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Pneumocephalus in Association with Lumbar Punctures

To the Editor:

The article by Jarjour and Wilson (Chest 1989; 96:1425-26) on pneumocephalus associated with nasal continuous positive pressure ventilation in a patient with sleep apnea syndrome was of particular interest to us. Recently, we noted the development of pneumocephalus in a patient admitted to the pulmonary/ICU service who had undergone multiple lumbar puncture attempts. A literature search revealed no report of pneumocephalus in association with lumbar punctures.

A 48-year-old actor was admitted to the hospital with penile trauma, lethargy and altered mentation. His medical history was significant for Gilles de la Tourette syndrome and a spinal injury sustained during childhood. His medications included haloperidol and pimozide. On presentation, he was alert with occasional lapses into incomprehensible, pressured speech. Physical examination was significant for gross blood from urethra and abrasion of the right scrotum. There was mild nuchal rigidity but other meningeal signs were absent. There was no external evidence of head trauma. Complete blood counts, blood biochemical studies and thyroid function test results were normal. Urologic evaluation revealed a bulbar urethral hematoma, and a suprapubic catheter was placed. An emergency CT scan of the head that included windows of the base of the skull was normal. Multiple (approximately 30) lumbar punctures were attempted by several physicians, but no fluid was obtained. All attempts were made with the patient positioned horizontally in the lateral decubitus position. No accidental or deliberate introduction of air into the subarachnoid space was reported by any of the physicians attempting spinal tap. The patient was empirically started on broad spectrum antibiotic therapy for possible bacterial meningitis, with a plan to obtain cerebrospinal fluid under fluoroscopy the subsequent morning. His mental status deteriorated rapidly during the night and he was transferred to the ICU. CT scan of the head was repeated using contrast medium and revealed pneumocephalus in the region of the right frontal lobe of the brain (Figure). A lateral view of the skull also showed pneumocephalus.

The patient underwent successful lumbar puncture under fluoroscopy the next morning. The cerebro-spinal fluid revealed no evidence of meningeal infection. A toxicology screen obtained on admission was positive for cocaine in the urine, and blood cultures were positive for Staphylococcus aureus. The patient’s mental status improved steadily. A third CT scan of the head obtained three days after the second showed no pneumocephalus. The patient was discharged from the hospital a few days later.

Although pneumocephalus has been reported in association with head trauma, mask CPAP, nasal CPAP, and rapid ascent to the surface by scuba divers, it has not been reported to occur with lumbar punctures. In fact, repeated lumbar taps have been used to treat increased intracranial pressure due to pneumocephalus. Our case shows that, in a case where lumbar puncture is difficult, it is probably more prudent to do the procedure under fluoroscopy rather than risk a pneumocephalus and its manifestations.

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Gemfibrozil Interaction with Warfarin Sodium (Coumadin)

To the Editor:

Gemfibrozil (Lopid) had been marketed for the treatment of severe triglyceridemia. In man, gemfibrozil therapy has been shown to inhibit peripheral lipolysis and to decrease the hepatic extraction of free fatty acids, thus reducing hepatic triglyceride production.

A 38-year-old woman with pulmonary embolism had been taking warfarin, 5 mg daily, for five months and had been well controlled prior to initiation of gemfibrozil therapy. Gemfibrozil (1,200 mg daily in divided doses) was prescribed for...
hypertriglyceridemia. She was seen in the office about two weeks later; her prothrombin time was much higher, and her menstrual cycle had been prolonged with lots of blood clots. She was advised to cut down on the dose of warfarin to 2.5 mg daily. Complete resolution of her menstrual cycle took place over the next two weeks. It appears that this patient had severe bleeding related to gemfibrozil-warfarin drug-interaction.

Gemfibrozil may inhibit the parahydroxylation of warfarin in the liver, thereby decreasing the serum levels of warfarin to a point where signs of warfarin intoxication (bleeding) occur. Because of chemical, pharmacologic and clinical similarities between gemfibrozil and clofibrate,1 this drug interaction is not surprising.

