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CASE REPORT

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Laceration of the thoracic aorta from a .22 lr bullet

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Abstract A gunshot wound (.22 long rifle) to the chest including perforation of the aorta ascendens is presented. The small wound tract in soft tissue was characteristic for this type of ammunition and did not show any special peculiarities. However, arterial injury was not restricted to two small perforations, as expected in the light of previous wound ballistic findings. Three large longitudinal ruptures (3.0–4.5 cm in length and two of them independent of the perforations) additionally occurred, which led to rapid exsanguination. This extraordinary extent of vascular injury can be explained by perforation of the artery during the ventricular ejection phase, which causes a considerable dilation of the aorta analogous to a windkessel. The pre-existing dilation enables intraluminal temporary cavitation to have an "explosive" effect despite the high elastic tolerance of the vessel wall. The importance of tissue characteristics in gunshot wounds in general and the possible role of temporary cavitation inside the vessel in vascular gunshot wound production in particular are stressed.

Key words Aorta · Arterial gunshot wounds · Wound ballistics

Introduction

Major arterial injuries and especially injuries to the aorta are severe complications of gunshot wounds. In an experimental study, Moore et al. (1954) showed that a .22 long rifle bullet produces small and isolated, stellate defects of entrance and exit when it perforates the normotensive aorta of a dog. The largest external size of the perforations varied from 0.2–1.1 cm. Amato and co-workers (Amato et al. 1970, 1971; Amato and Rich 1972) found that slow spheres (305 m/s) push the artery slightly ahead before penetration and that temporary cavity effects are negligible. This results in two small perforations and in local stretching of the vessel. Fast spheres (914 m/s) completely cut the arterial wall at impact followed by a considerable temporary cavity which stretches and sways the torn ends of the vessel. The same applies to centerfire rifle bullets (Amato and Rich 1972). If the high elastic tolerance of the arterial wall is exceeded by temporary cavitation, additional injury may occur with loss of the endothelium and ruptures of elastic membranes, the intima, the media or the whole vessel wall (Rich et al. 1969; Amato and Rich 1972). Pollak (1987) reported five fatal wounds of the aorta from handguns (caliber .22–.38) and found small perforations only 1–2 mm in diameter with short tears radiating from the defects. These findings were verified in experimental gunshots to aortas removed postmortem.

A simple but interesting case of a gunshot wound to the thoracic aorta is presented. Initially, it seems to contradict the reported experimental findings but on closer examination, the extraordinary extent of injury can add to the understanding of vascular gunshot wound production.

Case report

A 35 year old man sustained a gunshot wound to the chest from a .22 rimfire rifle from a distance of 1–2 m. The man then staggered several steps and collapsed. Upon arrival of the emergency ambulance he was declared dead. The trajectory crossed the thorax from right to left, backward and upward. The bullet entered 1 cm above the right nipple and then passed through the intercostal space, the upper lobe of the right lung, the pericardium, the aorta ascendens, the upper lobe of the left lung and the first intercostal space and was found embedded in the left M. trapezius during autopsy. The trajectory without bone contact had a total length of 31 cm and the aorta was perforated after 14 cm (Fig. 1). The circular entrance wound and the perforations of the pleura and pericardium were small, approximately 5 mm in diameter, and the tiny wound

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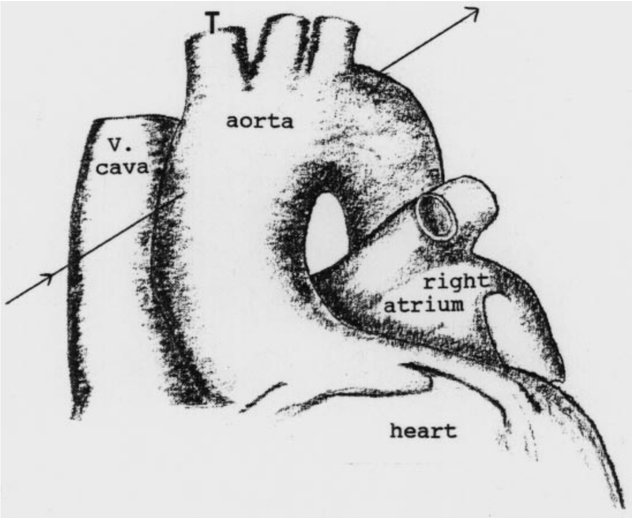

