Volume 8

Advances in Trauma and Critical Care

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Advances in Trauma and Critical Care

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Volume 8 • 1993

Mosby

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Mosby
Dedicated to Publishing Excellence

Vice President, Continuity Publishing: Kenneth H. Killion
Sponsoring Editor: Bernadette Buchholz
Project Manager: Denise Dungey
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Printed in the United States of America
Composition by The Clarinda Company
Printing/binding by The Maple-Vail Book Manufacturing Group

Mosby–Year Book, Inc.
11830 Westline Industrial Drive
St. Louis, Missouri 63146

Editorial Office:
Mosby–Year Book, Inc.
200 N. LaSalle Street
Chicago, Illinois 60601

International Standard Serial Number: 0886-7755
International Standard Book Number: 0-8151-6200-6

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Blunt Pulmonary Injury

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The spectrum of lung parenchymal injury following blunt chest trauma ranges from simple contusion to frank laceration. Pulmonary contusion is by far the most frequent variant and is characterized by hemorrhagic edema of the alveolar and interstitial spaces that typically worsens clinically and radiographically over a period of 24 to 48 hours and then slowly resolves unless complicated by infection, cavitation, or adult respiratory distress syndrome (ARDS).[1–6] With increased injury severity, more extensive tissue disruption results in contained intraparenchymal cavities. Since first described by Fallon in 1940, multiple terms have been applied to this unusual entity.[7–13] We feel that posttraumatic pulmonary pseudocyst (PPP) best describes these air- or fluid-filled intraparenchymal cavities that occur in the setting of blunt chest trauma.[14] A PPP usually resolves over several weeks to months unless complicated by infection. The term *pulmonary laceration* should be reserved for parenchymal tears that include visceral pleural disruption; these are typically manifested by persistent bleeding or a major air leak.[15–18] The rare extensive parenchymal tear requires early thoracotomy, and the mortality is high.[19] Blunt lung parenchymal injuries are frequently associated with other significant injuries, and coexisting hypovolemic shock complicates therapeutic priorities. Operations (laparotomy, craniotomy, pelvic fixation) coupled with radiologic evaluation (plain films, computed tomography, angiography) frequently delay recognition and appropriate intensive care unit (ICU) management of the pulmonary injury. Physiologic monitoring, prompt restoration of oxygen transport, shock resuscitation, selective mechanical ventilation, pain control, aggressive pulmonary care, judicious use of antibiotics, and early nutritional support are important for survival.

Epidemiology

Pulmonary contusion occurs in 10% to 20% of adults hospitalized following blunt chest injury and implies a high-energy transfer.[20–24] The vast majority of injuries are due to rapid acceleration/deceleration (motor vehicle accidents, falls); approximately 5% result from crushing mechanisms. Pulmonary contusion is primarily the result of direct impact, and it may be localized or diffuse. Two thirds of patients with a pulmonary contusion have associated intrathoracic injuries (Fig 1). Extrathoracic injuries are also common; head injuries occur in 40%, major fractures in 40%, and intra-abdominal trauma in 30%. Despite tremendous advances in trauma and

