all symptoms disappeared after treatment with a lubricant. Repeated fundoscopic examinations revealed no signs of endogenous infection. At the day of transfer to a rehabilitation centre, the ophthalmological examination showed a healthy right eye and a well healed evisceration wound at the left eye.

DISCUSSION

The corneal ulcer caused by Pseudomonas aeruginosa has several characteristics: it is usually centrally located, almost always secondary to corneal injury and has a fulminant progression. The cornea gets involved within 48 to 72 hours after inoculation. A hypopyon of unusual size can be present early in the disease. Pus, mucous in consistency and greenish in colour, appears in copious amounts. Unless specific adequate treatment is initiated immediately, panophthalmitis can develop (1,4,8).
At first, when evaluating this case, the diagnosis of a postsplenectomy syndrome (PSS), which is an endogenous endophtalmitis due to sepsis after splenectomy, was considered (5). However, this is more likely to occur after splenectomy and not after splenoraphy. Spleen insufficiency after splenoraphy was improbable in our patient since the counting of thrombocytes remained normal (<400.000) instead of being increased. Moreover, Streptococcus pneumoniae is the predominant organism found in PSS and not Pseudomonas aeruginosa.
In one patient, reported by Ommeslag et al (11), Pseudomonas aeruginosa was found in several blood cultures, suggesting that his keratitis originated from sepsis. The hypothesis of an endogenous dissemination was found unlikely by the authors because of the absence of retinal emboles on fundoscopy in the undamaged as well as in the damaged eyes. So they concluded that his keratitis originated from an exogenous source.
In our patient, the undamaged eye remained ophthalmoscopically clean before and after the corneal perforation of the left eye despite several episodes of sepsis with Pseudomones aeruginosa.
The hypothesis of an exogenous autoinoculation of the eye with organisms of the respiratory tract seems more likely in our patient whose bronchotracheal aspirates had been found pos-

itive for Pseudomonas aeruginosa several times. It is therefore more likely that the cause of this endophthalmitis was exogenous rather than endogenous.
The autoinoculation in ventilated patients by intubation can be the result of an enhanced bacterial adherence to bronchial mucosal cells (15,16,18), leading to inoculation of the eye when aspiration of the intubation tube is performed with uncovered or open eyes.
The normal respiratory tract efficiently removes bacteria that have been aspirated into the airways. It is extremely difficult to experimentally induce respiratory disease in animals by the aspiration of micro-organisms alone. Injury of the respiratory tract is also required. Viral infections, endotracheal intubation and chemical injury are factors that favor the induction of bacterial respiratory infections. Endotracheal intubation induces trauma to the mucous membranes so that micro-organisms can adhere to desquamated or desquamating epithelial cells and to the basement membrane if this is exposed, leading to the colonisation of the respiratory tract with micro-organisms. Pseudomonas aeruginosa is frequently found as this bacterium is all too often present in intensive care units.
A highly significant association has been found between copious sputum production and high colony count scores after suctioning the respiratory tract, suggesting that bacteria from the respiratory secretions were dispersed about the patients' eyes during suctioning (6). Observations of nurses carrying out the withdrawal procedure of endotracheal tubes revealed that patients' eyes were frequently uncovered and found open during the nursing procedure, allowing bacteria to be directly inoculated into the eye. Hilton et al (6) have analysed why the left eye was most commonly affected: right handed nurses tended to withdraw the catheter diagonally across the left side of the patient's face. A study emphasised that the eyes should be covered and the catheter not withdrawn across the patient's face after suctioning.
The eyes of critically ill patients are more exposed to injury (6). The absence of normal eyelid action and diminished tear production combined with the fact that the patients' eyes are often uncovered during nursing procedures, may contribute to the increased vulnerability of these eyes. It is generally agreed that the relative resistance of the healthy cornea to direct bacterial invasion is multifactorial: an intact epithelial layer, an optimal outer eye defence mechanism such as the lower temperature of the open eye, correct whipping and washing effect of the lids and tears, the presence of a certain concentration of secretory IgA, tear lysozymes and betalysins and the phagolytic activity of conjunctival mucosal and polymorphonuclear cells (10,19,20,22). Pseudomonas aeruginosa adheres little, if at all, to the normal cornea surface but collects in areas of damaged basal cells and the sites of injured epithelium (12, 14).
The combination of colonisation of the respiratory tract in intubated patients, the auto-inoculation through suctioning of the respiratory tract and the more vulnerable cornea in (semi) comatose patients makes it clear that these patients should be closely monitored.