Fig. 1 Schematic drawing of the aorta ascendens and the aortic arch. The trajectory is indicated by the arrow. T = Truncus brachiocephalicus

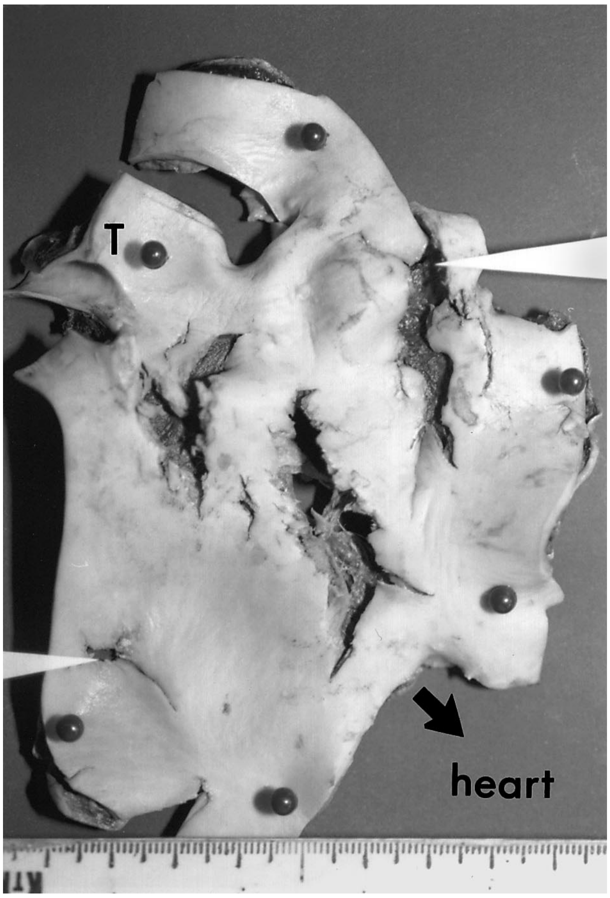

Fig. 2 Injury to the aorta ascendens after fixation of the vessel. Each mark on the scale represents 1 mm. The two arrows indicate the entrance (below) and exit (above) defects. T = Truncus brachiocephalicus. The anterior wall of the vessel was incized, resulting in a view on the whole circumference of the wall. The large central rupture 4.5 cm in length is complete. In the two lateral ruptures, the remaining adventitia is severely overstretched

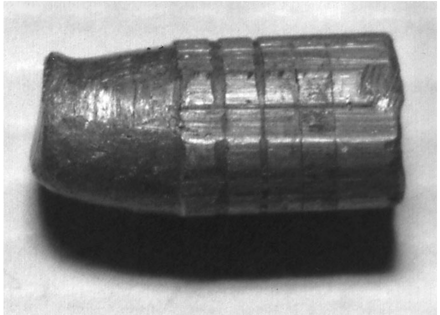

Fig. 3 The .22 long rifle round nose lead bullet after recovery from the body. The tip is flattened but the diameter is not expanded and the bullet did not fragment

tract in muscle and lung tissue showed minimal bleeding. However, injury to the thoracic aorta (Fig. 2) was not restricted to two small stellate perforations approximately 5 mm in diameter. Additionally, three roughly serrated longitudinal ruptures 3.0–4.5 cm in length resulted in laceration of the vessel. Two of these ruptures did not have contact with the perforations and one originated from the exit defect. The large defects caused profuse bleeding into the mediastinum and a haematothorax on both sides (1.5 l each). The arteries including the aorta showed only slight arteriosclerosis and hypertension was not known. The recovered projectile was a 2.6 g, .22 long rifle (lr) round nose (RN) lead bullet (Dynamit Nobel AG, Fig. 3).

