The coagulopathy associated with aortic aneurysms

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Summary  
The authors in this article record their experience with eighty-four patients with abdominal aortic aneurysms. Twenty-seven patients (32%) presented with ruptured aneurysms with an overall mortality of 56%. Of the unruptured aneurysms, 67% were operable with a mortality of 5.3%. The highest mortality amongst the patients with ruptured aneurysms was in the group who was shocked.

In the group with ruptured aneurysms, of those in whom platelet counts were performed, 50% were abnormally low, and 56% had evidence of abnormal coagulation. Seventy per cent of those with coagulation abnormalities died. In the unruptured group 28-2% had thrombocytopenia but no other abnormalities of coagulation.

All patients undergoing aneurysm resection should have a platelet count and a full clotting screen. Therapy should be directed to normalization of the coagulation system.

Introduction  
Since the report of Dubost, Allary and Oeconomos (1952) aneurysmectomy with homograft replacement has become commonplace. There have been occasional reports of coagulation disorders associated with aortic aneurysms, but more recently ten Cate, Timmers and Becker (1975) reported four patients with ruptured or dissecting aortic aneurysms in whom a consumptive type of coagulopathy was demonstrated. In this paper eighty-four patients with the diagnosis of aortic aneurysm seen during an interval of eighteen months are reviewed, and the association between coagulopathy and aortic aneurysms is emphasized.

Materials and methods  
Blood was collected into 10 ml plastic centrifuge tubes containing one ml of 3-13% trisodium citrate. The platelet count (Bull, Schneiderman and Brecher, 1965) was performed with an electronic particle counter, and the coagulation parameters

were screened by measurement of the prothrombin time (Quick, 1957) partial thromboplastic time (Biggs and MacFarlane, 1966) fibrinogen (Hannen, 1964) and fibrinogen degradation products (FDP) (Garvey and Black, 1972).

Patients  
There were eighty-four patients of whom seventy-three (87%) were male and eleven female. Seventy-eight patients (93%) were Caucasian and six were coloured. There were no African patients. The average age was 67 years with a range of 36-95 years (Table 1).

<table>
<thead>
<tr>
<th>Age group (yrs.)</th>
<th>No. of patients</th>
<th>%</th>
<th>Ruptured</th>
<th>Unruptured</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-39</td>
<td>1</td>
<td>1-2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>40-49</td>
<td>3</td>
<td>3-6</td>
<td>1</td>
<td>2</td>
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<tr>
<td>50-59</td>
<td>10</td>
<td>12-0</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>60-69</td>
<td>45</td>
<td>53-5</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>70-79</td>
<td>24</td>
<td>28-5</td>
<td>8</td>
<td>16</td>
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<tr>
<td>80-89</td>
<td>0</td>
<td>0-0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>90-99</td>
<td>1</td>
<td>1-2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>84</td>
<td>100</td>
<td>27</td>
<td>57</td>
</tr>
</tbody>
</table>

Twenty-seven patients (32%) presented with ruptured aneurysms. Three were inoperable, and of the twenty-four operable patients, twenty-two had resections of the aneurysm, and two had a palliative procedure, one aneurysm being wired and the second wrapped.

Fifty-seven patients (68%) presented with unruptured aneurysms. Of these, thirty-eight (67%) were operable and nineteen were considered to be irresectable, or inoperable for medical reasons. Of the fifty-seven unruptured aneurysms, thirty (53%) were asymptomatic and discovery of the aneurysm was incidental. The majority of the remaining twenty-seven patients complained of abdominal pain, backache, or a pulsatile abdominal swelling. Associated medical illnesses are given in Table 2. Of the patients with ischaemic heart disease, 50% had suffered one or more documented myocardial infarctions, and of those with peptic ulceration, four were gastric and three duodenal.

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Results

Coagulation studies

Of the twenty-seven patients whose aneurysms had ruptured, eighteen (67%) had platelet counts performed and nine of these (50%) were abnormally low. Eleven of the patients (41%) had a full clotting screen and a further three (11%) had at least a prothrombin time performed. Four patients (15%) had only a platelet count. The results are given in Table 3. Ten of the eighteen patients (56%) had one or more abnormal coagulation parameters, including two patients meeting the criteria for diagnosis of consumptive coagulopathy (Merskey et al., 1967).

Ten of the 57 patients (18%) with unruptured aneurysms had coagulation screens and a further seven patients (12%) had at least a prothrombin time measured. Thirty-nine patients (68%) had platelet counts performed, and eleven of these (28.2%) had thrombocytopenia. There were no other coagulation abnormalities. Of the eleven patients with thrombocytopenia, three were documented as having dramatic postoperative increases in the platelet count from 128 x 10^9/l to 220 x 10^9/l; from 54 x 10^9/l to 225 x 10^9/l; and from 83 x 10^9/l to 340 x 10^9/l, while a fourth patient rose from the low normal range of 182 x 10^9/l to 440 x 10^9/l. One patient with a low platelet count pre-operatively had a bone marrow examination which demonstrated a normal marrow with increased numbers of megakaryocytes compatible with a peripheral destructive defect.

