G131 Blast Overpressure After Tire Explosion: A Fatal Case

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The goal of this presentation is to present a rare case of fatal tire explosion, the rarity of the event and the typical histopathological findings make the case peculiar.

This presentation will impact the forensic science community by presenting an integrated study in association with engineers helping to investigate damaging effects of blast overpressure, in a world where tire-blast injuries are not so common and the injuries of the nature described are quite rare and are hardly reported in forensic literature.

Blast Overpressure (BOP) is defined as the increased pressure over atmospheric pressure which is associated with a blast from explosives or weapons. BOP may cause primary, secondary, tertiary, and quaternary injuries. Primary blast injury occurs from an interaction of the pressure wave and the body. Secondary blast injury results from other object invested by the pressure wave impact against the body surface. Tertiary blast injuries occur when the body is accelerated from the blast wave at first and is then abruptly decelerated on rigid objects. Quaternary blast injuries are defined as those injuries of victims of explosions that due to the collapse of a building where the explosion took place. Tire-blast injuries are not so common and the injuries of the nature described are quite rare and are hardly reported in forensic literature. A case of a fatal tire explosion occurred after tire repair and inflation will be presented. Explosion occurred suddenly, strictly close the man who was put five meters far from his site. Rescue maneuvers were unsuccessful and death was declared. Forensic pathologist's crew investigated the crime scene: a 44 cm in length tear was recorded on tire. Blood stains on the ground and environmental structures were also detected. A complete postmortem examination was performed the day after death. Large scalp laceration was detected at external examination. Bruises on face and chest were also recorded. At gross examination, cranium vault was unremarkable except for a mild hemorrhagic infiltration of the *galea capitis.* Brain was normal in size and volume with mild cerebral edema. Diffuse subarachnoid hemorrhage was observed in both hemispheres. Linear fracture of the skull base was detected. Multiple bilateral rib fractures with mild hemothorax were recorded. Lungs were increased in size and volume, reddish colored, both; hemorrhagic foam was detected on the main bronchi. Hemoperitoneum was also recorded; multiple lacerations of liver, kidney, and spleen, deeping into the parenchyma of the viscera were detected. At histological examination with H&E subarachnoid and intraparenchimal hemorrhage were detected on brain. Acute emphysema, pulmonary edema, and hemorrhages were also described with intense congestion of the septum vessel. Venous air embolism was also recorded and investigated by means of immunohistochemistry (antibodies anti-CD 61 and fibrinogen). Capsular multiple tears and subcapsular and intraparenchimal hemorrhages were also detected at liver, spleen and kidneys microscopic examination. Polivisceral stasis was recorded. Intoxication of alcohol or drugs of abuse was excluded at toxicological investigation. Acute respiratory failure was indicated as the cause of death. A

multidisciplinary approach with engineers let us to establish blast overpressure after tire explosion. After that, impact of blast wave on the thorax produced chest and pulmonary injuries (primary blast injuries). Also abdominal viscera injuries were also attributed to blast overpressure effect. The displacement of the body on the ground and environmental structures after explosion produced scalp laceration, subarachnoid hemorrhage, and cranium fractures (tertiary blast injuries).

Blast Injuries, Tire Explosion, Overpressure

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