high-volume, low-cost tests. Currently, the ADA procedure falls in the low-volume category, and no one knows what influence managed-care gatekeepers will have on future sample flow for this useful diagnostic procedure(s). To achieve wide acceptance, I stressed the low cost of the basic ADA analytic procedure. Dr. Gakis’ means for the differential assay of ADA-1 and ADA-2 are not much more expensive and can be instituted easily in a routine laboratory so motivated.

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REFERENCES


Pulmonary Hypertension and Obstructive Sleep Apnea

To the Editor:

I am writing with regard to the recent “Pulmonary and Critical Care Pearls” by Nancy A. Collop (April 1996). In particular, I take exception with her first clinical pearl, which states, “Pulmonary hypertension can occur in obstructive sleep apnea in the absence of clinically significant lung disease.” First, this statement implies that the patient presented had no clinically significant lung disease. Given the abnormal pulmonary function tests (which show restrictive disease) and the abnormal arterial blood gas measurement (which shows a chronic compensated respiratory acidosis in addition to the hypoxia), this patient likely has obesity-hypventilation syndrome, which I would consider a clinically significant pulmonary disease. I would suggest that pulmonary hypertension (PH) developed first, secondary to the daytime hypoxemia related to the obesity-hypventilation syndrome and the nocturnal hypoxemia related to the obstructive sleep apnea (OSA). Later, the hypoxemia worsened when the mean pulmonary artery pressure became sufficient to open the patent foramen ovale, resulting in shunt physiology. I do not believe that this case is an example of PH secondary to isolated OSA, as implied in this clinical pearl.

Second, the most recent study on the relationship between PH and OSA by Chaouat and colleagues excluded the possibility that OSA can be a primary cause of pulmonary artery hypertension (PH). The authors state that the presence of PH was linked to an obstructive ventilatory defect. Our patient did not have obstructive lung disease. She was indeed obese, however. The contribution of obesity to the PH is mentioned in the article, but clear conclusions are not made. Again, I would point out that this patient’s PH was significantly improved only with treatment of her OSA, suggesting that other causes for her PH are unlikely.

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To the Editor:

I disagree with Dr. Rowley’s statement that this patient had significant pulmonary disease. I would argue that her lungs are relatively normal; her restrictive lung disease is more related to her obesity than to abnormal lungs. This is further supported by the fact that her diffusion corrected for alveolar volume is normal. Obesity-hypventilation syndrome is not a “lung” problem, it is a problem of an abnormal ventilatory drive. I would further argue that this patient’s obstructive sleep apnea (OSA) is the major cause of her hypoxemia and hypercapnia, because both improved following treatment.

With regard to the second point, I do not believe that the article by Chaouat et al excludes the possibility that OSA can be a primary cause of pulmonary artery hypertension (PH). The authors state that the presence of PH was linked to an obstructive ventilatory defect. Our patient did not have obstructive lung disease. She was indeed obese, however. The contribution of obesity to the PH is mentioned in the article, but clear conclusions are not made. Again, I would point out that this patient’s PH was significantly improved only with treatment of her OSA, suggesting that other causes for her PH are unlikely.

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