Dying from cardiac tamponade
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Abstract

Background: To determine the causes of cardiac tamponade (CT), focusing especially on haemopericardium (HP), as a terminal mode of death, within a 430,000 rural English population.

Methods: Our hospital mortuary register and all postmortem reports between 1995 and 2004 inclusive, were interrogated for patients dying of CT or HP. The causes of CT/HP and selected morphological characteristics were then determined.

Results: 14,368 postmortems were performed in this period: of these, 461 patients died of CT. Three cases were due to non-haemorrhagic pericardial effusion. HP accounted for the remaining 458 cases of which, five were post-traumatic, 311 followed rupture of an acute myocardial infarction (RAMI), 138 after intra-pericardial rupture of dissecting ascending aortic aneurysms (RD3A) and four were due to miscellaneous causes. HP was more commonly due to RAMI. Men tended to die from RAMI or RD3A earlier than women. RAMI or RD3A were commoner in men <70 yrs, but more frequent in women after this. Two thirds of RAMI were associated with coronary artery thrombosis. Anterior free wall rupture was commonest overall, and in women, but posterior free wall rupture was commoner in men. The volume of intrapericardial blood in RAMI (mean = 440 ml) and RD3A (mean = 498 ml) varied between 150 and 1000 ml: intrapericardial blood volume was greater in men than in women dying from either RAMI or RD3A.

Conclusion: At postmortem, CT is most often related to HP, attributable to either RAMI or intrapericardial RD3A. Post-traumatic and other causes of CT are infrequent.

Background
The pathophysiology of cardiac tamponade (CT), as a cause of death, is related to an increase in intrapericardial fluid pressure that exceeds atrial venous pressures, thereby impeding venous return to the heart[1]. Rapidly evolving HP (200 to 300 ml) is more likely to cause death from CT than slowly evolving pericardial fluid accumulation (500 to 2000 ml), the latter allowing for accommodation of greater volumes due to gradual distension of the pericardial sac[1]. The normal volume of pericardial fluid (30 to
50 ml) reflects a balance between production and reab- 
sorption[1].

The causes of CT include active or passive pericardial effu-
sion and haemopericardium (HP) consequent on trauma, 
iatrogenic intervention or, either rupture of an acute myo-
cardial infarction (RAMI) or intrapericardial rupture of a 
dissecting ascending aortic aneurysm (RD3A)[1,2]. HP 
commonly follows RAMI, RD3A or trauma [1-3]; it has 
also been described in association with malignancy[3,4], 
chemotherapy[5], homicide[6], pacing wire[7] or central 
venous catheter[8,9] insertion, endocardial biopsy[10], 
open heart surgery[11], interventional coronary artery 
procedures (atheroablative, angioplastic or stenting)[12], 
exercise stress test[13], electroconvulsive therapy[14], cor-
onary artery vasculitis[15] or dissecting aneurysm[16], 
myocardial abscess[17], infective endocarditis[3] and, 
during amniocentesis[18] or in the prenatal period[19]. 
CT been described in association with pneumopericar-
dium [20] and as a complication of intrapericardial drain 
insertion[3]. HP has been described as a complication of 
5% to 10% of patients with AMI [21][22].

Post-AMI myocardial rupture includes ventricular free 
wall rupture, ventricular septal rupture or papillary mus-

cle rupture [1,3]. Risk factors for this include age >60 yrs, 
female gender, pre-existing hypertension and lack of left 
ventricular wall hypertrophy[1].

The lateral wall, at mid-ventricular level, is said to be the 
most common site for post-infarction free-wall rup-
ture[1]. However, the lateral and inferior aspects of the left 
ventricle have been reported as equally susceptible to 
post-infarctive rupture [21][23].

We have reviewed all cases of patients dying of cardiac 
tamponade in our hospital over a ten-year period to deter-
mine the causes of cardiac tamponade and highlighted 
some associated morphological features.

Methods
Our department performs postmortems at the request of 
Her Majesty (HM) Coroner, in the County of Cornwall, 
that has a population of ~430,000. Almost all deaths in 
this catchment area, falling within the jurisdiction of HM 
Coroner, are sent to our mortuary for a postmortem. 
These deaths are principally those within the community, 
or hospital, that fall within the purview of HM Coroner 
[24]: for the most part, these postmortems are performed 
because there is no firmly established cause of death.

The Mortuary Death Register of the Department Histopa-
thology, Royal Cornwall Hospital, was reviewed for 
causes of death listed as either "haemopericardium" or 
"cardiac tamponade" between 1995 and 2004 (inclusive). 
These results were compared to a search of our textual 
postmortem report database using "moper" (a unique 
string of letters in the word "haemopericardium") and 
"tamponade" as separate search criteria within the text of 
the reports; the results of these were amalgamated and 
duplicates discarded.