Renal function

Renal function was assessed in sixty-one unruptured and thirteen ruptured aneurysms. It was found to be abnormal in twelve (19.6%) of the unruptured and four (30.7%) of the ruptured aneurysms.

Mortality

As shown in Table 4, of the twenty-seven ruptured aneurysms, fifteen (56%) died. Fourteen patients were shocked on presentation and 76% of these patients died. Of thirteen non-shocked patients, 31%

<table>
<thead>
<tr>
<th>No. of patients</th>
<th>Deaths</th>
<th>% Mortality</th>
<th>Overall mortality</th>
</tr>
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<tbody>
<tr>
<td>Total ruptured</td>
<td>27</td>
<td>15</td>
<td>56</td>
</tr>
<tr>
<td>Shocked</td>
<td>14</td>
<td>11</td>
<td>79-0</td>
</tr>
<tr>
<td>Not shocked</td>
<td>13</td>
<td>4</td>
<td>31-0</td>
</tr>
<tr>
<td>Coagulation abnormalities</td>
<td>10</td>
<td>7</td>
<td>70-0</td>
</tr>
<tr>
<td>Total unruptured</td>
<td>57</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Operable patients</td>
<td>38</td>
<td>2</td>
<td>5-3</td>
</tr>
</tbody>
</table>

Table 3. Coagulation studies in patients with ruptured aneurysms

<table>
<thead>
<tr>
<th>Patient</th>
<th>Survival</th>
<th>Platelet count n.r. 150-400 x 10^9/l</th>
<th>Prothrombin index n.r. 80-100%</th>
<th>Partial thromboplastin time (sec)</th>
<th>Control (sec)</th>
<th>Fibrinogen n.r. 200-400 mg/100 ml</th>
<th>FDPs n.r. 10 &lt; 40 μg/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Died</td>
<td>53</td>
<td>66</td>
<td>52</td>
<td>35</td>
<td>580</td>
<td>&gt; 80 &lt; 160</td>
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<tr>
<td>2</td>
<td>Died</td>
<td>235</td>
<td>42</td>
<td>350</td>
<td>47</td>
<td>36</td>
<td>&gt; 640</td>
</tr>
<tr>
<td>3</td>
<td>Survived</td>
<td>83</td>
<td>69</td>
<td>&gt; 180</td>
<td>47</td>
<td>240</td>
<td>&gt; 40 &lt; 80</td>
</tr>
<tr>
<td>4</td>
<td>Died</td>
<td>108</td>
<td>74</td>
<td>73</td>
<td>49</td>
<td>298</td>
<td>&gt; 40 &lt; 80</td>
</tr>
<tr>
<td>5</td>
<td>Died</td>
<td>64</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Survived</td>
<td>80</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Died</td>
<td>107</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>8</td>
<td>Survived</td>
<td>199</td>
<td>92</td>
<td>43</td>
<td>47.5</td>
<td>280</td>
<td>&lt; 10</td>
</tr>
<tr>
<td>9</td>
<td>Survived</td>
<td>126</td>
<td>90</td>
<td>40</td>
<td>40</td>
<td>530</td>
<td>&lt; 10</td>
</tr>
<tr>
<td>10</td>
<td>Survived</td>
<td>177</td>
<td>98</td>
<td>36</td>
<td>46.5</td>
<td>470</td>
<td>&gt; 10 &lt; 40</td>
</tr>
<tr>
<td>11</td>
<td>Survived</td>
<td>270</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Survived</td>
<td>168</td>
<td>70</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>13</td>
<td>Died</td>
<td>540</td>
<td>88</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>14</td>
<td>Survived</td>
<td>360</td>
<td>89</td>
<td>45</td>
<td>46</td>
<td>520</td>
<td>&gt; 10 &lt; 40</td>
</tr>
<tr>
<td>15</td>
<td>Died</td>
<td>80</td>
<td>68</td>
<td>52</td>
<td>48</td>
<td>180</td>
<td>&gt; 80 &lt; 160</td>
</tr>
<tr>
<td>16</td>
<td>Survived</td>
<td>239</td>
<td>84</td>
<td>43</td>
<td>52.5</td>
<td>&gt; 10 &lt; 40</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>Died</td>
<td>106</td>
<td>89</td>
<td>52</td>
<td>41.5</td>
<td>375</td>
<td>&lt; 10</td>
</tr>
<tr>
<td>18</td>
<td>Died</td>
<td>269</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

n.r. = normal range.
died. Of the inoperable ruptured aneurysms all patients died. Both patients undergoing palliative procedures survived for discharge from hospital. Seventy per cent of those ruptured aneurysms with coagulation abnormalities died.

Only two (5.3%) of the thirty-eight patients with unruptured aneurysms who underwent operation died, one of small bowel volvulus with aspiration pneumonia and the second of pneumonia, ischaemic necrosis of the sigmoid colon and progressive renal failure.

