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Review Article

Fusobacterial head and neck infections in children

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ABSTRACT

Fusobacterium species are increasingly recognized as a cause of head and neck infections in children. These infections include acute and chronic otitis, sinusitis, mastoiditis, and tonsillitis; peritonsillar and retropharyngeal abscesses; Lemierre syndrome; post-anginal cervical lymphadenitis; and periodontitis. They can also be involved in brain abscess and bacteremia associated with head and neck infections. This review describes the clinical spectrum of head and neck fusobacterial infection in children and their management.

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Contents

1. Introduction

Fusobacterium species inhabits the oropharynx, gastrointestinal tract, and female genital tract and are important potential pathogens in children [1]. These organisms are increasingly recognized as a cause of head and neck infections in pediatric patients. These include acute and chronic otitis, sinusitis, mastoiditis, and tonsillitis; peritonsillar and retropharyngeal abscesses; Lemierre syndrome; post-anginal cervical lymphadenitis; periodontitis; and bacteremia and brain abscess complicating these infections [2–7].

This review outlines the clinical spectrum of fusobacterial head and neck infection in children and their clinical management.

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2. Epidemology

Fusobacterial infections and their invasive and disseminated complications have different age related patterns: in children they usually originate from the middle ear and cervical lymph nodes; in adolescents from the throat and tonsils; in adults from the sinuses, carious teeth and periodontium, and in older adults from the gastrointestinal or genitourinary tracts [5–7].

The recently observed increase in reports about recovery of Fusobacterial species in head and neck infections in children may be due to the decrease in the number of tonsillectomies, increased utilization of corticosteroids for infectious mononucleosis, decreased empiric administration of antibiotics for sore throat, otitis and sinusitis, improvement in blood culture methodologies and techniques for isolation and identification of anaerobic organisms, and usage of molecular diagnostic methods such as polymerase chain reaction (PCR) for the identification of *Fusobaterium* spp. [2–4]. Precise estimates, however, of the true incidence of isolation of *Fusobaterium* spp. in head and neck infections have been complicated by the difficulties in recovery of anaerobic bacteria, and the required use of special methods of specimen transportation and cultivation needed for their isolation.

3. Microbiology

Fusobacteria are strict anaerobic gram-negative, thin, long, filamentous, nonmotile, and nonsporulating. The genus *Fusobacterium* is a heterogenous group of 13 species. The clinically important group members are *Fusobacterium necrophorum*, *Fusobacterium nucleatum*, *Fusobacterium gonidiaformans*, *Fusobacterium naviforme*, *Fusobacterium mortiferum*, and *Fusobacterium varium* [8–10]. More than half of fusobacterial infections are polymicrobial, and unlike other Gram negative anaerobic bacteria *Fusobacteriae* can invade the human host as primary pathogens [2,3]. Studies in animals demonstrated *Fusobacteriae* increased virulence in the presence of other gastrointestinal and oral aerobic and anaerobic flora organisms [11].

4. Pathogenesis

Underlying host factors can predispose children to fusobacterial infection. A molecular thrombophilic predisposition was observed in children with invasive infection [12–15], and a single nucleotide polymorphism in the toll-like receptor 5 gene was found in an affected child [12].

Because Fusobacteria are part of the normal flora of the oral cavity they can cause a contiguous, polymicrobial infection adjacent to these sites, as well as distant locations [1]. These infections are characterized by blood vessel invasion, inflammation, and thrombosis. Compromised blood supply or tissue injury follows accidental or surgical trauma can facilitate low oxidation–reduction potential which enhances fusobacterial bacterial growth [2].

Fusobacterial virulence factors include the production of lipopolysaccharide capsule [16], leukocidins, lipases, DNAases, hemolysins, hemagglutinins, neutrophil-cytotoxic factors, deoxyribonuclease [17–19], and the ability to aggregate platelets (*F. necrophorum*) [4,7,9], and produce proteolytic enzymes, that enhance invasion [20].

Fusobacteria (mostly by *F. nucleatum*) can produce the enzyme beta-lactamase, which protects these organisms as well as other co-pathogens against beta-lactam antibiotics [19]. Fusobacteria can enhance the growth of other anaerobic and aerobic organisms as was demonstrated in animal studies [11,21].

Fusobacterial infections often occurs as a result of disruption of the normal mucocutaneous barrier that leads to tissue invasion. This may occur in Epstein–Barr virus pharyngitis [22] or in mucositis due to chemotherapy and neutropenia. *F. necrophorum* is second only to the Group A beta hemolytic streptococci (GABHS) amongst bacterial pathogens causing sore throat [23,24]. Tissue adhesion and invasion by *F. necrophorum* may be dependent on viral co-infection [25,26].

5. Clinical manifestations

Fusobacterial infection is often localized but can also be invasive. *Fusobacterium* species are associated with a variety of clinical infections that are age dependent, with a bimodal occurrence in adolescents and the elderly [27,28]. When adequate methods are used for cultivation and isolation of anaerobic bacteria, fusobacterial infections are often found to be polymicrobial, where the number of isolates of 5–10 organisms per site. The type of co-pathogens depends on the body site and the circumstances leading to the infection [2].

5.1. Bacteremia and Lemierre syndrome (postanginal sepsis)

About 3% of all cases of anaerobic bacteremia and 5% of all anaerobic infections in children are caused by *Fusobacterium* spp. [27–29]. The majority of the infections are caused by *F. necrophorum* and *F. nucleatum.* Bacteremia in children is generally associated with a primary focus of infection in the head, neck, or upper respiratory tract [27]. Primary parapharyngeal infection usually spreads to the blood vessels causing a local septic thrombophlebitis and subsequent septic embolization, leading to necrotizing pneumonia or involvement of multiple viscera or joints. This clinical syndrome was described by Lemierre as postanginal sepsis [30,31].

F. necrophorum is the most common species causing Lemierre syndrome. Other Fusobacteria include *F. nucleatum*, *F. gonidiaforum* and *F. varium.* Other isolates recovered alone or in combination include pigmented *Prevotella*, *Bacteroides* and *Peptostreptococcus* spp. [30].

The most common symptoms and clinical findings of Lemierre syndrome are: sore throat, fever, rigors (a distinctive feature of this disease), neck mass and pain, trismus, tenderness along the neck vasculature, malaise, anorexia, chest pain, shortness of breath, cough, and prostration [4,6,7,13,31]. It usually occurs in a previously healthy adolescent male who develops sore throat, followed by fevers and rigors on the fourth or fifth day, and painful swelling in the neck (often wrongly attributed to lymphadenopathy) [4,6,7,13]. Metastatic foci of infection usually occur in the lungs, but also can emerge in muscles, bones, joints, liver, spleen, skin, and endocardium [32].

Isolation of *Fusobacterium* spp. from blood should lead to investigation for primary and metastatic foci of infection. Primary infection has been associated with lymphadenitis or infectious mononucleosis and can be trivial in nature and easily overlooked.

5.2. Head and neck infections

Fusobacterium is implicated in approximately half of anaerobic infections of the head and neck, including gingival and dental infections, [32] chronic tonsillitis, [33] chronic sinusitis, [34] acute, [35,71] and chronic otitis media, [36,37] mastoiditis, [38–40] and parapharyngeal [41,42] and mouth floor infections. *F. nucleatum* is the most commonly isolated species. Fusobacterial sinusitis can be dangerous, especially because contiguous spread through tissue planes can take place [22].

Fusobacterium spp. were recovered from 4 to 8% of children with chronic otitis media [36,37] and from 8% with chronic mastoiditis [38].

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