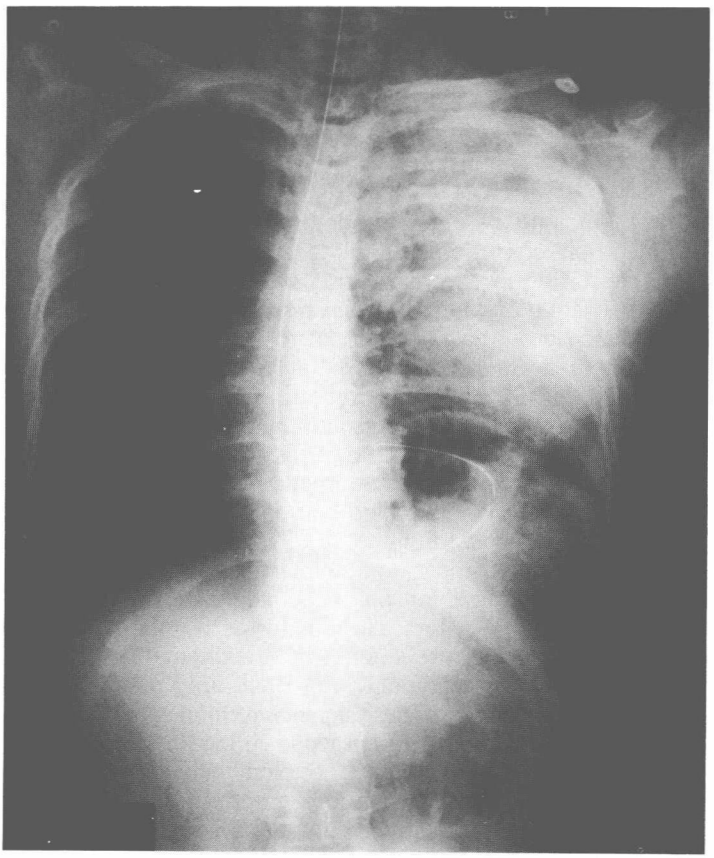

FIG 1.
Emergency department chest radiograph of a 29-year-old male involved in a high-speed head-on motor vehicle accident. Note the early pulmonary contusion, multiple left rib fracture, and ruptured left diaphragm.

critical care, the mortality rate remains 15%. Early deaths are due to hemorrhage and head injury; late mortality relates to sepsis and multiple organ failure. Factors that portend a poor outcome include shock (blood pressure less than 90 mm Hg), a high injury severity score (>25), head injury (Glascow Coma Scale score of 7 or less), coexisting flail chest, falls from great heights (greater than 20 ft), pre-existing disease (artherosclerotic heart disease, chronic obstructive pulmonary disease, Laënnec's cirrhosis), and advanced age (older than 65 years).

Pathophysiology

Pulmonary contusion is fundamentally a bruise of the lung. Direct injury causes pulmonary vascular damage with secondary alveolar hemorrhage.[1–6] In the early phase, these flooded alveoli are poorly perfused; consequently, little shunt exists. However, tissue inflammation develops rapidly, and the resultant surrounding pulmonary edema produces regional alterations in compliance and airway resistance leading to a localized ventilation/perfusion (V/Q) mismatch that progresses over a period of 24 to 48 hours. With increasing magnitude of injury, more extensive tissue disruption results in contained intraparenchymal cavities as well as frank lung lacerations.[7–19] A basic argument is whether shear stress or bursting forces produce these advanced lesions; it is likely that both factors are involved.[25–28] In a rabbit model, Lau and Viano[29] demonstrated that both impact velocity and chest wall displacement determine the severity and distribution of parenchymal injury. A high-velocity, low-displacement impact (lateral motor vehicle accident) causes alveolar lung injury, whereas a low-velocity, high-displacement impact (crush) produces central parenchymal and major bronchial disruptions.

Pulmonary parenchymal injury is a well-recognized risk factor for ARDS. Changes in the noninjured lung were originally attributed to contrecoup trauma, but the progressive congestion and atelectasis observed experimentally are more consistent with a capillary leak syndrome.[6, 30–32] Whether mediators are being released from the injured lung or elaborated from an extrathoracic site is unclear because the majority of these patients have other risk factors for ARDS (i.e., shock, hypertransfusion, long-bone fractures, and head injury). Chest wall pain along with secondary splinting of rib fractures is another source of respiratory failure and is compounded by a thoracotomy or midline laparotomy, which further compromises tidal volume, sigh volume, and forced expiratory volume and decreases functional residual capacity (FRC) below closing volume. Pain also impairs effective coughing and predisposes to atelectasis and retained secretions in an area of contused lung where bacterial clearance is already impaired.[33] Additionally, these severely injured patients have an acquired immune deficiency, with impairment of both nonspecific and specific immune mechanisms, and are likely to be colonized with virulent, antibiotic-resistant organisms.[34–38] Associated pelvic and long-bone fractures and head inju-

ries limit early mobilization and participation in respiratory care. Thus it is not surprising that pneumonia is a frequent complication.[39]

Diagnosis

Pulmonary contusion is predominantly a radiologic diagnosis.[40, 41] The classic finding is a nonsegmental pulmonary infiltrate corresponding to the area of external chest impact that is typically manifested within 12 to 24 hours of injury. The infiltrate may consist of irregular nodular densities that are discrete or confluent, a homogeneous consolidation, or a diffuse patchy pattern. These findings on an early postinjury chest radiograph indicate a severe injury (Figs 2 and 3). In the majority of cases, the infiltrates

