Tracheal Injury Caused by Ingested Paraquat*

Manuel Ruiz-Baillén, MD; María del Carmen Serrano-Córcoles, MD; and José Ángel Ramos-Cuadra, MD

Paraquat is a potent herbicide of lethal toxicity. Its injury mechanism is attributed to the generation of very-reactive oxygen species, such as superoxide radicals, through which multiple injuries are produced on mucosa, although there have been no reports of injuries on the trachea. We describe a case of fatal paraquat poisoning with tracheal injuries, where the clinical debut was acute respiratory insufficiency and a spontaneous pneumothorax.

(CHEST 2001; 119:1956–1957)

Key words: paraquat; pneumothorax; poisoning; trachea

CASE REPORT

A 20-year-old man came to our emergency department with a 1-day history of odynophagia, pharyngeal pruritus, general malaise, and fever, but without presenting severe symptomatology. He denied any history of suicide attempts and received a diagnosis of pharyngotonsillitis. He received outpatient treatment with amoxicillin and paracetamol. After 16 h, he returned to the emergency department with acute respiratory insufficiency; on this occasion, he admitted the ingestion 3 days earlier of approximately 100 mL of a 20% paraquat compound, because of a transient suicidal impulse. The examination showed major bilateral subcutaneous emphysema (from mandible to lower extremities). Radiography revealed right pneumothorax of 35%, pneumomediastinum, and pneumopericardium. He presented with hyperthermia, hypotension, tachycardia, tachypnea, and a low level of consciousness. Basal analytical findings were as follows: leukocytosis; prothrombin activity, 47%; urea, 148 mg/dL; serum creatinine, 8.8 mg/dL; direct bilirubinemia, 8.8 mg/mL; aspartate aminotransferase, 175 mg/dL; plasma amylose, 5,349 U; hyponatremia and hyperkalemia; PaO2/fraction of inspired oxygen ratio, 95; and metabolic acidemia. The concentration of paraquat in blood was 1.25 μg/mL, determined at the time of hospital admission (3 days after the ingestion) by spectrophotometry. Findings of culture analyses were all negative. The chest CT scan revealed right pneumothorax, pneumomediastinum, and pneumopericardium, and ruptured continuity of the posterior tracheal wall (Fig 1). The bronchoscopy findings were as follows: multiple pearly ulcerations in the oral cavity, friable and hyperemic tracheal mucosa with abundant material of purulent appearance, necrotic ulcers in the trachea and entire bronchial tree, and a necrotic lesion that permitted visualization of a break in the continuity of the posterior tracheal wall. The patient developed ARDS and distributive shock that progressed to hyperacute multiorgan failure, with hepatic, renal, neurologic, respiratory, hemodynamic, and hematologic failures that caused the death of the patient 24 h after his admission.

DISCUSSION

Paraquat (1,1 dimethyl-4,4-bipyridilium chloride) has been used as a herbicide since 1962 and is the most widespread weed killer among the group of bipyridilium compounds with quaternary structures. Paraquat is heavily used in greenhouse cultivation in our region (Almería, Spain). It is a potent herbicide that rapidly destroys plants in the presence of light and oxygen. Poisoning in humans is normally produced by ingestion because of accident or suicide attempt (although percutaneous absorption has also been reported), which produces, as in the present case, a multisystem involvement, with injuries to the lungs, kidneys, liver, brain, heart, suprarenal gland, or muscles until multiorgan failure, ARDS, or lung fibrosis develops. A large ingestion of this toxin, as in our case, is

*From the Intensive Care Unit (Drs. Ruiz-Baillén and Ramos-Cuadra) and Emergency Unit (Dr. Serrano-Córcoles), Critical Care and Emergencies Department, Hospital de Poniente, El Ejido, Almería, Spain.

Manuscript received June 13, 2000; revision accepted November 15, 2000.