All postmortem reports were retrieved and the cause of 
death confirmed as being attributable to HP or CT; the fol-
lowing parameters were then confirmed and retrieved 
from each postmortem:

1. The gender and age of patients
2. The causes of the CT or HP
3. If HP due to myocardial infarction, the
   a. Presence of coronary artery thrombosis
   b. Site of myocardial rupture and
4. Volume of intrapericardial haemorrhage

Results
14,368 postmortems were performed at the Royal Corn-
wall Hospital in the ten years under review. There were 
461 cases of CT (3.2% of all postmortems), of which there 
were three cases of non-haemorrhagic effusion (passive = 
2; infective = 1), five cases of post-traumatic HP and four 
miscellaneous causes (Table 1); these were excluded from 
subsequent analysis. Of the rest (n = 449; 98%), CT was 
due HP, as a consequence of either RAMI or 
RD3A, secondary to RAMI (n = 311; 69%) or 
RD3A (n = 138; 31%) (Figure 2).

Death due to HP, as a consequence of either RAMI or 
RD3A, is commoner in women than men (Figure 3).

Table 1: Other causes of haemopericardium.

<table>
<thead>
<tr>
<th>Cause of HP</th>
<th>Sex</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>#Left ventricular rupture</td>
<td>male</td>
<td>19</td>
</tr>
<tr>
<td>following RTA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>#Partial avulsion of right</td>
<td>male</td>
<td>33</td>
</tr>
<tr>
<td>pulmonary vein</td>
<td></td>
<td></td>
</tr>
<tr>
<td>#Avulsion crush injury to</td>
<td>male</td>
<td>35</td>
</tr>
<tr>
<td>chest</td>
<td></td>
<td></td>
</tr>
<tr>
<td>#Stab injury</td>
<td>female</td>
<td>83</td>
</tr>
<tr>
<td>#Perforation of right ventricle</td>
<td></td>
<td>84</td>
</tr>
<tr>
<td>Angiosarcoma of pericardium</td>
<td>female</td>
<td>38</td>
</tr>
<tr>
<td>Metastatic carcinoma from lung</td>
<td>male</td>
<td>69</td>
</tr>
<tr>
<td>Warfarin therapy</td>
<td>female</td>
<td>77</td>
</tr>
<tr>
<td>Malignant pericarditis (cause unknown)</td>
<td>male</td>
<td>78</td>
</tr>
</tbody>
</table>

This table defines the causes of HP, other than RAMI or RD3A, 
causing cardiac tamponade. There were 5 cases of post-traumatic 
HP. Four patients died of HP from other causes.
RAMI is a more common cause of HP than RD3A in both men and women.

The age range of non-traumatic HP in men (33 to 99 years) and women (21 to 99 years) are similar. The mean age of men dying of HP/CT as a result of RAMI (73.4 ± 8.6) (n = 120) and RD3A (72.5 ± 13.0) (n = 62) is less than those of women dying of HP/CT from RAMI (78.7 ± 9.0) (n = 191) and RD3A (76.6 ± 11.1) (n = 76). The incidence of RAMI and RD3A in each age group is shown in Figures 4 & 5; death from HP due to either cause is commoner in men than female before 70 yr but reverses after this and is commoner in women.

Coronary artery thrombosis (CAT) was recognised in 210 postmortems (67%: 82 M, 128 F); CAT was not identified in the remainder (n = 101: 38 M, 63 F). There were no significant differences in the ages of men or women with CAT as a cause of RAMI.

The site of ventricular rupture was defined in all postmortem reports (table 2), with the anterior wall (33.8%) being the most common site of rupture. Cumulative figures show anterior (42.8%) and posterior ventricular wall rupture (37.3%) being more common than apical (6%) or lateral (22.5%) ventricular wall rupture. These figures are
probably skewed by the larger numbers of females in the RAMI group.

Men were more likely to have posterior (12.2%) than anterior (10.6%) or lateral (7.4%) ventricular wall rupture; women, by contrast, had anterior (23.2%) ventricular wall rupture more commonly than posterior (19.3%) or lateral (5.8%) wall rupture.

**Figure 4**
Men die from RAMI more frequently, than women do, before the age 70 years than after it.

Patients dying from intrapericardial RD3A formed a smaller but significant group (30.5%; n = 138) of patients dying from HP. This mode of death was slightly more frequent in women (n = 76; 55%) than in men (n = 62; 45%) (Figure 3).

**Intrapericardial blood volume**
The volume of blood in the pericardial space was recorded in 246 (54%) instances (99 M, 147 F); this ranged between 150 ml and 1000 ml of blood in RAMI or RD3A (figure 6).

In addition, 800 ml of blood was seen in association with pericardial angiosarcoma, 750 ml in association with metastatic lung carcinoma and 1650 ml in association with malignant pericarditis. The volume of blood in association with Warfarin therapy was not recorded.