Histology of the vessel wall (staining: HE, Azan and Elastica van Gieson) exhibited additional damage with loss of endothelium and elastic membrane ruptures. Small hemorrhages inside the media extended at most a few mm from the macroscopical injury. Arteriosclerosis, medianecrosis or vital signs (e.g. infiltration of inflammatory cells) were not found.

Discussion

The injury reported did not differ from the usual "harmless" appearance of .22 rimfire injuries except for the wound of the aorta ascendens. The large longitudinal ruptures independent of the small stellate perforations could not be expected from the wound ballistic findings reported before. Wound ballistics is centred around the missile-tissue interaction. The .22 long rifle bullet used has a mass of 2.6 g and a muzzle velocity of approximately 330 m/s (Sellier and Kneubuehl 1994). The bullet did not fragment or deform and the trajectory showed no indication of tumbling since the pleural and pericardial perforations were small. The importance of tissue characteristics (density, elasticity, anatomical structure, etc) for the outcome of gunshot wounds has been stressed repeatedly. Elastic tissues such as the lung, skin, muscle or bowel wall are able to absorb much of the radial tissue displacement from temporary cavitation, thereby limiting additional injury around the permanent tract (e.g. Kocher 1875; Harvey et al. 1945; Fackler et al. 1984; Karger 1995). In particular, the high degree of elasticity of arteries counteracts the effect of temporary cavitation (Amato and Rich 1972; Harvey et al. 1945). Therefore, a bullet possessing a high wounding potential does not necessarily realize this potential (Fackler 1989).

The case reported is yet another, albeit contrary example for the importance of tissue characteristics in ballistic injury. A slow and light-weight bullet can have a tremendous wounding effect which cannot easily be suspected from it's ballistic characteristics if the appropriate tissue characteristics are present. A vessel can be compared to a liquid filled cavity. The possible "explosive" cavitational effect of a missile in a liquid filled cavity such as a can or a head full of water (e.g. Kocher 1875) or an intact head (e.g. Butler et al. 1945; Karger 1995) has been well documented. The aorta belongs to the elastic type of arteries functioning as a windkessel. These act like an elastic chamber storing blood during the ventricular ejection phase and immediately thereafter when the aortic valve is closed. When storing blood, the walls of the thoracic aorta are already displaced by the increased pressure from within. A less than normal stretching would displace the walls beyond the tolerable elastic limits and therefore the extent of injury should be greater compared to non-dilated vessels.

The .22 lr bullet reported likely perforated the vessel during this phase of considerable dilation. Although the temporary cavity inside the vessel was certainly small due to the ballistic characteristics of the missile, the intraluminal pressure increase and fluid displacement was obviously sufficient to exceed the tolerable elastic limit and thus cause a massive laceration of the vessel secondary to the perforation defects. This theoretical explanation is strongly supported by experimental gunshots to aortas of dogs (Moore et al. 1954), where the size of the resulting vascular defects in hypertensive aortas approximately doubled those in normotensive aortas and showed a pronounced longitudinal splitting. Blunt trauma causing a distension of the aorta usually results in a transverse rupture of the vessel wall (e.g. Tedeschi et al. 1977), whereas the longitudinal splittings observed resemble those seen in ruptures of aneurysms, where the wounding mechanism (intraluminal pressure) is comparable.

Amato and co-workers (Amato et al. 1970, 1971; Amato and Rich 1972) did not specifically mention effects of temporary cavitation inside the artery. In "low-velocity" wounds the small temporary cavity did not exceed the elastic limit of the vessel wall, in "high-velocity" wounds the artery was completely transected during perforation and the temporary cavity subsequently acted from outside. The case reported, however, indicates that intraluminal temporary cavitation can result in ruptures independent of the perforations, which can combine to a massive laceration. So radial fluid displacement inside the vessel additionally may play a major role in wounding of arteries despite their high elastic limit, especially if their windkessel function creates moments of high vulnerability to stretching from inside.

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