Correspondence to: Manuel Ruiz-Baillén, MD, C/ Las Torres 57, 23650 Torredonjimeno, Jaén, Spain; e-mail: MRB1604@teleline.es

Figure 1. CT image showing tracheal injury (arrow).
rapidly followed by the onset of a major inflammatory process in the oropharynx, esophagus, and stomach that translates into nausea, vomiting, abdominal pain, and diarrhea,\(^1\) and normally generates toxic hepatitis with centrolobular necrosis and myocardiitis, occasionally with a reduced level of consciousness. Very rarely, spontaneous extra-alveolar air appears (pneumothorax, pneumomediastinum, pneumopericardium, subcutaneous emphysema), which has been ascribed to a direct provocation by the toxin of acute lung injury, with the formation of subpleural bullae followed by rupture.\(^3\)

We present a case of massive paraquat poisoning, with the peculiarity that the corrosive effects of the paraquat itself also caused injuries to the trachea and principal bronchi. Although there have been reports that the local corrosive effect of paraquat produces ulcerated lesions in the oropharynx, esophagus, and stomach,\(^4\) the tracheal injuries seen in the present patient have not previously been described. These injuries appeared on the trachea and bronchial tree and ruptured the posterior tracheal wall. They were directly caused by the caustic action of the toxin, probably after its aspiration. The effects at cell level, especially on tracheal epithelial cells, which have shown experimentally a greater sensitivity to paraquat, could be produced by the generation of very-reactive oxygen species, such as superoxide radicals. These radicals have deleterious effects on the cells by attacking the proteins and membranous organelles, inhibiting macromolecular synthesis, and enhancing lipid peroxidation.\(^5\)

The tracheal injuries contributed to the development of extra-alveolar air and to the demise of the patient. Although there has been no previous report of a similar injury, the Toronto Lung Transplant Group\(^6\) described a patient with acute paraquat poisoning who required a lung transplant, with death caused by complications derived from a trachea-innominate artery fistula. The fistula was initially described as a complication of the trachetomy, although the deleterious effect of the paraquat could also have influenced the genesis of this tracheal injury. There is no efficacious treatment for paraquat poisoning in the clinical setting, but it has been experimentally demonstrated that a reduction in the intracellular nicotinamide adenine dinucleotide phosphate can protect the tracheal cells against paraquat poisoning,\(^5\) which suggests that future studies may show more satisfactory results.

REFERENCES


Severe Pectus Excavatum Associated With Cor Pulmonale and Chronic Respiratory Acidosis in a Young Woman*

Rachichandra Theerthakarai, MD; Walid El-Halees, MD; Seyed Javadpoor, MD; and M. Anees Khan, MD, FCCP

Pectus excavatum has never been reported to cause hypercapnic respiratory failure. In this report, we describe the first such case in a young woman with severe pectus excavatum who presented with chronic respiratory acidosis, pulmonary hypertension, and chronic cor pulmonale. An extensive diagnostic workup failed to uncover any other cause of respiratory acidosis, which led us to conclude that the severe chest wall deformity and the resulting severe restrictive defect were responsible for the development of chronic respiratory acidosis and cor pulmonale.


Key words: alveolar hypoventilation; cor pulmonale; pectus excavatum; respiratory failure

Abbreviations: TLC = total lung capacity; VC = vital capacity

Unlike deformities of the spine, pectus excavatum rarely results in a measurable impairment of lung function and is said to have never produced hypoventilation and respiratory failure.\(^1\) A MEDLINE search failed to identify a report of a patient who had experienced respiratory failure attributed to pectus excavatum. Congestive cardiac failure also is said to be almost unheard of\(^2\) and has not been observed in extensive hemodynamic studies.\(^3,4\) Some patients may show a decreased diastolic filling of the right ventricle as a result of compression, but pulmonary arterial and pulmonary wedge pressures have been normal.\(^3,4\) We describe the case of a young woman with severe

*From the Pulmonary Division, St. Joseph’s Hospital and Medical Center (Drs. Theerthakarai, El-Halees, and Javadpoor), Paterson, NJ; and Seton Hall University (Dr. Khan), School of Graduate Medical Education, South Orange, NJ. Manuscript received March 14, 2000; revision accepted November 9, 2000.

Correspondence to: M. Anees Khan, MD, FCCP, Chief, Pulmonary Division, St. Joseph’s Hospital and Medical Center, 703 Main St, Paterson, NJ 07503