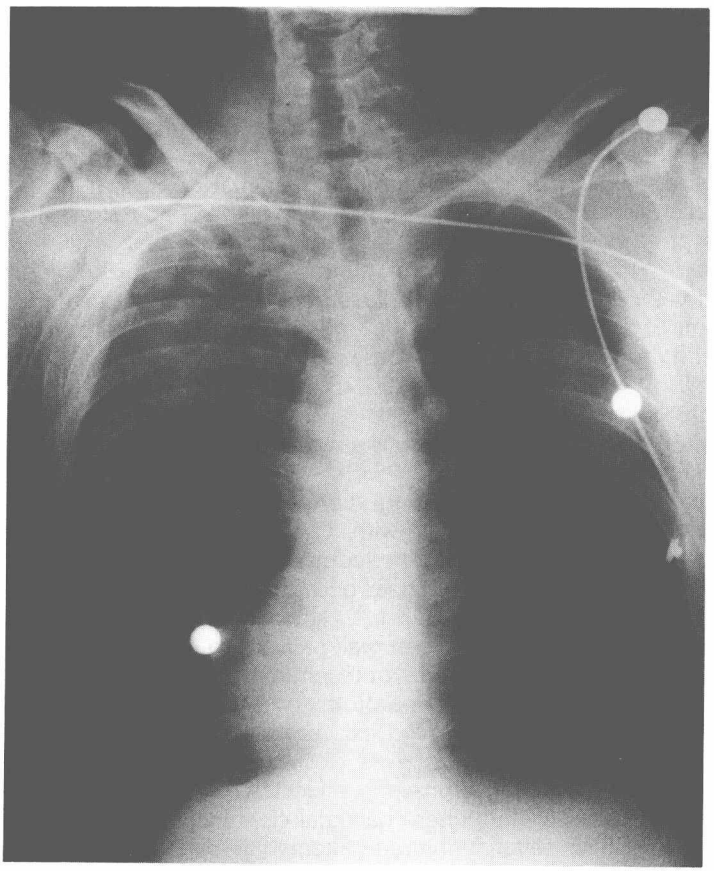

FIG 2.
Emergency Department chest radiograph of a 37-year-old male involved in a high-speed ejection rollover motor vehicle accident. Note the early bilateral pulmonary contusions, left pneumothorax, and multiple rib fractures.

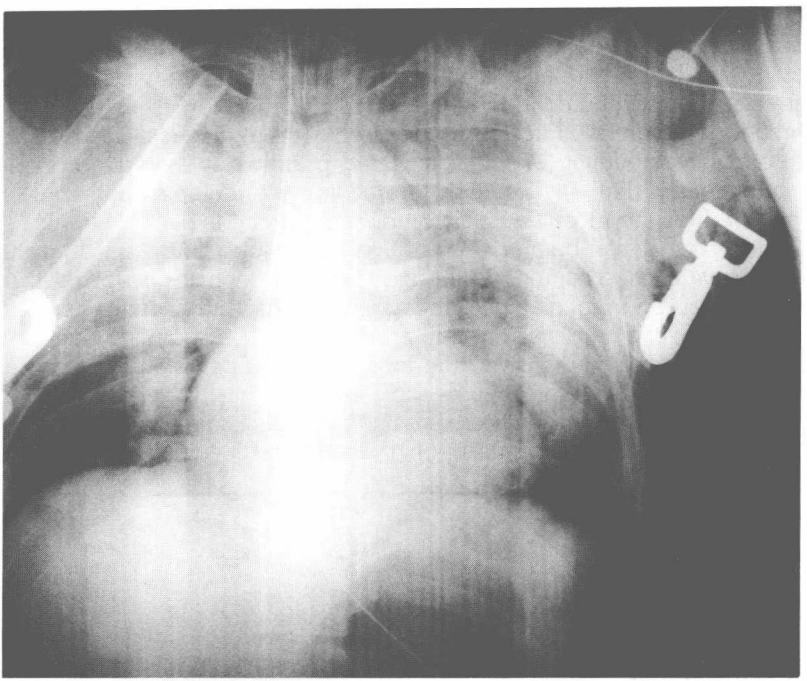

FIG 3.
Same 37-year-old patient as in Figure 2. Three hours later chest radiograph reveals rapidly progressive pulmonary contusions, and the patient is at this point in severe hypoxemic respiratory failure. Note the left pulmonary hilum sign suggestive of a leaking torn thoracic aorta.

become apparent after fluid resuscitation. Indeed, pulmonary contusions tend to worsen over a 24- to 48-hour period and then slowly resolve unless complicated by infection, cavitation, or ARDS.

