CASE REPORT

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Delayed Fatal Outcome After Possible Ru-Tuss® Overdose

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ABSTRACT: Death resulted from the delayed onset of the acute respiratory distress syndrome and disseminated intravascular coagulation with left ventricular mural thrombus formation and nonbacterial thrombotic endocarditis approximately five days after an alleged attempted suicide by the ingestion of ten to twelve prolonged-action Ru-Tuss® tablets. Although these lesions are thought to be similar in pathogenesis, this combination has not been previously reported in association with a drug overdose. The delay in onset is also of interest because of its clinical implications.

KEY WORDS: pathology and biology, toxicology, death

Ru-Tuss® is a vasoconstrictor-antihistaminic combination with belladonna alkaloids used for the treatment of colds, hay fever, and nasal allergies. In a prolonged-action tablet, it contains phenylephrine hydrochloride, 25 mg; phenylpropanolamine hydrochloride, 50 mg; chlorpheniramine maleate, 8 mg; hyoscyamine, 0.1936 mg; atropine sulfate, 0.0362 mg; and scopolamine hydrobromide, 0.0121 mg. Toxic effects may consist of drowsiness, xerostomia, anorexia, nausea, vomiting, dizziness, hypertension, tachycardia, blurring of vision, and urinary retention. Large overdoses are described as causing tachypnea, delirium, fever, stupor, coma, and respiratory failure [*1*].

Case Report

The decedent was a 19-year-old white female who was hospitalized after allegedly intentionally ingesting, in a fit of depression, ten to twelve Ru-Tuss prolonged-action tablets. She was seen in the emergency room approximately 1 h later and complained of drowsiness. She was given ipecac and vomited a large quantity of yellow material in which no pills were seen. She was admitted and received intravenous 0.5 normal saline and 5% glucose and a saline cathartic. Vital signs on admission were recorded as within normal limits and stable, and an electrocardiogram gave normal results. Antihistamine levels in blood were not measured.

The patient remained drowsy and slept off and on during the afternoon and night of

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the first hospital day, and on the second day, she was more alert and afebrile. The white blood cell count was 14 700 and a chest X-ray showed a right lower lobe infiltrate. Sputum was cultured and Keflex® administered, 2 g every 6 h intravenously. She was recovering with supportive care and was alert and ambulatory until the third hospital day, when she began to complain of shortness of breath and pleuritic-type chest pain; she developed wet rales bilaterally and labored respirations, which progressed rapidly in the next 4 h to a cardiopulmonary arrest. During the interval before arrest, she received meperidine hydrochloride, 50 mg intramuscularly; Organidin®; and methylprednisolone sodium succinate intravenously. She was intubated and after emergency measures for resuscitation, which included a respirator, aminophylline, steroids, and furosemide intravenously, she was transferred, still on a respirator, to University Hospital; upon arrival, she was unresponsive and had bloody secretions around the mouth and nose. Physical examination revealed sinus bradycardia and bilateral rales, and a diagnosis of respiratory distress syndrome of adult, or "shock lung," was made. Tests of blood gases and pH showed metabolic acidosis. Soon after admission she became partly responsive to verbal stimuli. Twenty-two hours after admission she pulled out her orotracheal tube but was immediately reintubated. She received antibiotics, propanolol hydrochloride, calcium chloride, epinephrine, sodium bicarbonate, digoxin, metaraminol bitartrate, lidocaine hydrochloride, dopamine, atropine, and Plasmanate®. A few minutes before death she had an episode of ventricular tachycardia and asystole. She was given cardiac shock but was unresponsive to all measures and died.

At autopsy there was a thrombus attached to the inferior anterior and the posterior apical left ventricular endocardium (Fig. 1). There was a 2-mm "papillation" attached to the posterior mitral leaflet. The right lung weighed 950 g and the left lung, 780 g. On the cut surface they were firm, wet, and frothy, and there were multiple thromboemboli standing up in peripheral vessels. Blood drawn at autopsy for a toxicology screen contained small amounts of meperidine and lidocaine. Exact levels were not reported by the laboratory.

