Traumatic Rupture of the Aortic Isthmus: An Emergency?

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Abstract. The objectives of this study were to explore the hypotheses that: (1) patients with traumatic rupture of the aortic isthmus (TRA) who have not exsanguinated into the pleural cavity upon hospital presentation are unlikely to develop rupture of the hematoma during the time necessary to investigate all injuries and attend to those of more immediate danger; and (2) appropriate medical therapy can prevent free rupture of the hematoma. The medical records of 112 patients who were proved to have TRA at the isthmus resulting from injury within the preceding 7 days were reviewed. Fifty of these patients received medical treatment aimed at decreasing aortic wall stress; 46 were managed under a formal protocol. The available English language and European literature for the past 15 years was surveyed for evidence of the effects of delay between injury and aortic repair. Eight patients died before aortic repair, six of aortic exsanguination (all within 4 hours of injury). Of 77 patients for whom the time of injury was recorded and the aorta was repaired, 36 were repaired within 12 hours of injury and 41 between 12 hours and 24 weeks; none developed aortic hemorrhage. No patient receiving adequate medical therapy died of rupture of the hematoma. Other major surgery preceded aortic repair in 33 patients. We conclude that the concept that traumatic rupture of the aorta should always take priority over other injuries is incorrect. Pharmacologic reduction of wall stress appears to decrease the probability of rupture of the periaortic hematoma.

It has been conventional teaching that traumatic rupture of the aortic isthmus (TRA) represents a dire emergency because of the imminent danger of rupture of the periaortic hematoma, with sudden exsanguination, and that surgical repair must be carried out at the earliest possible moment. This concept is primarily based on autopsy studies, some 36 years old. The seminal publication of Parmley et al. [1] emphasized that more than 90% of the patients died within 24 hours of injury. This point is widely cited as indication for surgery at the earliest possible time; however, this figure includes 86.2% who died before reaching the hospital. Of the 125 cases of isthmus injury, only 24 reached the hospital alive: two died within minutes and eight (36.4%) died in less than 4 days. Priority of management of other injuries and transfer to trauma centers are greatly influenced by this concept. Other life-threatening injuries, which are present in most patients, increase the risks of aortic surgery or transfer and raise serious dilemmas regarding appropriate management.

Death due to TRA is caused by exsanguination into the pleural cavity. When it occurs prior to arrival at the hospital, no clinical decision is required of the surgeon, although these individuals are frequently included in published reports. Patients who are actively bleeding into the pleural space (class IA) and cannot be stabilized hemodynamically usually die prior to or during desperate thoracotomy. In most patients seen by surgeons, the pseudoaneurysm or hematoma is still contained within the mediastinum (class IB). In these patients there are two critical questions: What is the risk of catastrophic free intrapleural hemorrhage? Can conversion from a contained pseudoaneurysm (class IB) to a free rupture (class IA) with exsanguination be predicted or even prevented? To help estimate this risk, the proportions of patients who rupture while awaiting aortic repair and the time lapse between injury and repair (or death prior to surgery) should be considered. Likewise, the proportion of patients who develop chronic pseudoaneurysms and live for years, usually without symptoms, would be relevant, but the likelihood of this occurrence is unknown.

The hypothesis that patients with TRA who have not yet exsanguinated into the pleural cavity (class IB) are unlikely to develop free rupture of the hematoma (class IA) during the time necessary to methodically investigate all injuries and attend to those that are of more immediate danger is explored. We reviewed our cases of acute (n = 112) and chronic (n = 9) TRA treated between August 1964 and January 15, 1993 (Table 1) and those in the recent published literature. Because medical therapy might have a favorable impact on the probability of free rupture and allow delay of aortic repair while other injuries are managed, pharmacologic control of aortic wall stress has been investigated in the last 46 consecutive patients.

Only ruptures at the isthmus (which constitute more than 95% of clinical cases) diagnosed within 1 week after injury (acute: ATRA) and chronic traumatic aneurysms (CTRAs) in the same location are reported.

Methods

Clinical Series of ATRA

Patients who were dead on arrival were excluded. Of the 112 patients admitted to the study, 93 (83.1%) were male. The ages of the patients ranged from 15 to 87 years (mean 33 years) (Fig. 1). Motor vehicle accidents were the cause in 104 of the patients. The
Table 1. Treatment and associated mortality of all patients in this series.

<table>
<thead>
<tr>
<th>Condition</th>
<th>No. of patients</th>
<th>No. of deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta repaired</td>
<td>101</td>
<td>11</td>
</tr>
<tr>
<td>Died before repair (aortic</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>exsanguination, n = 6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical treatment only</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>112</strong></td>
<td><strong>21</strong></td>
</tr>
</tbody>
</table>

Six patients died of their TRA prior to surgical repair.

*Medical treatment also (n = 47).

None died of TRA.

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Fig. 1. Age distribution of all patients in this series.

Anurysms were deemed to be “chronic traumatic” when they were localized to the area of the aortic isthmus, there was no evidence of other significant aortic disease or peripheral aneurysm, and there was a clear history of prior decelerating, accelerating, or serious blunt chest trauma.

Specific factors investigated included (1) time from injury to arrival; (2) time from injury to aortogram; (3) time from injury to aortic repair; (4) occurrence of massive intrapleural (or chest tube) exsanguination and its relation to time variables and specific circumstances at that moment (e.g., aortogram, anesthesia, thoracotomy, hemodynamic instability); and (5) cause of death. Not all data were recorded on all records; therefore the denominator varied among the various observations (but was indicated).

**Literature Review**

A search was carried out on Unabridged MEDLINE, 1982-1993 and previous computerized databases for all publications during the last 15 years on: “Thoracic Aorta, Traumatic, Blunt, Rupture, Transection, Tear, Injury, or Laceration, of.” Those reports that contained more than 10 cases and data adequate to estimate study variables mentioned above (e.g., relevant time periods and cause of death) were analyzed. Few reports contained complete information, but attempts were made to tabulate those variables that were specified.

**Results**

About half (52.8%) of our patients were brought directly from the scene of an accident, and the others were transferred from other institutions. There were no significant differences in mortality rates or causes of death between these two groups. Of the last 53 patients, 24 (45.3%) had systolic blood pressures < 70 mmHg and 9 (17.0%) > 140 mmHg on arrival. Hemotorax was found in more than half of the cases (64 of 112, 57.1%); it was bilateral in about half of these patients. Chest tube insertion yielded preoperative drainage of > 500 ml in 33 of 62 patients (53.2%) and > 1000 ml in 9; six of the nine exsanguinated before repair.