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Qualitative Studies to Explore Barriers to Spirometry Use: A Breath of Fresh Air?

In the December 2005 issue of *RESPIRATORY CARE*, Kaminsky et al reported a pilot study on primary-care practitioners' knowledge about and use of spirometry for detecting chronic obstructive pulmonary disease, and the feasibility of improving knowledge and use of spirometry in primary care.¹ They conducted a mailed survey among primary-care physicians, to identify barriers to spirometry use in primary-care offices. The survey had a 51% response rate. The physicians' main reasons to refrain from office spirometry were the uncertain impact of the test, unfamiliarity with the testing and interpretation of the results, financial barriers, and the time required to fit spirometry into daily practice. Kaminsky et al suggest making efforts to reduce "bottlenecks" and to come to a better implementation of office spirometry.

Other similar surveys have been published that also reveal barriers to spirometry in primary care.²⁻⁵ The barriers previously described include lack of adequate training in spirometry, low confidence in ability to interpret spirometry results, lack of owning a spirometer, and physician reluctance regarding the value of spirometry in diagnosing chronic obstructive pulmonary disease. Kaminsky et al did not find any additional barriers, probably because their study was designed as a mailed questionnaire survey, and they did not have a true qualitative-research design. We believe that the use of such a qualitative-research approach would reveal additional and important barriers to spirometry in daily practice. Qualitative research methods, such as in-depth interviews and focus-group studies, are a flexible and powerful tool to reveal various aspects of physicians' views and feelings regarding barriers to spirometry in primary care. Although 2 qualitative studies have been published on this topic,^{5,6} the results of those studies should be interpreted with caution because of their poor methods.

It is time to design a mixed-methods study to explore barriers to spirometry use.

In this form of research, quantitative and qualitative data will be integrated, related, or mixed at some stage of the research process.⁷ Quantitative methods alone are not sufficient to capture the details in the complex phenomenon of barriers that primary-care physicians experience when starting or refraining from office spirometry in primary care.

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Cystic Fibrosis and Celiac Disease in a Family: Adding a Fourth Reminder

In the May 2005 issue of *RESPIRATORY CARE*, I read with great interest the article "Diagnosis of Cystic Fibrosis and Celiac Disease in an Adult: One Patient, Two Diseases, Three Reminders," by Rabinowitz.¹ The author mentioned the rarity of the 2 diseases in combination. Two other case reports of the rare coexistence of these 2 conditions have been published.^{2,3} In this letter I present the short history of one additional case.

A 20-year-old man (born 1969) was referred to our adult pulmonology unit in 1989, because of nodular opacities in the upper lobes on his chest radiograph, chronic cough, and purulent sputum. The following features in his medical history were of special interest. Since birth he had frequent bulky, malodorous stools. His somatic development was normal. At the age of 17, jejunal biopsy was performed, because celiac disease was diagnosed in his 3-year-old brother, who had similar symptoms. The jejunal biopsy was negative. Because of recurrent maxillary sinusitis, frequent punctions were necessary. His upper respiratory tract was colonized with *Staphylococcus aureus* and *Haemophilus influenzae*. Considering the abnormalities on the chest radiograph, he was treated with anti-tuberculars for 3 months, in another hospital, but without any change in his radiograph. His sputum was negative for acid-fast bacilli.

On the basis of his history and the clinical symptoms (eg, chronic sinopulmonary disease and pancreatic insufficiency), we considered the possibility of cystic fibrosis. The results of repeated sweat tests (chloride concentrations of 96 mmol/L and 113 mmol/L) and mutation analysis ($\Delta F508$ homozygote) supported the diagnosis of cystic fibrosis. His treatment began accordingly, but his compliance was poor. At the age of 24 he was diagnosed with obstructive azoospermia as well. After 6 years of follow-up he died because of progressive respiratory failure.

In light of the diagnosis of cystic fibrosis, we explored the health condition of his

sister and brother. The symptomless sister (born in 1972) proved to be a $\Delta F508$ carrier. The 14-years-younger brother (born 1983) had similarly frequent, bulky stools, steatorrhea, and malnutrition since infancy. Jejunal biopsy performed at the age of 3 years confirmed the diagnosis of celiac disease. A gluten-free diet was instituted. After 1 year of that diet, a repeat biopsy showed marked improvement. Based on our recommendation, he was also examined for cystic fibrosis, at 6 years of age. His main symptoms were pansinusitis, pancreatic insufficiency, and digital clubbing. High chloride concentration in the sweat (measurements of 106 mmol/L, 97 mmol/L, and 112 mmol/L) and $\Delta F508$ homozygote status were found, so cystic fibrosis was the diagnosis. The gluten-free diet was stopped; the possibility of the patient having 2 rare diseases was neglected.

At the age of 12 years, despite the pancreatic enzyme substitution, his weight gain was not satisfactory, and he had iron-deficiency anemia (hemoglobin 121 g/L, mean corpuscular volume 86.9 fL, serum iron 7.8 $\mu\text{mol/L}$, ferritin 10.61 ng/mL). The possibility of simultaneous celiac disease arose. Tests were made for anti-endomysial and

anti-jejunal antibodies. Both tests were positive, at a titer of 1:640. Jejunal biopsy again confirmed the diagnosis of celiac disease. The combination of the 2 very rare diseases was confirmed. Gluten-free diet was re-instituted. The markers normalized and the control biopsy showed remission after 6 months of the strict diet. He requires the gluten-free diet lifelong.

The severity of his pulmonary status worsened at the age of 14 years. He has been colonized with *Pseudomonas aeruginosa* since that time. Several functional endoscopic sinus surgeries were performed. Gastroesophageal reflux, hiatal hernia, and hepatosplenomegaly complicate his condition. He started using nocturnal oxygen therapy.

He was referred to our unit for adult care at the age of 22 years, in 2005. He has been referred for lung-transplantation, because of end-stage respiratory insufficiency (forced expiratory volume in the first second 15% of predicted, P_{aO_2} 49.5 mm Hg, P_{aCO_2} 48.4 mm Hg, blood oxygen saturation 83.1%). He keeps the gluten-free diet.

Concluding, I fully agree with Rabinowitz's 3 reminders, and this family case history underlines the importance of those. In

addition, I want to call attention to a fourth reminder. In case of a genetic disorder it is highly recommended to examine the patient's siblings. In the family reported above, the diagnosis of cystic fibrosis in the older brother could have supported earlier diagnosis of a second disorder in the younger sibling.

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