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1 Jones AM, O’Driscoll R. Do all patients require supplemental oxygen during flexible bronchoscopy? Chest 2001; 119:1906–1909
2 Golpe B, Mateos A. Influencia de la broncofibroscopia en la saturación de O₂ [abstract]. Arch Bronconeumol 2001; 37(S1):121

To the Editor:

We are pleased that Drs. Golpe and Mateos found our article of interest and that they had similar findings in their smaller study. Their study confirms that transient minor desaturation is common during bronchoscopy, but we differ in our response to this finding. We are unaware of any reports of adverse consequences from transient minor desaturation of the type described in both articles. Therefore, any recommendation to administer oxygen to all patients during flexible bronchoscopy must be regarded as a non-evidence-based suggestion.

Golpe and Mateos have also reported minor desaturation after the bronchoscopy procedure, and they suggest that oxygen treatment should be continued for an unspecified time after the procedure. This would increase the cost and complexity of running a bronchoscopy service (our present practice is to continue oxygen treatment after bronchoscopy only if oximetry indicates hypoxia following the procedure). The significance of transient mild desaturation (mean ± SD arterial oxygen saturation, 91 ± 3%) during the recovery phase is of very uncertain significance. The patients had no instrumentation during this phase. For example, it is known that patients with moderate COPD but normal resting oxygen saturation have significant hypoxia develop at an oxygen pressure equal to that of a commercial airliner, with a further fall to a mean saturation of 80% during mild exercise. These patients were all asymptomatic, and it has never been suggested that COPD patients with normal resting blood gas should be administered oxygen during flight.

We are confident about the safety of our present practice of administering oxygen only to patients who have sustained hypoxia develop (or to those with high-risk features, such as angina).

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REFERENCE

Endobronchial Spread of Parenchymal Metastases

To the Editor:

We read with interest the recent article in CHEST by Kiryu et al (March 2001). The authors classified endobronchial metastases (EM) on the basis of developmental mechanisms and attempted to correlate patient survival times with the different developmental modes. We applaud the authors’ efforts to define and morphologically classify EM; however, conclusions based on such a small number of patients are tenuous. The authors reported that the type II mode of EM is rare. We describe here the case of a patient with EM from a soft-tissue sarcoma that could be categorized as type II based on the proposed criteria. Soft-tissue sarcoma is a rare neoplasm. Pulmonary metastases are a common manifestation but rarely present as the initial finding. Although parenchymal metastases are a frequent occurrence, endobronchial spread is extremely rare.2,3

We present the case of a 36-year-old man with no significant medical problems but with a history of progressive dyspnea and right-sided diffuse chest pain for 2 months. A physical examination revealed dullness to percussion and decreased breath sounds over the right lung base. A chest radiograph showed multiple lung nodules bilaterally with a large right-sided pleural effusion. Pleural fluid was serosanguineous, exudative, and negative for malignant cells. A video-assisted thoracoscopic biopsy of the nodules showed features that were consistent with low-grade sarcoma. A bronchoscopic examination revealed a submucosal lesion obstructing the right bronchus intermedius. A thorough medical history and a physical examination subsequently revealed a 6-month-old swelling in the right thigh that was pathologically similar to the lung lesions. The pulmonary lesions were deemed unresectable, and chemotherapy (doxorubicin [Adriamycin; Pharmacia & Upjohn; Peapack, NJ] and ifosfamide) was initiated.

Unresectable lung disease, the number of nodules, shorter doubling time, and the histologic type (ie, spindle cell) were poor prognostic factors in our patient. Although arising from a low-grade primary sarcoma, the aggressive behavior seen is similar to metastases from high-grade lesions. Our case clearly illustrates the difficulty in the management of endobronchial and pulmonary metastatic disease and the various factors that affect prognosis.