Drug interaction, therefore, exerts a special hazard in such cases.4 Because of the wide use of gemfibrozil, heightened clinical awareness of the dangerous potential for interaction with warfarin is rather important.

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Pulmonary Hypertension and Sickle Hemoglobinopathy

To the Editor:

We read with interest the article by Rubin on "Approach to the Diagnosis and Treatment of Pulmonary Hypertension" (Chest 1989; 96:659-64). The author based the classification of pulmonary hypertension on the primary site of involvement, namely precapillary and postcapillary causes of pulmonary hypertension.

The diagnostic approach to thromboembolic forms of precapillary pulmonary hypertension deserves further comment in view of our recent case of hereditary spherocytosis with pulmonary hypertension, and known literature data on sickle cell hemoglobinopathies and thalassemia.

The vast majority of pulmonary emboli are due to dislodgement of venous thrombi with subsequent impaction in the pulmonary circulation. However, it is necessary to distinguish in situ thrombosis in cases of sickle cell hemoglobinopathies SS, SC and SB thalassemia.1 In such cases, autopsy specimens have revealed asymmetric intimal expansion and widespread occlusions of small- and medium-sized arteries with old, organized, recanalized thrombi.2 In these hemoglobinopathies, red cell sequestration may occur in the lungs, resulting in increased viscosity, vascular stasis and even complete blockage of the microvasculature.

This in situ vaso-occlusion is precipitated by gross sickling of red blood cells, especially in hypoxic areas, leading to microvascular thrombosis and microinfarcts with necrosis. Bone marrow and fat emboli released from areas of ischemic bone necrosis can contribute to the vaso-occlusion.3 Extensive vascular narrowing and occlusions lead to increased pulmonary resistance, pulmonary hypertension and cor pulmonale.

The venous-thrombus pulmonary embolism pattern is of little importance in such cases since the incidence of pulmonary embolism is not increased in patients with sickle cell hemoglobinopathies in comparison to the general population.

Therefore, we would suggest adding peripheral blood film examination, sickle cell test and hemoglobin electrophoresis to the diagnostic evaluation of anemic patients with complaints suggestive of pulmonary hypertension; this hypertension may already be present before the diagnosis of hemoglobinopathy is made. 2

Even though the true incidence of pulmonary hypertension and sickle hemoglobinopathy is largely unknown, it is likely that the effects of repeated pulmonary vascular occlusions may become even more apparent in view of the improved life expectancy of patients with sickle cell hemoglobinopathy.

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To the Editor:

The comments made by Doctor Verresen and his colleagues are valid and ones with which I agree. Patients with sickle cell disease can develop pulmonary hypertension due to in situ thrombosis with microvascular sickling; furthermore, this process may be complicated by pulmonary venous hypertension, which can result from the cardiomyopathy associated with sickle cell anemia. Patients with unexplained pulmonary hypertension and anemia should undergo a thorough evaluation to exclude hereditary or acquired conditions (such as hemoglobinopathies, granulomatous diseases, vasculitis, and malignancies with tumor embolization to the lungs) which may be associated with these two processes.

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Pleural and Lung Cryobiopsies

To the Editor:

We have recently published a paper entitled, "Pleural and Lung Cryobiopsies During Thoracoscopy" (Chest 1989; 95:3). The number of patients in the series has now reached 35 and our conclusions are the same (i.e., analgesic effect, low risk of hemorrhage, and low risk of air escape). Using cryotherapy in pleural and lung lesions, perfectly adequate samples can be obtained for histologic examination using a single cycle of freezing and immediate fixing. However, in the latest series of patients we have used electron microscopy of lung and pleura biopsies and found degenerative changes in type 2 pneumocytes with very condensed cytoplasm and cellular detachment from the basal membrane. The collagenous and elastic framework of the alveolar wall is distorted by marked