


**Throw Caution to the Wind Instruments**

*To the Editor:*

We read with interest the two articles in CHEST (September 2010) from Metzger et al1 and Metersy et al2 that implicate wind instruments as a possible source of antigens triggering hypersensitivity pneumonitis (HP). We commend these authors for rigorously documenting antigen sensitization in one patient and documenting a classic clinical course of HP in another. Taken together, these articles provide evidence that suggests a causal relationship between microorganisms present in wind instruments and HP. However, we caution that overemphasizing the role of wind instruments not only stigmatizes wind instruments as a cause of lung disease but also creates a pitfall for clinicians that could cause other types of exposure and other types of interstitial lung disease to be overlooked.

As a case in point, we consulted on a 62-year-old male music teacher and professional saxophone player who sought care for a low-grade fever associated with fatigue and a dry cough. He had no other significant medical history. Physical examination was notable for bibasilar inspiratory rales. A panel of autoantibody tests was negative. Pulmonary function tests demonstrated normal spirometry, borderline lung volumes (total lung capacity, 81% predicted), and impaired diffusion capacity of the lung for carbon monoxide (62% predicted). High-resolution CT scan revealed subpleural reticulation, mild architectural distortion, and bronchiolectasis within the middle and lower lung zones. A surgical lung biopsy specimen demonstrated patchy interstitial fibrosis around the small airways accompanied by small lymphoid aggregates, multinucleated giant cells, and numerous loosely formed granulomas in a peribronchioriolar distribution. This pattern was diagnostic for HP.

Upon further questioning, the patient revealed that rotting wood was removed from his home bathroom prior to the onset of symptoms, implying a likely diagnosis of “dry rot lung” that is associated with Merulius.3 Specific antibodies were not obtained at the time of the initial consultation, yet based on this history, there was little risk of reexposure. The patient was treated with a low dose of oral corticosteroids for several months, and pulmonary function improved. More importantly, he continued to play the saxophone without interruption and without extraordinary measures to clean the instrument.

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**REFERENCES**


**Response**

*To the Editor:*

We would like to thank Drs Rackley and Meltzer for their interest in our recent report in CHEST (September 2010)1 of a patient with hypersensitivity pneumonitis (HP) due to use of a trombone colonized with *Fusarium* sp. and *Mycobacterium chelonae abrascessus* group organisms. Although we do not think we were guilty of overemphasizing the role of wind instruments in HP, or stigmatizing wind instruments, Drs Rackley and Meltzer correctly caution pulmonologists against assuming that a wind instrument is always the cause of pulmonary symptoms or interstitial lung disease in an exposed patient. A complete exposure history is always important, as more than one potentially relevant exposure may occur. This is especially true in the case of wind instruments. Hundreds of thousands of children and adults are regularly exposed in the United States, and the vast majority do not develop HP.

Although we would no more wish to stigmatize all wind instruments than Drs Rackley and Meltzer would stigmatize all bathrooms, wind instruments, like bathrooms, provide the perfect environment for the growth of pathogenic organisms. The inside of a wind instrument is warm and moist and provides nutrients in the form of desquamated cells (seen in the biofilm from the instruments we studied) and saliva that are introduced into the instrument during use. The magnitude of contamination of these instruments is quite extreme. Assuming that one organism visible per 100 X field is approximately equivalent to 10^6 organisms/mL, the biofilm obtained from several of the instruments we studied contained > 10^6 organisms/mL, higher than the colony count seen in most outbreaks of HP due to contaminated metalworking fluids and hot tubs.

Although the likelihood of wind instrument-related disease is low in any single individual, given the large number of exposed individuals and the degree of contamination, it is highly likely that there are substantial numbers of afflicted persons. Our report received significant attention in the lay press and prompted responses from more than one musician who recounted similar respiratory symptoms that remitted when they started to more diligently clean their instruments. Other musicians pointed out prior reports of similar illness in bagpipers, previously referred to as piper’s lung. We reiterate our conclusion that pulmonologists should consider the possibility of HP in wind musicians who have