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Case report

Non-hemolytic group B streptococcus as a cause of chemotherapy port infection

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Introduction

Lancefield Group B Streptococci (GBS) are common causes of infection in adults with diabetes as well as a classical cause of perinatal meningitis. The organism is classically a beta-hemolytic streptococcus. We present an unusual case of human infection by a phenotypically rare manifestation of GBS, in which a chemotherapy port exhibited bacteremia of an organism without hemolysis (so-called gamma hemolysis).

Case report

A 63 year old male was admitted for fever, chills, and altered mental status. He had been recently diagnosed with pancreatic carcinoma and had had a stent placed in his common bile duct. One week before this admission the patient also had a chemotherapy port placed. On admission he had not yet started chemotherapy, and the port had no local signs or symptoms of infection. His leukocyte count was 15,000/μl and he had no clear focal findings on neither physical examination nor chest and abdominal imaging. 2 blood cultures were positive for Gram positive cocci in pairs and chains which revealed a gamma (or non-) hemolytic streptococcus that was subsequently identified as a Group B streptococcus. In the absence of another source, the port was explanted and the patient recovered without further positive cultures.

Discussion

While chemotherapy port infections are not uncommon, the microorganism isolated from this particular patient is. Non- (gamma) hemolytic Group B Streptococcus comprises only 1–3% of the entire GBS population [1,2]. Its unorthodox characteristics arise from its impaired expression of 2 clinically significant molecules; β-hemolysin/cytolysin, and Granadaene [1,3].

β-hemolysin is a pore forming toxin that provides Lancefield Group B Streptococcus with its classic beta-hemolytic activity when plated on blood agar [1–4,9]. GBS's unique version of the molecule is coded for by the *cyl* operon, a cluster of 12 genes that collectively must be entirely functional for complete synthesis and expression of the molecule. Mutations throughout any portion of this gene cluster or its transcriptional regulation have been shown to cause defective β-hemolysin expression [1]. Some well-documented examples include truncated, nonfunctional forms of the molecule or lack of β-hemolysin export due to an impaired protein transporter [1–4,10].

Without β-hemolysin, non-hemolytic strains of GBS are without a potent virulence factor associated with increased levels in invasion, especially in lung epithelia. In fact, studies have shown that both non-hemolytic GBS strains and classically beta-hemolytic strains combined with a β-hemolysin inhibitor exhibited significantly reduced levels of cellular invasion and pro-inflammatory cytokine release [5]. This finding however, does not mean that non-hemolytic strains are non-pathogenic. Non-hemolytic GBS strains are still able invade the amniotic cavity during pregnancy or preterm labor, albeit at a lower frequency than the wild-type, and cause invasive infections such as meningitis and endocarditis [6–8].

The *cyl* operon also codes for Granadaene, an isoprenoid polyene that provides GBS with its characteristic red-orange "brick" colored pigmentation when cultured on Granada medium [3,8]. Due to similarly involved gene locations, mutations resulting in defective GBS β-hemolysin expression have been highly associated with the absence of this identifying pigmentation on cultures [11].

Granadaene has been identified with antioxidant properties and has been proposed as a possible source of membrane lipid protection against reactive oxygen species associated with phagolysosomal killing [11]. However, studies have yet to show significant differences in survivability between non-pigmented strains and their wild-type counterparts against macrophages [12]. The significance of lacking this pigment molecule becomes more notably apparent in the clinical diagnosis of infection, as preliminary cultures could potentially lead one down a different identification path.

Conclusion

We present an unusual case of GBS infection causing an infusion port infection with an organism that was non-hemolytic. Non-hemolytic GBS represent only 1–3% of all GBS and are generally are caused by

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mutations in synthesis or a transporter that normally exports the β-hemolysin extracellularly. The mutation is also associated with the lack of GBS orange/red pigment production. These non-hemolytic GBS isolates are generally considered to be less virulent but not non-pathogenic. It is important to remember that non-hemolytic streptococci can be pathogenic and may be part of GBS that are usually beta-hemolytic.

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