

Double Trouble...



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Abstract

Acinetobacter baumannii and *Candida auris* are associated with nosocomial outbreaks. They can be multidrug resistant and are highly transmissible between patients and the environment. To have both in one patient is certainly a management and infection control dilemma. A 56 year old male had road traffic accident in Nairobi, Kenya, and subsequently had craniotomy for brain haemorrhage/oedema, chest drains for haemothorax and pneumothorax and antibiotics (Vancomycin and meropenem) when he spiked temperatures while in a hospital in Kenya.

He was stabilised and transferred to the UK and was isolated in an ICU room. *C. auris* was initially isolated from his blood culture and it was only fully sensitive to Amphotericin and Flucytosine. Subsequently, *A. baumannii* was isolated but it was resistant to all antibiotics tested including meropenem (Tigecycline had the lowest MIC) but for Colistin. Both organisms were also isolated from other body fluids and only *Acinetobacter* was identified in the pleural fluid.

Though high doses of Caspofungin and Amphotericin treated the *C. auris* effectively, the inflammatory markers only improved after Meropenem was added and Colistin dose increased in addition to the high dose Tigecycline. This illustrates the effectiveness of high dose combination therapy in situations of confirmed phenotypic resistance. Two other patients got colonised with the *Acinetobacter* leading to a 24 hour shut down of the ICU. Screening and environmental sampling was negative but decontamination and strict infection control practices ensured no new cases were identified subsequently.

While MALDI-TOF correctly identified *C. auris*, The VITEK wrongly identified it a *Candida haemulonii*

Results

Table 1: Sensitivity and MIC results

<i>Acinetobacter baumannii</i>	<i>Candida Auris</i>
Amikacin- R	Amphotericin- S (0.5)
Gentamicin-R	Caspofungin- R (0.5)
Tobramicin- R	Flucytosine- S (<0.125)
Ceftazidime-R	Miconazole- I (4)
Imipenem- R	Voriconazole- I (0.5)
Meropenem- R	-
Piperacillin/ Tazobactam-R	-
Ciprofloxacin- R	-
Minocycline-I (8)	-
Colistin- S (<0.5)	-
Ceftazidime/Avibactam- (64)	-
Ceftolozane/Tazobactam- (24)	-
Fosfomycin- (128)	-
Tigecycline- (8)	-

Please Note: for sensitivities (R=Resistant, I= intermediate, S=Sensitive). MICs (mg/L) are indicated in the brackets. Where no sensitivities are indicated for the MICs, interpretation are derived from other methods such as PK/PD, CLSI, closest species, etc. Results were confirmed from the reference laboratory except for those antibiotics in red print.

Introduction

Antibiotic resistance is a major concern globally. *Acinetobacter* spp and *Candida auris* are organisms associated with significant antimicrobial resistance and have been associated with nosocomial outbreaks. They can be multidrug resistant and are highly transmissible between patients and the environment. Multi-drug resistant *A. baumannii* strains with additional resistance to carbapenems (MRAB-C) have been identified in several UK hospitals including paediatric settings. *C. auris*, since it was first identified in Japan, has continued to be a major concern; not just because it can cause fatal bloodstream infections that are difficult to treat, but also because it can be mis-identified in the laboratory causing a delay in initiating infection control practices which ensures that it can cause prolonged outbreaks. There is also little evidence that regular decontaminants like chlorhexidine are effective in killing the fungus. The screening process for both organisms is a little easier because similar sites (for instance, the nose, throat, perineum, wounds, sputa, tracheostomy, faeces, ante-cubital fossa) but the economic implications to the NHS cannot be ignored. To have both in one patient is certainly a management and infection control dilemma. This is not helped by the fact that there is no clearly defined approach for treating resistant organisms when there are few antibiotic options. It is often left to the discretion of the specialists.

Discussion

Caspofungin, and amphotericin treated the *C. auris* effectively. The inflammatory markers only improved after high dose meropenem (2g tds) was added and the colistin dose (increased from 3MU tds to 4MU tds) in addition to the high dose tigecycline (100mg BD) at about 5 days into treatment. Treatment continued for 2 weeks subsequently. This illustrates the effectiveness of high dose combination therapy in situations of confirmed phenotypic resistance.

Strict infection control practices ensured that no new cases of *C.auris* were identified. The two patients that were colonised with *Acinetobacter* were both isolated and staff practices, hand hygiene practices, use of fomites and equipment, and care bundles were reviewed and corrected. Screening and environmental sampling was negative and it was believed that staff spread was the likely reason for the spread. Terminal clean was done using hydrogen peroxide for the patient cubicles and subsequently the whole of the ICU. Flucytosine, as well as change of the urinary catheter, was used to treat the urinary *C. auris* in the index case and topical terbinafine was used at cannula entry sites.

While MALDI-TOF correctly identified *C. auris*, The VITEK wrongly identified it as *Candida haemulonii*. The availability of the MALDI-TOF and its prompt use has ensured that the fungus was identified early to ensure that infection control acted swiftly to avert further spread of the organism.

The Case

A 56 year old man had road traffic accident in Nairobi, Kenya, and subsequently had craniotomy for brain haemorrhage/oedema, chest drains for haemothorax and pneumothorax and received antibiotics (Vancomycin and Meropenem) when he spiked temperatures while in hospital in Kenya. He was stabilised and transferred to the UK and was isolated in an ICU room. *C. auris* was initially isolated from his blood culture and was only fully sensitive to Amphotericin and Flucytosine. Subsequently, *A. baumannii* was isolated which was resistant to most antibiotics tested including meropenem (Tigecycline had the lowest MIC) except for Colistin. Both organisms were also isolated from other body fluids (*Acinetobacter* was identified in the pleural fluid, *C. auris* was also identified in the urine).

Two other patients acquired colonisation with *Acinetobacter* which had the same genotype as that of our index case. Both were isolated and strict infection control practices were initiated. No secondary cases of *Candida auris* were noted.

Learning Points

1. The use of high dose antibiotics in combination can be used to treat infections caused by multiresistant organisms even when Laboratory results indicate resistance.
2. Early identification of multiresistant organisms is essential for quick control of its spread.
3. The MALDI-TOF and recent technology can be very useful in the early identification of *Candida auris*

References

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