ABSTRACT

Tonsilitis is a common pharyngeal infection affecting young adults and children. Peritonsillar abscess (PTA) is an occasionally identified complication and intratonsillar abscess (ITA) is rarely reported. Prevailing models of the reported pathogenesis of tonsillitis, PTA and ITA were found lacking, suggesting that these conditions were either unrelated or the general conceptual framework was inadequate.

A PTA is a collection of pus between the tonsil fibrous capsule and the pharyngeal constrictor muscles. Individuals commonly present with uvular deviation, fever, odynophagia, muffled voice and dysphagia. The true pathophysiology of PTA formation is unclear. Authors have proposed that blockage of tonsillar crypts results in inflammation and acute tonsillitis. Outward drainage is prevented, leading to failure of the suppurative focus to drain and thus coalescence by extension into the peritonsillar space. Others have proposed PTA as a consequence of abscess formation in salivary glands in the supratonsillar space.

INTRODUCTION

We postulate that acute tonsillitis represents a cellulitis of the tonsil epithelium. The lack of subepithelial lymphatic channels results in bacterial antigens, but not bacteria, transported to lymph nodes for antigenic processing.

Methods: The histopathology of cases of acute tonsillitis, ITA and PTA were reviewed and classified.

Results: Prevailing models of ITA were able to account for only a minority of cases. Evidence of hematologic spread of infection as the cause of ITA or PTA is weak. Discussion: A novel unifying model of tonsillitis, ITA and PTA is presented. We postulate that acute tonsillitis represents a cellulitis of the tonsillar epithelium. The lack of subepithelial lymphatic channels in the tonsil allows rapid transport of bacterial antigens, but not bacteria. Rapid lymphatic transport quickly involves regional lymph nodes in antigenic processing. When direct extension of infection penetrates the surface epithelium into core lymphatic channels, rapid intra-tonsillar lymphatic transit makes true ITA unusual. Local infectious agent factors and relative lymphatic channel obstruction caused by rapid swelling of tonsillar follicles may combine to foster development of PTA and much less commonly ITA. Lymph flow may be less superiorly than more dependent inferior routes accounting for the superior location of many PTA. The development of PTA further compromises lymphatic flow within the tonsil predisposing the patient to ITA development. The model accounts for the frequent association of PTA and ITA, the development of both crypt and true ITAs, and the location of PTA.

METHODS AND MATERIALS

The histopathology of 2 cases of ITA and 10 representative cases of PTA and acute tonsillitis were reviewed and classified.

RESULTS

Acute tonsillitis specimen show ulceration of stratified squamous surface epithelium with invasion of neutrophils (PMN). No abscess formation or inflammation deep to surface.

PTA specimen show erosion of surface epithelium with PMN invasion. The tonsillar parenchyma is uniformly unremarkable throughout with hyperplasia of follicles. A collection of PMN infiltration, tissue necrosis and abscess formation is found deep; between tonsillar fibrous capsule and skeletal muscle of the pharyngeal wall.

ITA specimen display erosion of stratified squamous surface epithelium and sheets of PMN extending into tonsillar crypts. Abscess is completely surrounded by unremarkable appearing tonsillar parenchyma. One specimen also reveals concomitant PTA with abscess formation between tonsil and pharyngeal wall.

REFERENCES