
To the Editor:

We are indeed encouraged to learn of the successful use of trimethoprim and sulfamethoxazole in combination with other antimicrobial agents for treating disseminated melioidosis, as reported by Tanphaichitr and coauthors.

In our single experience with life-threatening disease limited to the lungs (Chest 74:222-224, 1978), antagonism occurred when trimethoprim and sulfamethoxazole were used in various combinations with tetracycline, chloramphenicol, and kanamycin. This antagonism, which was suspected because of the failure of the patient’s condition to improve clinically, was confirmed by studies of the serum showing an absence of bactericidal activity; however, bactericidal activity was excellent when the combination of trimethoprim and sulfamethoxazole was used alone in high dosage.

Although no significant antagonism occurred in the cases of Tanphaichitr et al, it would have been interesting to know the serum bactericidal levels of the combinations of drugs vs that of the trimethoprim-sulfamethoxazole combination used alone at high dosage. If the combination of trimethoprim and sulfamethoxazole alone could be used in disseminated melioidosis, it would avoid the well-known potential side effects of therapy with the other antibiotics.

We continue to recommend determining serum bactericidal levels in serious cases of melioidosis. These levels are rapidly obtained from the bacteriologic services of most hospitals, whereas determinations of minimum inhibitory concentrations often must be sent out to other institutions. Moreover, bactericidal levels have the advantage of being an in vivo test. Since it is conceivable that antagonism may occur whenever combinations of drugs are used, such as was seen in our case, bactericidal levels are a simple method of detecting such an occurrence and altering the therapy.

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Pleural Effusion and Renal Cell Carcinoma
An Angiographic-Pathologic Correlation

To the Editor:

Renal cell carcinoma is notorious for its myriad of clinical presentations. Pleural effusion is rarely the initial clinical problem with this tumor.

CASE REPORT

A 53-year-old man was admitted to San Francisco General Hospital in November 1977 with a cough and a four-month history of loss of weight. The patient smoked two packs of cigarettes per day; otherwise, the findings from the review of systems were unremarkable.

Examination showed a thin normotensive man in no respiratory distress; his temperature was 38.5°C (101.3°F). Per-

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DISCUSSION

Adenocarcinoma of the kidney has been called the “internist’s tumor” because of its variable presentation and course. Our case report describes another unusual presentation of this neoplasm, and the diagnostic evaluation illustrates an unexpected angiographic finding.

It is well known that hypernephroma may spread by direct invasion, lymphogenous extension, lymphohematogenous spread, or direct hematogenous dissemination. In the present case, the pleural metastases must have developed from posterior intercostal arteries, the bronchial circulation, or Batson’s vertebral plexus. The unusual incidental angiographic finding of these pleural lesions was helpful in guiding our diagnostic evaluation and staging of this patient’s disease.

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REFERENCES


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Intravenous Therapy with Terbutaline

To the Editor:

Intravenous administration of β-adrenergic agonists in the treatment of life-threatening status asthmaticus has been reported previously.1,2 A major untoward reaction to therapy with these drugs is the occurrence of cardiac arrhythmias, particularly in the presence of hypoxemia;3 however, newer β-adrenergic agonists such as terbutaline have less cardiac stimulating effects for a given bronchodilating effect. Terbutaline sulfate was administered intravenously to a patient who appeared to be moribund from status asthmaticus, with a remarkably salutary effect.

Case Report

A 20-year-old woman with chronic bronchial asthma was admitted to the emergency room of a hospital with a one-day history of progressively severe wheezing that was unresponsive to oral therapy with bronchodilator drugs and was associated with the onset of an inhaler supplying epinephrine. In the emergency room, she was given epinephrine subcutaneously, a combination of isothantracene hydrochloride and phenylephrine hydrochloride (Bronkool) by intermittent positive-pressure breathing, and rectal and intravenous therapy with aminophylline, with little effect. During this time the patient became increasingly anxious and was given 100 mg of phenobarbital by mouth.

Twenty minutes later, the patient suffered respiratory arrest with severe hypotension. An endotracheal tube was immediately inserted, but the patient could not be ventilated because of severe bronchospasm. She was then given 100 mg of succinylcholine chloride and 89.2 mEq of sodium bicarbonate intravenously and was ventilated with a manual resuscitator (Ambu bag) and oxygen. Intravenous therapy with hydrocortisone was also started at this time. Blood gas levels just prior to intubation included an oxygen pressure (P02) of 30 mm Hg, a carbon dioxide tension (Pco2) of 135 mm Hg, and a pH of 6.88, with a base excess of -16.

Although there was an immediate decrease in wheezing following the administration of succinylcholine, during the next 15 minutes the bronchospasm became worse. One milligram of terbutaline sulfate was placed in 100 ml of a 5 percent solution of dextrose in water within an intravenous injection set (Volutrol) and was administered intravenously within 20 minutes at a rate which allowed the monitored heart rate to vary between 110 and 130 beats per minute. Frequent inspection of the monitor and electrocardiographic rhythm strips failed to reveal abnormalities.

As the administration of terbutaline was finishing, the patient began to breathe spontaneously with loud wheezing and within one hour was fully awake and free of audible wheezing. She was extubated at that time, upon demonstration of a tidal volume of 450 ml. Blood gas levels after extubation, while the patient was breathing room air, showed a P02 of 86 mm Hg, a Pco2 of 47 mm Hg, a pH of 7.39, and a base excess of +2. Because of emesis during intubation, with possible aspiration, the patient received oral therapy with cephradine. On the next day, oral therapy with theophylline and terbutaline was initiated. The wheezing subsided completely in 48 hours. Aspiration pneumonia did not develop, and no neurologic, renal, cardiac, or hepatic sequelae occurred.

The patient was discharged from the hospital four days after admission. She has been observed for 12 months and is fully functional as a bank teller, and her asthma has been well controlled by oral therapy with theophylline and terbutaline sulfate.

Discussion

This case illustrates the following two pitfalls in the treatment of asthma: (1) overuse by the patient of a handheld bronchodilator-containing inhaler; and (2) administration of barbiturates to an anxious subject with asthma. Although the patient received a large dose of succinylcholine, which was apparently life-saving, the degree of relief was not sufficient to allow continued adequate ventilation. Relief of the bronchospasm after intravenous therapy with terbutaline was so rapid and striking that its effect was not likely to be misinterpreted.

The relative decrease in the incidence and severity of cardiac side effects by the use of therapy with these newer agents has been documented.4,5 Inasmuch as hypoxic patients are more prone to the cardiac effects of β-adrenergic agonists, terbutaline might be considered safer in this respect than the less specific agents, such as isoproterenol, in this situation. Intravenous therapy with β-adrenergic agonists is not recommended for the routine treatment of status asthmaticus but may be employed in difficult situations where oxygenation, passive ventilation, parenteral therapy for alkalization, and adequate monitoring are available.

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References


Expiratory Flow Patterns in Amyotrophic Lateral Sclerosis

To the Editor:

Amyotrophic lateral sclerosis, a chronic progressive disease of the corticobulbospinal and lower motor neurons, results in a mixed spastic and atrophic muscular weakness. Bulbar or pseudobulbar palsies that affect control of the soft palate and tongue may alter the pattern of flow independently or in conjunction with the respiratory muscular weakness associated with the spinal involvement.