A difference in intrapericardial blood volume was noted between men and women whether as a consequence of either RAMI (M = 473 ml, F = 418 ml) or RD3A (M = 514 ml, F = 474 ml).

Death was associated with a slightly smaller volume of HP in-patients dying as a consequence of RAMI (mean = 440 ml) when compared to RD3A (mean = 498 ml).

**Conclusion**
There is little evidence in the literature reflecting on the causes or demographics of HP in routine postmortem practice. With the exception of individual case reports [4][6][7][10][11][12][13][14][15][16][17][18][19][20]
most of the evidence available in standard textbooks is non-referenced.

Only 3.2% of all our deaths, over a 10-year period, were attributable to CT. In our series RAMI and RD3A were the principal causes of CT, causing death as a consequence of HP. Pericardial effusion, post-traumatic CT and miscellaneous causes of CT together formed only a small group of CT-related deaths.

Although the lateral wall at mid-ventricular level [1][21][23] or the inferior wall [21] are said to be the most common sites for post-infarction free-wall rupture [1][21][23], in our series anterior left ventricular wall rupture was more common than rupture at other ventricular wall sites. This might be a reflection of the greater numbers of women in our study, anterior free wall rupture being more common in women and posterior free wall rupture in men.

By contrast to traditional risk factors of female gender and age >60 years[1], men appear to die from CT due either RAMI or RD3A earlier than women. Indeed, death from either of these causes is commoner in men than women before, but not after, 70 years of age. This probably reflects the effects of cardiogenic risks in women evolving in the postmenopausal period and of men dying earlier from other causes, including AMI without myocardial rupture.

Traditionally, acute cardiac tamponade is associated with between 200 ml and 300 ml of sudden accumulation of intrapericardial fluid[1] or, in chronic slowly evolving accumulation, of volumes between 1000 and 2000 ml[1,3]. This contrasts our experience with volumes of between 440 ml and 500 ml in association with CT due to acute pericardial accumulation of blood, in women and men respectively. In cases where HP volumes were larger than this, we speculate that this was associated with a slow leak, with or without a terminal phase of catastrophic rapid haemorrhage. We also demonstrated that slightly less intrapericardial blood volume is associated with RAMI than RD3A in fatal HP.

In conclusion we have defined the aetiology of CT in a postmortem population in rural South-West England and have described some of the salient morphological features associated with CT.

**Abbreviations**

CT – Cardiac Tamponade

<table>
<thead>
<tr>
<th>Site of rupture</th>
<th>Men</th>
<th>Women</th>
<th>Total</th>
<th>Cumulative figures, by site</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apex</td>
<td>6 (1.9)</td>
<td>13 (4.2)</td>
<td>19 (6.1)</td>
<td>Apex 6%</td>
</tr>
<tr>
<td>Anterior</td>
<td>33 (10.6)</td>
<td>72 (23.2)</td>
<td>105 (33.8)</td>
<td>Anterior n = 133 (42.8)</td>
</tr>
<tr>
<td>Antero-septal</td>
<td>5 (1.6)</td>
<td>9</td>
<td>14 (4.5)</td>
<td></td>
</tr>
<tr>
<td>Antero-lateral</td>
<td>4 (1.3)</td>
<td>10 (3.3)</td>
<td>14 (4.5)</td>
<td></td>
</tr>
<tr>
<td>Lateral</td>
<td>23 (7.4)</td>
<td>18 (5.8)</td>
<td>41 (13.2)</td>
<td>Lateral n = 70 (22.5)</td>
</tr>
<tr>
<td>Postero-lateral</td>
<td>8 (2.6)</td>
<td>7</td>
<td>15 (4.8)</td>
<td></td>
</tr>
<tr>
<td>Posterior</td>
<td>38 (12.2)</td>
<td>60 (19.3)</td>
<td>98 (31.5)</td>
<td>Posterior n = 116 (37.3)</td>
</tr>
<tr>
<td>Postero-septal</td>
<td>1 (0.3)</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>120 (38.6)</td>
<td>191 (61.4)</td>
<td>311 (100.0)</td>
<td></td>
</tr>
</tbody>
</table>

This table shows the documented sites of RAMI’s in men and women dying from HP/CT. Figures in () define the % occurrences of each type of ventricular free wall rupture to the total population of patients with RAMI. Cumulative figures represent the total of each of anterior, lateral or posterior free wall rupture as described, alone or in combination.
HP – Haemopericardium
AMI – Acute Myocardial Infarction
RAMI – Ruptured Acute Myocardial Infarction
D3A – Dissecting Ascending Aortic Aneurysm
RD3A – Rupture Dissecting Ascending Aortic Aneurysm
PM – Postmortem

Competing interests
The author(s) declare that they have no competing interests.

References

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