Posttraumatic pulmonary pseudocyst, although unusual, should be considered in all adults sustaining a major pulmonary contusion. A PPP typically evolves over the first week from a dense pulmonary contusion into a nonspecific air- or fluid-filled loculation seen on plain films. Early assessment of blunt chest trauma with computed tomographic scanning has shown that the majority of pulmonary contusions are associated with unsuspected benign pseudocysts.[42] Long recognized to be benign entities in children,[11–13] PPPs typically follow relatively minor chest injuries. A pliable chest wall presumably transmits kinetic energy more efficiently to the underlying lung in these younger patients. Usually there is subtle symptomatology consisting of fever, minimal hemoptysis, and leukocytosis associated with plain-film chest x-ray findings of an overt cavitary lesion. But virtually always there is progressive resolution over a 2-month period in these children. This has not been our experience with adults. The majority of our patients with PPPs have sustained a severe pulmonary contusion

secondary to massive blunt trauma. Associated shock and extrathoracic injuries are common, and the PPP can progress into recalcitrant lung abscesses.[14] When adult patients with severe chest trauma have signs of persistent sepsis, computed tomographic scan clarification is mandatory to search for a potentially infected pseudocyst. Prompt diagnostic aspiration is a key triage maneuver. A simple infected pseudocyst should be drained percutaneously (Fig 4), but early thoracotomy and lobectomy must be considered for the more unusual complex pseudocysts.

Bronchoscopy can be a valuable diagnostic adjunct in the initial evaluation of severe chest injuries. Although tracheobronchial disruption is uncommon, it is an immediate life-threatening injury.[43–46] Cough, hemoptysis, subcutaneous emphysema, and a large air leak are clinical clues; suggestive chest x-ray findings include lobar collapse, persistent pneumothorax, pneumopericardium, deep cervical emphysema, or peribronchial air. Unfortunately, these are neither sensitive nor specific indicators. The clinical presentation also depends upon the size and location of the disrup-

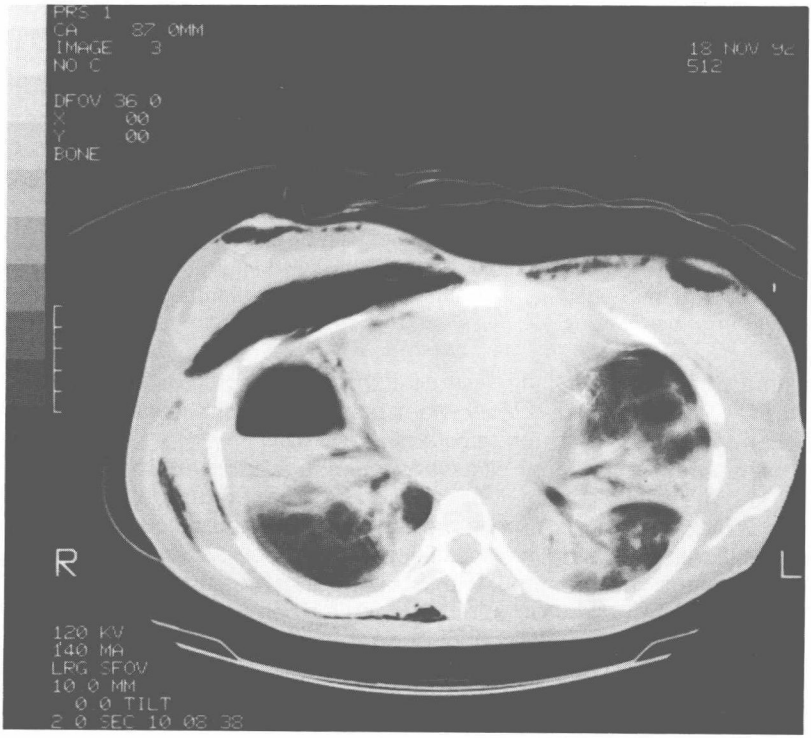

FIG 4.
Chest computed tomographic scan of an 18-year-old female involved in a high-speed motor vehicle rollover on postinjury day 15. Note the 5-cm cavity with an air fluid level. This sample infected posttraumatic pulmonary pseudocyst was drained by a transthoracic catheter.