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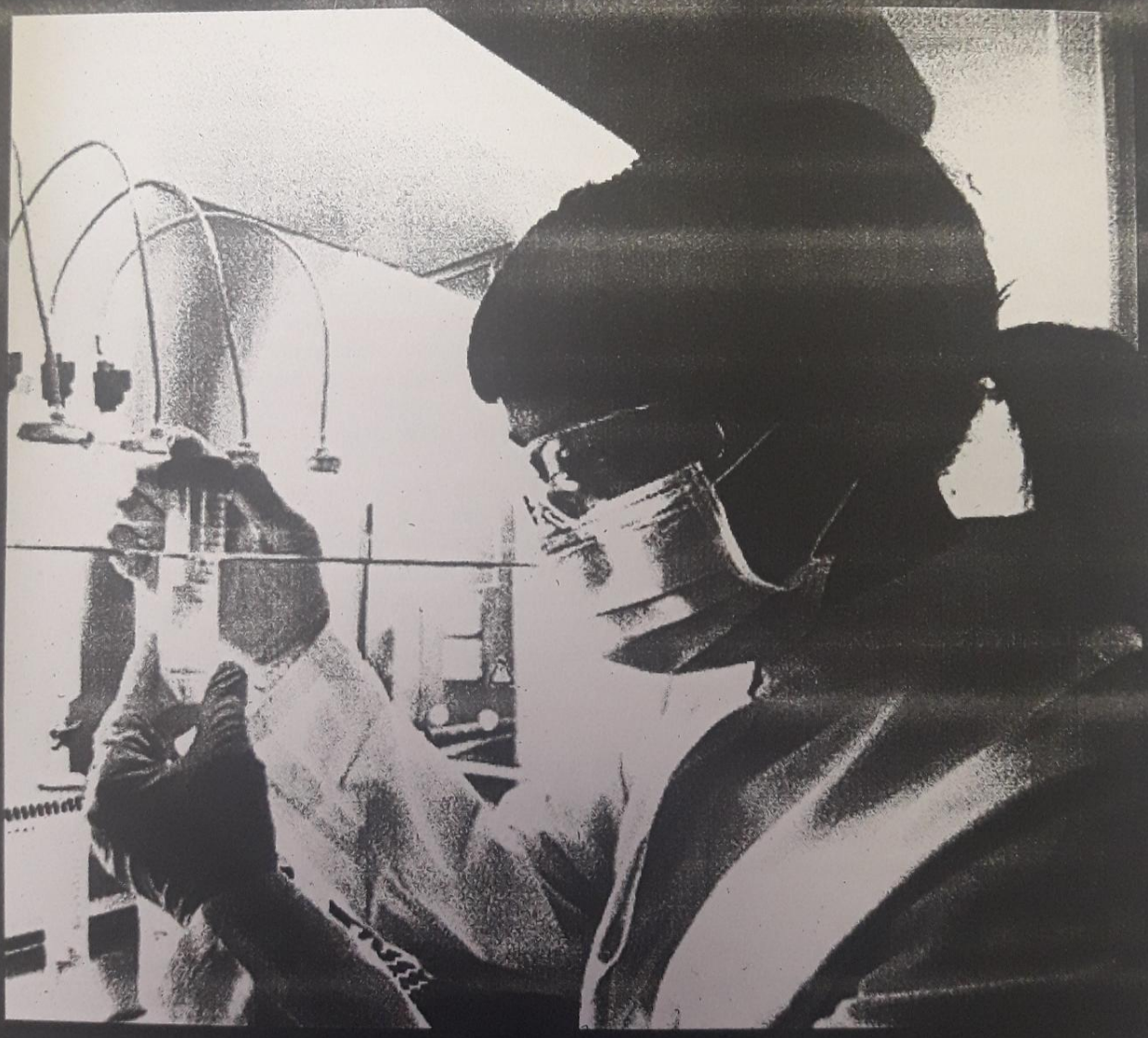

VRE: A SUPER BUG INFECTION FOLLOWING COLONIZATION

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Introduction

A 28-year old lady was transferred from a local hospital to the Intensive Care Unit (ICU) of Princess Alexandra Hospital, Australia. On admission she was febrile with a temperature of 39 °C, her Glasgow coma scale was 6, respiratory rate was 50 per minute, pulse rate was 131 per minute, and oxygen saturation was 67% hence the diagnosis was type 1 respiratory failure. Chest X ray showed bilateral lung infiltrates while influenza A H1N1 DNA detected from nasopharyngeal aspirate.

The patient had to be managed connected to extracorporeal membranous oxygen (ECMO) providing instrument for more than two weeks and by that time she had been on intra venous ceftriaxone, azithromycin and oseltamivir which she was started initially.

Despite of antimicrobial treatment the patient was febrile continuously. Routine screening carried out for multi resistant organisms was positive for vancomycin resistant enterococci (VRE). As the patient was confirmed colonized with VRE, the clinical team in charge changed the treatment to linezolid and gentamicin and isolated the patient. Although septic screening done culturing specimens from different sites in order to isolate the probable causative organisms was negative initially, later VRE organisms were isolated from blood and urine specimens.

Both isolates from the clinical samples had minimal inhibitory concentrations (MIC) of 32 µg/ml and 2 µg/ml for vancomycin and teicoplanin respectively. All VRE organisms isolated from the patient were of Van B phenotype.

At the time of discharge, the patient had spent 40 days in ICU, had received long term expensive antibiotics and was nursed isolated with VRE control precautions.

Discussion

The super bug vancomycin-resistant enterococci (VRE) was first reported in France and Europe in 1988 (1). Out of all enterococcal species, most of the human infections are caused by *Enterococcus faecalis* (80-90%) and *Enterococcus faecium* (5-10%).

Although multiple factors predispose to infection with VRE, colonization precedes most. In the United States, nosocomial transmission of VRE from patient to patient had been reported (2). A study confirmed that, during the routine examination of patients colonized with VRE, 67% of examiner's gowns, gloves, and stethoscopes became contaminated with the organism (3).

To minimize the colonization and subsequent infections with VRE, all the patients at potential risk are screened in Australian health care setting. Rectal, perineal and groin swabs or faeces (with a special request) are collected and transported to the laboratory. In the laboratory the swabs are added to VRE (Enterococcosel) broth containing 6 mg/L of vancomycin, azide and esculin. After 24 hours of incubation VRE broths which have been changed in to black colour only are subculture on Colombia colistin nalidixic acid (CNA) agar plates. All the preliminary tests are carried out for the isolated colonies. VRE are Gram positive, catalase positive, L-pyrrolidonyl-beta-napthylamide (PYR) test positive organisms. The suspected VRE are identified by VITEK 2 automated machine which provides a definitive identification of organisms. MIC for vancomycin and teicoplanin is detected by epsilometer test (E test). The isolates reporting MIC for vancomycin more than 4 µg/ml are sent to the central laboratory for genotyping which confirms VRE.

Enterococci acquire resistance to several classes of antibiotics either by mutation or by receipt of foreign genetic material through the transfer of plasmids and transposons (4). Six different types of vancomycin resistance are shown by VRE, namely Van-A, Van-B, Van-C, Van-D, Van-E and Van-F. Of these, only Van-A, Van-B and Van-C have been seen in clinical practice. The importance is that Van-A VRE is resistant to both vancomycin and teicoplanin, Van-B VRE is resistant to vancomycin but sensitive to teicoplanin, and Van-C is only partly resistant to vancomycin, and sensitive to teicoplanin.