CONCLUSION

Patients who are intubated should be followed and nursed carefully. They are definitely more at risk for developing an infectious keratitis because of a decreased corneal resistance and a higher concentration of micro-organisms present in the respiratory tract.
Special precautions must be taken in case of proven colonisation with Pseudomonas aeruginosa since this organism is known to cause a very rapid and very destructive corneal ulcer (13,21). Possible prevention is by means of special attention by the nursing staff while cleaning and suctioning the respiratory tract. Generous lubrication of the eyes is mandatory from the beginning on and a vigorous and prompt treatment is needed as soon as clinical symptoms, suggestive for keratitis appear.

RFERENCES

(1) ALFONSO E., KENYON KR. – Pseudomonas corneoscleritis *Am J Ophthalmol* 1987;103:90-98.
(2) BAUM JL., JONES DB. – Initial therapy of suspected microbial corneal ulcers *Surv Ophthalmol* 1979;24:97-116.
(3) CODÈRE F., BROWNSTEIN S., JACKSON WB. – Pseudomonas aeruginosa scleritis *Am J Ophthalmol* 1981;91:706-710.
(4) DART JKG., SEAL DV. – Pathogenesis and therapy of Pseudomonas aeruginosa keratitis *Eye* 1988;suppl 2:46-55.
(5) DE GROOT V. – Endogenous Pneumococcal endophthalmitis after splenectomy: report of two cases. *Bull.Soc.belge Ophtalmol.* 1992; 243: 147-151.
(6) HILTON E., ULISS A., SAMUELS S., ADAMS AA., LESSER ML., LOWY FD. – Nosocomial bacterial eye infections in intensive care-units. *Lancet* 1983;i:1318-1320.
(7) HUTTON WL., SEXTON RR. – Atypical Pseudomonas corneal ulcus in semicomatose patients. *Am J Ophthalmol.* 1972;73:37-39.
(8) JONES DB. – Decision making in the management of microbial keratitis *Ophthalmology* 1981;88:814-820.
(9) KROPEC A., HUEBNER J. – Exogenous or endogenous reservoirs of nosocomial Pseudomonas and Staphylococcus aureus infections in a surgical intensive care unit *Intensive Care Medicine* 1993;19:161-165.
(10) NANDA M., PFLUGFELDER SC., HOLLAND S. – Fulminant Pseudomonal keratitis and scleritis in Human Immunodeficiency Virus-infected patients.*Arch Ophthalmol* 1991;109:503-505.
(11) OMMESLAG D., COLARDYN F., DE LAEY JJ. – Eye infections caused by respiratory pathogens in mechanically ventilated patients. *Critical Care Medicine.* 1987; 15: 80-81.
(12) PARKIN B., TURNER A., MOORE E., COOK S. – Bacterial keratitis in the critically ill *Br J Ophthalmol* 1997; 81:1060-1063.
(13) RABER IM., LAIBSON PR. – Pseudomonas corneoscleral ulcers *Am J Ophthalmol* 1981; 92:353-362.
(14) RAMPHAL R., MCNIECE M., POLACK F. – Adherence of Pseudomonas aeruginosa to the injured cornea: a step in the pathogenesis of corneal infections *Ann Ophthalmol* 1981;421-425.
(15) RAMPHAL R., SMALL PM. – Adherence of Pseudomonas aeruginosa to tracheal cells injured by influenza infection or by endotracheal intubation. *Infect Immun* 1980;614-619.
(16) REDMAN LR., LOCKEY E. – Colonisation of the upper respiratory tract: *Anaesthesia* 1967;22-2:220-227.
(17) ROSENKRANZ L. – Pseudomonas keratitis. *Am J Ophthalmol* 1983;95:566-567.
(18) STERN GA., LUBNIEWSKY A., ALLEN C. – The interaction between Pseudomonas aeruginosa and the corneal epithelium: *Arch Ophthalmol* 1985; 103:1221-1225.
(19) STERN GA., WEITZENKORN D., VALENTI J. – Adherence of Pseudomonas aeruginosa to the mouse cornea *Arch Ophthalmol* 1982;100:1956-1958.
(20) STEUHL KP., DORING G. – Relevance of host-derived and bacterial factors in Pseudomonas aeruginosa corneal infections *Invest Ophthalmol Vis Sci* 1987;28:1559-1568.
(21) TWINING SS., LOHR KM., MOULDER JE. – The immune system in experimental Pseudomonas keratitis. *Invest Ophthalmol Vis Sci* 1986;27:507-515.
(22) WILSON LA. – Acute bacterial infection of the eye: bacterial keratitis and endophthalmitis. *Trans Ophthalmol Soc U.K.* 1986;105:43-60.

.....

Requests for reprints:
Wynants S.,
Universitair Ziekenhuis Antwerpen,
Wilrijkstraat 10, 2650 Edegem.