Haemorrhagic necrotic brain lesions and irreversible brain damage caused by *Serratia marcescens* in a premature neonate.

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Purpose

To report the case of a very low birth weight neonate born at 28+1 weeks gestation who developed a brain abscess caused by *Serratia marcescens* and to discuss the cause, pathophysiology, diagnostic and therapeutic management.

Background

Serratia marcescens is a rare cause of sepsis in premature neonates and neurological spread can be ominous. *S.marcescens* is a motile, encapsulated Gram negative bacillus which belongs to the *Enterobacteriaceae* family. It has been known to be an important opportunistic pathogen causing severe infections of various organ systems since the 1950s. Epidemic outbreaks of *S.marcescens* infection have been reported in neonatal intensive care units. The types of infection affecting newborns include septicemia, respiratory and urinary tract infections, omphalitis, meningitis and conjunctivitis. Cerebral infection is common among neonates and immunocompromised adults. Predisposing conditions in neonates include prematurity, respiratory distress syndrome, necrotising enterocolitis, neural tube defects, local wounds, intravenous and urinary catheters, parenteral nutrition, previous antibiotic therapy and tracheal cannulas.

Case Report

The baby girl was born by planned caesarian section at 28+1 weeks gestation to allow her mother to proceed to high strength chemotherapy for an aggressive carcinoma of the breast. At the time of delivery, the baby girl was in a stable condition with initial respiratory distress requiring surfactant and intubation and subsequent Continuous Positive Airway Pressure.

On day 6 of life there was a sudden deterioration in the health of the neonate and she was treated with antibiotics for suspected sepsis. There was subsequent deterioration over the following hours which resulted in severe respiratory acidosis requiring her to be intubated and ventilated. A cranial ultrasound was performed and identified bilateral widespread intracerebral bleeding. Repeat ultrasound after 24 hours demonstrated widespread damage with liquefaction of the original blood. Blood cultures were taken at the time of deterioration which were reported positive for ***Serratia marcescens***. Antibiotics were altered but because of the very poor prognosis redirection of care was agreed upon.

Clinical data	
Weeks of gestation	28+1
Birth weight (g)	1058
Apgar scores	7/10/10
Background/Risk factors	Maternal inflammatory breast cancer under chemo, prematurity
Colonisation (day,site)	6, Blood
Positive blood culture (day)	6
Abscess (day)	8
Specific antibiotics	Vancomycin, Ceftazidime then Meropenem
Start of treatment (day)	6 altered on 7

References

1) Cytotoxic Action of *Serratia marcescens* Hemolysin on Human Epithelial Cells, R. Hetle et al, Infect Immun. 1999 Feb; 67(2): 817–825.
2) Brain abscesses after *Serratia marcescens* infection on a neonatal intensive care unit: differences on serial imaging, A. Messerschmidt et al, Neuroradiology (2004) 46: 148–152.
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Pathophysiology

The haemolytic activity of *S.marcescens* is determined by two proteins, ShIA and ShIB that exhibit protein sequence homologues in *Proteus mirabilis*, *Haemophilus ducreyi*, *Edwardsiella tarda* and *Erwinia chrysantemi*. ShIA is activated by ShIB, integrates into the erythrocyte membrane and causes osmotic lysis through channel formation. In addition, ShIA forms pores in fibroblasts and epithelial cells.

S.marcescens as a pathogen has the capacity to damage tissue, and this depends on ShIA secretion. Adhesion to epithelial cells mediated by type 1 fimbriae of *S. marcescens* is also known. Fimbriae of *S. marcescens* contribute to superoxide production and phagocytosis.

Conclusion

Clinical signs are often non-specific when neonates are septic from *S.marcescens*. Sepsis caused by *S.marcescens* is often difficult to treat due to the high incidence of resistance to antibiotics such as beta-lactams and aminoglycosides. The use of cranial ultrasonography by the neonatal cot and early, aggressive antibiotic therapy are essential for these high-risk patients to minimise rapid spread and high mortality rate.

A: D1 Colour Doppler Normal systolic/diastolic flow

B: D7 Colour Doppler Reverse diastolic flow (brain oedema)