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To the Editor:

We appreciate the interest in our article. Our article, classifying endotracheal/endobronchial metastases (EEM) into four types, was intended to elucidate the developmental modes of bronchoscopically visible EEM and to correlate patient survival times with the different types.1

As Muniyappa et al mentioned, EEM from sarcomas are rare, and there have been only a few reports.2,3 In our series, EEM from sarcomas were found in only 2 of 16 cases (12.5%); both were osteosarcoma of the bone. However, King and Castleman4 reported that, of 11 patients with sarcoma accompanied by intrathoracic metastatic lesions, 6 patients had endobronchial metastases on the basis of pathologic study, for an incidence of...
55.6%. As stated in our earlier discussion, it is important to recognize that the frequencies of EEM vary by definition.

Of the four types of EEM we proposed, we consider that type II is a rare condition because only 1 of 16 subjects demonstrated this type in our series. Although it is difficult to differentiate type II lesions from type IV lesions (type IV being the most common type) using clinical findings and chest imagings alone, it is possible to differentiate these two types by the depth of lesion, and by whether there is mucosal or submucosal invasion. Because of the difference in pathogenesis and the clinical significance, we think that types II and IV should be strictly separated.

Surprisingly, the incident of type II EEM reported by Munipayapp et al showed markedly aggressive behavior despite low-grade primary sarcoma. We encountered one patient with type II EEM who had maxillar carcinoma with histologically adenoid cystic carcinoma. The recurrence interval and survival time were 196 months and 40 months, respectively. This discrepancy is rather interesting.

We thank the authors for bringing to our attention the existence of an aggressive case of type II and for providing the additional reference by Greelish et al, and we certainly agree that, because of the small number of patients evaluated in our series, further evaluation with larger numbers of patients is essential.

To the Editor:

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REFERENCES

To the Editor:

We appreciate the comments about our case report. Regarding the possibility of a neuromuscular disease as the cause of our patient’s pectus deformity, there is no clinical evidence for any such disorder in our patient. Her neuromuscular system is entirely normal. Muscle power and deep tendon reflexes are within normal limits. She is able to generate a negative inspiratory force of 60 cm, and the sniff test has excluded the possibility of diaphragmatic weakness, as she could move both her diaphragms for at least one intercostal space. Advanced neuromuscular disease during childhood usually leads to considerable deformity of the spine and the trunk. Our patient is devoid of any other deformities except for severe pectus excavatum.

Pectus and Hypoventilation

To the Editor:

We read with interest a recent article in CHEST [June 2001] that considered pectus excavatum as the cause of hypoxic respiratory failure. Patients with generalized neuromuscular weakness that results in scoliosis, such as those with Duchenne muscular dystrophy and those with pectus due to the generalized weakness associated with spinal muscular atrophy type 1 (SMA1), develop hypoxic respiratory failure. Also, even when not associated with generalized weakness, thoracic deformity from kyphoscoliosis, when severe enough, itself can result in hypoventilation. However, in the cases of patients with Duchenne muscular dystrophy and SMA1, the ultimate cause of the skeletal impairment is muscular. Likewise, idiopathic scoliosis may be due to paraspinal muscle weakness, just as the pectus in these patients may be due to, or at least may be associated with, intercostal muscular impairment. Although diaphragmatic function was tested in these patients with pectus, the values of the sniff test were not given. An assessment of maximum inspiratory pressures would reflect the strength of inspiratory muscles, including those of the rib cage. If the root cause of the problem is an underlying muscular impairment, then the hypercapnia may be due more to that condition, and the pectus deformity may be only secondary, just as it is for patients with SMA1. The fact that the pectus deformity of the spine in this patient had a history of being easily fatigued and frail as a child implies a possible neurologic or muscular disorder that worsened with time.

It is also unclear when the pectus began. Just as for SMA1 patients whose pectora resolve once they are placed on nocturnal high-span bilevel pressure ventilation, this patient’s pectus may have been milder had high-span bilevel ventilation been instituted much earlier. It is of concern that, despite the rigidity of the chest wall in this patient and the severity of the pectus, only low bilevel spans were used. We suspect that, had this patient been introduced to noninvasive ventilation at adequate levels long ago, hypoxic respiratory failure would not have developed. We are also concerned about oxygen supplementation, and we suggest that the patient should use increasing periods of daytime ventilatory support via a mouthpiece to normalize alveolar ventilation rather than oxygen therapy, which entails a risk of developing increasingly severe hypercapnia.

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