Histological examination showed fibrin thrombi in small vessels of the heart (Fig. 2), adrenal medulla, renal glomeruli and arterioles (Fig. 3), and segmental renal veins. The

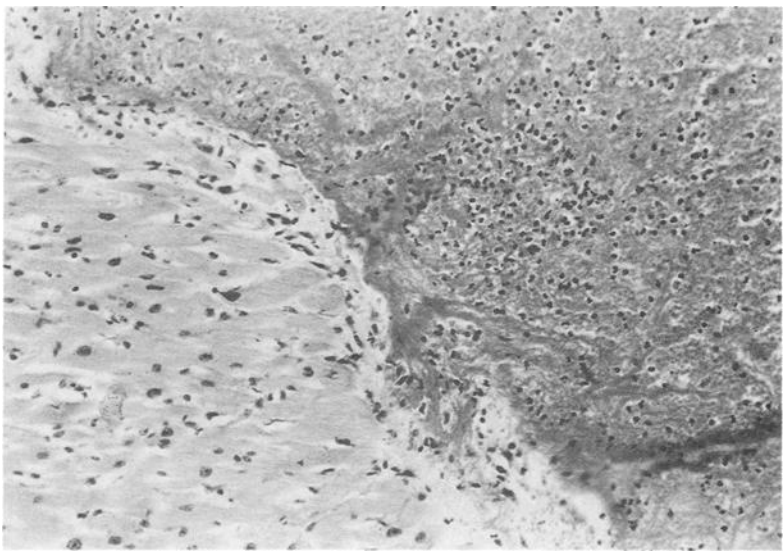

FIG. 1—*Mural thrombus attached to left ventricular endocardium.*

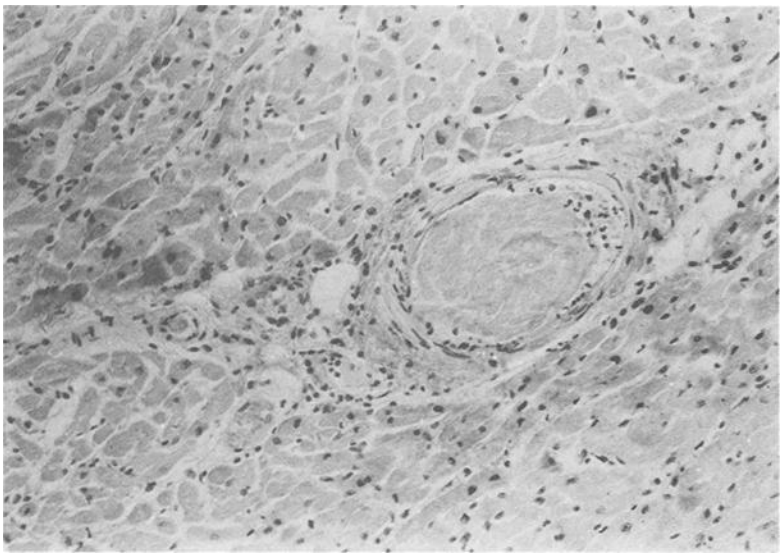

FIG. 2—*Thrombus in a small interstitial left ventricular artery demonstrating disseminated intravascular coagulation.*

lungs specifically showed extensive hyaline membrane formation, organizing pneumonia, and scattered pulmonary vascular thromboemboli (Fig. 4). On the posterior leaflet of the mitral valve was attached a tiny, partially organized fibrin thrombus.

