- Dissemination of Acinetobacter in the environment can be a major problem.
- It has been recovered from respiratory equipment, bed linen, tables, patients' charts, sink traps, the floor and atmosphere, especially in the vicinity of an infected or colonized patient
- Furthermore, Acinetobacter is able to persist in the environment for several days, even in dry conditions, on particles and dust.
- Some strains are tolerant to soaps and disinfectants.
- The nosocomial spread of Acinetobacter is most often attributed to exogenous contamination from equipment, environmental surfaces and the hands of hospital personnel rather than endogenous infection.
- Carbapenems are currently considered the antimicrobials of choice, although epidemic outbreaks and endemic situations involving carbapenem-resistant Acinetobacter species have been described
 - Colistin, polymyxin B & ampicillin-sulbactam have all been described in treatment of carbapenem-resistant strains

transmission

Acinetobacter [created by Paul Young 02/10/07]

treatment

- Acinetobacter are non-lactose fermenting, Gram-negative coccobacilli that are strictly aerobic and non-motile.

- Acinetobacter baumanii is the most important species associated with infections and nosocomial outbreaks

 levels of environmental contamination with A. baumannii correlate with patient colonization and infection. This organism is very hardly and survives dessication.

> - Acinetobacters form part of the normal bacterial flora of the skin, particularly in moist regions such as the axillae, groin and toe webs.

- Up to 25% of normal individuals carry cutaneous Acinetobacter, and it is the most common gram-negative organism isolated from the skin of hospital personnel.

- It causes a wide range of nosocomial infections including ventilator-associated pneumonia, bacteraemia, urinary tract infections, skin and wound infections and meningitis

- Acinetobacter isolates are typically even more resistant than Pseudomonas spp. to most antimicrobials, including broad-spectrum cephalosporins, penicillins, fluoroquinolones and aminoglycosides.

 Known resistance mechanisms include plasmid-mediated beta-lactamases, which are also frequently associated with resistance to fluoroquinolones and aminoglycosides.

- Chromosomal cephalosporinases may be responsible for the high prevalence of ceftazidime resistance. However, the relationship between observed antibiotic resistance patterns in vitro and the presence of these beta-lactamases remains unclear. It is suggested that altered penicillin-binding proteins and membrane impermeability may be the major cause of high level resistance to beta-lactams, including imipenem

colonisation

General

infections

resistance