Bilateral Pneumothorax Following Air Bag Deployment*

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Air bags have been shown to decrease mortality from automobile accidents. Herein is a unique case of bilateral pneumothorax following deployment and rupture of an air bag with no other associated chest trauma. One may posit that rupture of the air bag allowed high-pressure gases to be expelled into the patient’s lungs resulting in explosive barotrauma. (CHEST 1998; 114:624–626)

Key words: air bags; barotrauma; pneumothorax

Air bags are an automatic crash protection system that has been shown to decrease mortality from automobile accidents. Analysis of crashes involving motor vehicles equipped with this supplemental restraint system have shown them to provide a reduced fatality risk of 31% in purely frontal crashes, 19% in all frontal crashes, and 11% in all crashes.1 While these devices protect against major life-threatening injuries, they have been reported to be responsible for multiple minor ones.2 A review of the medical literature has revealed several examples of injuries to the chest associated with functioning air bags. Often, major thoracic trauma has been associated with concomitant rib fractures resulting in underlying pulmonary parenchymal injury. Lung injury without chest wall pathologic findings is less common. Herein is a unique case of bilateral pneumothorax following deployment and rupture of an air bag with no other associated chest trauma.

CASE REPORT

A 36-year-old woman was involved in a motor vehicle accident on January 23, 1997. She was the unrestrained driver of a Chrysler minivan when she collided with an embankment. The van was reported to have been traveling at 35 miles per hour (mph). The steering wheel was noted to be deformed, and the driver’s side air bag deployed and ruptured. There was no fracture of the windshield, and the inner cabin of the vehicle was intact. She presented to the emergency room at Allegheny University Hospitals, Hahnemann Division, Philadelphia, with a complaint of chest pain on deep inspiration. There was a history of loss of consciousness. She denied dizziness, nausea, or vomiting. She denied visual, auditory, or cardiac complaints.

The past medical history was noncontributory. There was no history of asthma or emphysema. She denied smoking or illicit drug use and took no medications. There was no history of previous trauma, surgery, exposure to chemicals, or foreign travel.

The patient was cooperative and in no apparent distress. The blood pressure was 94/62 mm Hg; heart rate, 90 beats per minute; and respiratory rate, 20 breaths per minute. The Glasgow coma scale score was 15, Glasgow coma scale points 5. Champion trauma scale 11, for a total trauma score of 16. The physical examination disclosed upper cervical tenderness. The face, neck, and upper thorax demonstrated no abrasions or erythema. The lungs had equal breath sounds. There was mild costal tenderness to palpation on the left side. The sternum was stable and nontender. The abdominal examination revealed left upper quadrant pain without rebound or guarding. Examination of the extremities showed no abnormalities.

Routine trauma laboratory work was completed, and all values were within normal limits. The patient underwent a CT scan of the head, chest, and abdomen. The head and abdominal CT scans were normal. The CT scan of the chest demonstrated large bilateral pneumothorax (Fig 1). Two Heimlich valve thoracostomy tubes were placed. A follow-up chest roentgenogram showed improvement on the right side but failed to show expansion of the left lung. A 20F thoracostomy tube was inserted on the left side, and the Heimlich tube was removed. Serial chest radiographs over the next several days demonstrated improvement. On the 5th hospital day, both thoracostomy tubes were removed. A small apical pneumothorax on the left and complete collapse of the right lung subsequently occurred. A 20F thoracostomy tube was inserted into the right pleural space. The right lung improved but never completely expanded. The drainage system demonstrated a continuous air leak. On the 17th hospital day, another CT scan of the chest showed a large right anterior pneumothorax. On the 19th hospital day, the patient underwent video-assisted thoracoscopic surgery. During the procedure, there was no demonstrable evidence of chest wall trauma. A small reddish lesion was identified at the apex of the lung. In addition, whitish deposits were identified along the parietal pleura, the nature of which was unclear. The apical lesion was resected, followed by lysis of fibrinous adhesions and mechanical pleurodesis of the right hemithorax. Anterior and posterior 28F thoracostomy tubes were placed. On the 4th postoperative day, the thoracostomy tubes were removed with complete resolution of the pneumothorax. The pathology report of the pulmonary lesion was a ruptured bleb. The patient was discharged to home without any further complications.