Discussion

This patient was a victim of the acute respiratory distress syndrome of the adult, which has been reported as a sequela of traumatic and nontraumatic shock including drug

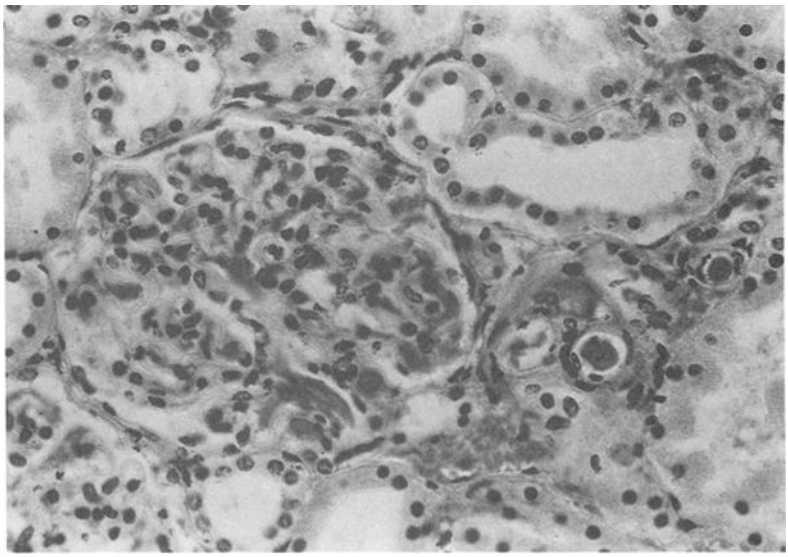

FIG. 3—*Intravascular coagulation in glomerular capillaries and arterioles.*

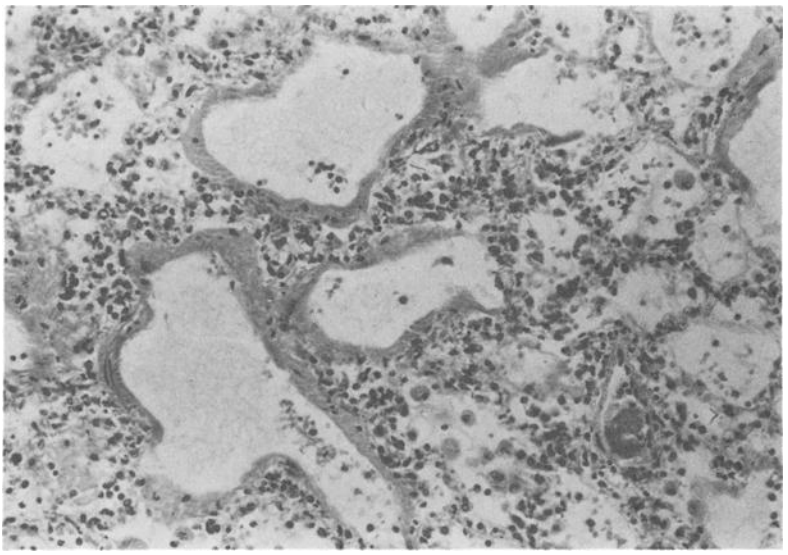

FIG. 4—*Pulmonary hyaline membranes and intravascular coagulation, a manifestation of acute respiratory distress syndrome of adult, or "shock lung."*

overdoses of various types [*2*]. Characteristically, it may occur when the patient seems to be improving clinically from the original insult, as it did in this patient. The exact mechanism of this syndrome is not known and its pathogenesis is still obscure. There is extensive literature concerning this disease and its occasional association with disseminated intravascular coagulation and nonbacterial endocarditis [*3,4*]. A case involving mural thrombus of the left ventricle, however, was not encountered in my search of the literature, so I think that this is an unusual case. The extensive changes in the lung accompanying this syndrome are frequently complicated by the onset of bronchopneumonia, as they were in this patient. The treatment of the acute respiratory distress syndrome is entirely symptomatic [*5*] and the occurrence of other associated problems, such as disseminated intravascular coagulation, bronchopneumonia, thromboemboli in the lungs, nonbacterial thrombotic endocarditis, and mural thrombus, could certainly account for the severity of the syndrome in this patient and its apparent irreversibility.

References

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