Discussion

Mortality

The mortality of 56% in patients with ruptured aneurysms and 5.3% in unruptured aneurysms represents a significant decrease when compared to the earlier series of Louw et al. (1972) who had an overall mortality of 70% in ruptured and 11.6% in unruptured aneurysms. Those patients who were shocked had the greatest mortality and the most important factor is probably hypotension resulting from free intra-abdominal rupture with the ensuing anuria and cardiac arrest. Operative intervention is indicated in ruptured aneurysms since without repair the mortality is 100%.

There is divergence of opinion as to whether asymptomatic aortic aneurysms should be resected. Louw et al. found that of unoperated patients, 19% survived for 5 years and, of those undergoing laparotomy without aneurysmectomy, the 5-year survival was 9%. The overall 5-year survival of their patients without resection was 14% but there were no 10-year survivors. Szilagyi et al. (1966) and Foster et al. (1969) reported overall 5-year survivals in untreated patients of 19% and 18% respectively, and 10-year survival of 6% and 0% respectively. Data collected from various series shown that 10–20% of small aneurysms (<6 cm) and over 40% of large aneurysms rupture within 5 years of diagnosis. However, during this 5-year period a considerable proportion died of other causes, with the result that the 5-year survival rates are only 30% in those with small aneurysms and less than 10% in those with large aneurysms.

The policy adopted was to resect all aneurysms in good-risk patients, and large symptomatic aneurysms in borderline and poor-risk patients.

Coagulopathy

Fine et al (1967) reported the first case of dissecting aneurism with multiple coagulation defects, but in a review of 505 cases by Hirst, Johns and Kime (1958) no mention was made of any abnormality in the coagulation system. In three subsequent papers on aortic aneurysms (Bromley, 1967; Mannick, 1967; Ottinger, 1975) no mention was made of thrombocytopenia or of bleeding diathesis.

Kazmier et al. (1969), Cutucudache, Brailescu and Gorun (1970), Straub and Kessler (1970) reported cases of consumption coagulopathy and Bieger et al. (1971) reported three further cases. Ten Cate et al. (1975) recently reported four patients, three with dissecting aneurysms and one with a leaking atherosclerotic aneurysm, who had abnormal coagulation parameters. One of their patients, however, had a Salmonella typhimurium septicaemia which may have contributed to the clinical picture (Gétaz and Staples, 1977).

Among eighteen patients with ruptured aneurysms in whom a platelet count was performed, 50% had an abnormally low count and two patients had the frank picture of a disseminated intravascular coagulation. In those with unruptured aneurysms, 28.2% had thrombocytopenia without other coagulation abnormalities. Bone marrow examination of one patient showed increased numbers of megakaryocytes and in four patients marked increase in platelet count was noted following the resection of the aneurysm.

The aneurysm itself may thus be the origin of the coagulopathy and contact with underlying aortic tissue, particularly pathological adventitia with increased fibrinolytic activity (Astrup and Coccheri, 1962) may be the trigger. Contact with collagen has been shown to activate the intrinsic clotting system (Niewiarowski, Bankowski and Rogowicka, 1965) and Baumgartner, Stemerman and Spaet (1971) showed a rapid and intense deposition of platelets on denuded aorta. Local turbulence in flow in the diseased vessel, perhaps with localized thrombosis may also be a contributing factor. Straub and Kessler (1970) demonstrated localization of 111I-fibrinogen in the aneurysm of their patient, and after aneurysmectomy, the aneurysm contained a large mass of fibrin which gave a positive autoradiogram.

The single most important measurement in the clotting profile of these patients is the platelet count. Fifty per cent of patients with ruptured aneurysms are operated upon with abnormally low counts. In view of the urgency of the surgery, the blood used for transfusion is usually 'bank blood' with no platelets, and the massive quantities often required for pre- and intra-operative resuscitation ensure that the platelet count is further depressed, as are other coagulation factors (Ingram, 1965).

For the patient whose aneurysm has not ruptured, a pre-operative platelet count and coagulation screen should be done and, if necessary, platelet concentrate or whole fresh blood made available. In the patient with a ruptured aneurysm a platelet count should be
performed immediately and if there is thrombocytopenia, platelet concentrates given when the aneurysm has been isolated from the circulation. After resection, it would appear that the coagulopathy corrects itself, but if there is excessive bleeding, fresh blood, or fresh frozen plasma and platelet concentrate should be given.

The mortality of operable patients whose aneurysms have not ruptured is acceptably low, but that for patients presenting with a ruptured aneurysm could be improved. Detection and treatment of abnormalities of the coagulation system should contribute to a significant reduction in mortality by ensuring better intra-operative haemostasis, and less leakage on release of the aortic clamps. Both these factors decrease the duration of anaesthesia.

Garg, Lackner and Karpatkin (1972) have shown that in those patients with peripheral destruction of platelets, there is an increased percentage of large young platelets so that platelet sizing may be a rapid and easy method of demonstrating the increased platelet turnover of a peripheral consumptive defect.

Acknowledgments

We wish to thank Ms N. Horn, Ms M. Bracher and Ms J. Hughes for expert technical assistance, Professor P. Jacobs for encouragement, and the Medical Superintendent for permission to publish.

References


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doi: 10.1136/pgmj.53.625.668

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