DISCUSSION

The first production of vehicles in the United States to offer seatbelts as an option came from Ford and Chrysler in 1955. In 1964, most US automobiles were equipped...
with standard lap belts, and in 1968 lap and shoulder belts were required on all US production cars. In 1974, the three-point seat belt with the lap and shoulder belts combined at the buckle became the industry standard. Air bags were mandated for a fixed percentage of cars sold in the US by all manufacturers beginning with model year 1987. Since 1990, all new US production cars have been required to have some form of automatic protection.

A typical air bag system consists of three units: air bag module, crash sensor, and diagnostic unit. The bag itself is either vented or porous. When the crash sensor, located in the front of the car, senses a deceleration indicative of a crash severity that is associated with high-risk injury, a signal is sent to the inflator triggering the device. Inflation occurs when the force anticipated is equal to hitting a brick wall at 10 to 15 mph or a similar sized vehicle at 20 to 30 mph.\(^1\) The system fully inflates in approximately 1/20 of a second after impact and deflates in approximately 4/20 of a second.\(^1\)

The current technology involves pyrotechnic oxidation of sodium azide by several oxidizing agents producing a nitrogen gas which inflates the bag. The major by-product of this chemical reaction is a metallic sodium aerosol. This reacts with water vapor and carbon dioxide to produce sodium hydroxide. This quickly converts to sodium carbonate.\(^3\) However, there are other chemicals used, such as metal oxide, chlorates, nitrates, and sulfides, which serve as oxidizing agents.\(^4\) This reaction can cause the bag to inflate at an average velocity from 50 to 138 mph\(^2\) and deliver a shock wave traveling at 200 kilometer/h.\(^6\) As one author states,\(^7\) the force of impact against a human torso can be hazardous should the air bag interact with the occupant during that period.

Although fatalities from automobile collisions have been reduced by the introduction and use of passenger restraint systems,\(^8\)-\(^10\) including air bags,\(^11\)-\(^13\) injuries from these mechanisms have been described.\(^14\),\(^15\) A review of the medical literature revealed several types of air bag injuries, including ocular\(^6\),\(^16\),\(^17\) and orthopedic.\(^18\),\(^19\) One report\(^20\) suggests that the air bag deployment threshold is set too low, resulting in an increase in air bag-related injury. Others\(^4\),\(^6\) have demonstrated that the aerosols and gases generated by air bag deployment are harmful to asthmatics. Finally, some reports suggest that the air bags themselves were the principle cause of death.\(^12\),\(^21\),\(^22\)

Our review of the medical literature revealed no similar scenarios as the one presented in this case. The explosion of high pressure gases could take the form of a jet of positive ventilation, causing the lungs to be overinflated. Barotrauma such as this could easily cause a pneumothorax. Metallic sodium aerosol, the by-product of the air bag inflation, would be inhaled and converted to sodium hydroxide in the lungs. This could explain the whitish deposits and the local inflammatory reaction found during the video-assisted thoracoscopic surgery procedure. Another explanation for the bilateral pneumothorax without associated chest wall damage relates to the properties of the chest wall in younger patients. It is believed that the more elastic and pliable chest walls of young people permit the transmission of kinetic energy more efficiently to the underlying lung parenchyma. High-velocity impacts with low displacement of the chest wall (concussive forces) result in tear or shearing stress that exceeds the elastic properties of the lung. Low-velocity impacts with high displacement of the chest wall (compressive forces) result in high intrapulmonary and airway pressures generating a bursting explosive-like phenomenon.\(^23\) In either case, it is conceivable for minor or major lung trauma to occur without concomitant musculoskeletal involvement.

In conclusion, the occurrence of bilateral pneumothorax following deployment and rupture of an air bag is a unique case, particularly without associated chest wall trauma. This report supports the idea that rupture of the air bag allowed high pressure gases to be expelled into the patient’s lungs, resulting in explosive barotrauma. Efforts on the part of the motor vehicle industry need to readdress the safety, efficacy, and quality of material used to manufacture air bags.

REFERENCES


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