CORRELATION BETWEEN PRESUMED SINUSITIS-INDUCED PAIN AND PARANASAL SINUS COMPUTED TOMOGRAPHIC FINDINGS


Purpose of the Study. Sinusitis is typically a clinical diagnosis based on history and symptoms. This study investigated the correlation between clinical symptoms of facial and/or head pain and actual localized findings consistent with a sinusitis on sinus computed tomographic (CT) imaging.

Study Population. Two hundred patients with a clinical history of sinusitis that were referred by their internist or otolaryngologist for CT imaging of the paranasal sinuses.

Methods. Before the CT scanning, each patient was asked to complete a sinus questionnaire that inquired about pain in 8 different areas of the head and neck, the duration of illness, use of allergy medications, smoking, pets, and seasonal variation of pain symptoms. All CT scans were independently scored by 3 radiologists who were blinded to the patients’ questionnaire responses. The scores were then averaged.

Results. Eighty-two percent reported having some form of facial pain or headache; the right temple/forehead was the most commonly reported site. Six percent were considered to have acute sinusitis, 14% had a history of sinus surgery, 12% were smokers, and 53% owned pets. Nine percent had no abnormalities on the CT scan. The maxillary sinus was the most frequently (68%) involved sinus. No correlation could be found between the reported sites of pain and findings on CT. Furthermore, no relationships were found between the sinus CT findings and smoking, owning a pet, or duration of pain symptoms. Similar numbers of sites of pain (5.45 and 5.88) were reported between patients with and without CT findings.

Conclusions. This study demonstrated that there was a lack of correlation between reported site of pain and CT findings. Two key points the author stated are 1) symptoms of pain alone may not be sufficient to diagnose sinusitis and 2) the limited value of CT scans for evaluating patients with facial pain/headaches only or patients with a low suspicion for sinusitis.

Reviewers’ Comments. Most clinical diagnoses of sinusitis involve more than facial pain or headache alone. The diagnosis is based on a compilation of symptoms that may include fever, facial swelling/tenderness, purulent drainage, cough, malodorous breath, and nasal congestion. It is not clear that these patients had more than facial pain alone, which would not be sufficient for clinical diagnosis. Although these patients were referred by internists and otolaryngologists for imaging of their paranasal sinuses, the specific indications for the CT scan are not clear. For example, CT scans may have been ordered after antibiotic treatment to rule out structural abnormalities. Overall, this article does make an important point that facial pain does not equate with sinus disease.

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Asthma

PATHOPHYSIOLOGY

VIRAL INDUCTION OF A CHRONIC ASTHMA PHENOTYPE AND GENETIC SEGREGATION FROM THE ACUTE RESPONSE


Purpose of Study. To address the role of persistent infection and cytokine bias in the development of the chronic asthma phenotype after paramyxoviral infection.

Methods. The investigators used a mouse model of paramyxoviral bronchiolitis with acute pathology similar to the human condition. Wild-type C57BL/6J, same-strain interferon gamma (IFN-γ)-null mice and same-strain intercellular adhesion molecule-1 (ICAM-1)-null mice were maintained under pathogen-free conditions for study at 7 to 9 weeks of age. Mice were inoculated with mouse influenza virus type 1 (Sendai virus; SeV Fushimi strain) or ultraviolet (UV)-inactivated SeV. Histochemistry of the mouse lung, bronchoalveolar lavage fluid analysis, and airway reactivity measurements to aerosolized methacholine were performed. In addition, allergen challenges were performed with ovalbumin using sensitized and nonsensitized C57BL/6J mice.

Results. Following a single paramyxoviral infection of mice (C57BL6/J strain), the investigators demonstrate that not only does this produce acute bronchiolitis, but also a chronic lung response with airway hyperreactivity and goblet cell hyperplasia lasting at least 1 year after complete viral clearance. During the acute response to virus, same-strain ICAM-1-null mice are protected from airway inflammation and hyperreactivity despite similar viral infections rates; however, the chronic response proceeds despite ICAM-1 deficiency. Neither response is influenced by IFN-γ deficiency, but the chronic response is at least partially prevented by glucocorticoid treatment. In contrast to viral infection, allergen challenge caused only short-term expression of asthma phenotypes.

Conclusions. Paramyxoviruses cause both acute airway inflammation/hyperreactivity and chronic airway remodeling/hyperreactivity phenotypes. These 2 phenotypes can be segregated by their dependence on the ICAM-1 gene and so depend on distinct controls that appear critical for the development of lifelong airway diseases such as asthma. These findings raise the possibility that asthma not only resembles a persistent antiviral response, but also may be caused by such a response. These data may help
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