Z Rechtsmed (1987) 99:129–134

Zeitschrift für
Rechtsmedizin


# A Cytologic Study of the Sediments in Pericardial Fluid as it Relates to a Diagnosis of the Mechanism of Death

**A. Luna[1], M. Gomez Zapata[1], V. Vicente[2], and E. Osuna[1]**

[1]Department of Forensic Medicine, University of Murcia, Espinardo, Murcia, Spain
[2]Department of Pathology, University of Murcia, Spain

**Summary.** We have studied the sediments obtained from the pericardial fluids of 70 cadavers subject to different causes of death. The fluids were taken in the course of corresponding legal autopsies. The samples were organized according to the cause of death and cellular predominance, in the following groups, respectively: hanging, multiple trauma, craniocerebral trauma, other violent deaths, myocardial infarction, pulmonary embolism, and other natural deaths. According to cell type followed these categories: Group 1 (isolated mesothelial cells), group 2 (isolated and plated mesothelial cells), group 3 (inflammatory cells and mesothelial cells, isolated and plated), and group 4 (inflammatory cells and mesothelial cells). The statistical analysis was attained through Pearson's coefficient.

We have found a significant statistical relation ($P \leq 0.05$) between the presence or lack of inflammatory cells and the mechanism of death. In those cases with a short survival period and without cardiac affectation, the presence of inflammatory cells was practically null. Furthermore, differences in the cross-sections of inflammatory cells reflected the duration of the death process, with elements characteristic of acute inflammation revealing acute cardiac process.

**Key words:** Pericardial fluid, cytologic studies of the sediment – Cause of death, diagnostic utility of the pericardial fluid

**Zusammenfassung.** Bei 70 an verschiedenen Ursachen Verstorbenen wurde im Rahmen von gerichtlicher Sektion Perikardflüssigkeit gewonnen und das Zentrifugat untersucht.

Die Proben wurden gemäß der Todesursache in acht Gruppen geordnet: Erhängen, Polytrauma, Schädelhirntrauma, andere gewaltsame Todesfälle, Myocardinfarkt, Lungenembolie und andere "natürliche Todesfälle". Entsprechend der Zell-Typisierung wurden folgende vier Gruppen gebildet: 1. Einzeln liegende Mesothelzellen. 2. Einzelne Mesothelzellen neben

*Offprint requests to:* A. Luna (address see above)

Mesothelzellplatten. 3. Entzündungszellen und Mesothelzellen – einzeln und im Zellverband. 4. Entzündungszellen neben einzelnen Mesothelzellen. Die statistische Analyse erfolgte gemäß dem Pearson's Koeffizienten. Wir wiesen eine statistisch signifikante Korrelation ($P \leq 0{,}05$) zwischen dem Vorhandensein bzw. dem Fehlen von Entzündungszellen und dem Todesmechanismus nach. In Fällen mit kurzer Überlebenszeit und ohne Herzleiden ließen sich praktisch keine Entzündungszellen nachweisen. Andererseits spiegeln die Unterschiede in der Anzahl der Entzündungszellen die Dauer des Absterbevorganges wider. Bei akuten Herzprozessen lassen sie sich am häufigsten nachweisen.

**Schlüsselwörter:** Perikardflüssigkeit, morphologische Bestandteile – Todesursache, Zellformen in der Perikardflüssigkeit

## Introduction

The pericardial fluid provides many possibilities for the study of myocardial alterations in the cadaver. There are, however, important gaps in relation to an exact knowledge of the mechanism of its production.

In previous works, we have demonstrated such diagnostic utility [1–5] and have elaborated upon a hypothesis concerning the mixed mechanism of its production, namely the process of ultrafiltration and passive diffusion [3]. The biochemical data, complete with an analysis of the cellular elements present in the fluid, proved indispensible to this study. While the issue of secondary pericardial effusion has been addressed in previous systemic pathologic and contiguous studies [6–10], none of these studies has specifically addressed the relation between the composition of the pericardial fluid and different causes of death.

## Materials and Methods

We have studied the sediments obtained from the pericardial fluids of 70 cadavers subject to different causes of death, obtained in the course of corresponding autopsies. The pericardial fluid was extracted through the open pericardial sack, employing a sterile syringe. During extraction, hemorrhagic liquids (caused by the cardiac rupture or contamination from the thoracic cavity) were discarded. The pericardial fluid was centrifuged at 1000 *g* for 10 min. From the sediments thus obtained, four different slides were taken from each sample using Papanicolau's tinction.

The samples have been arranged in the following groups according to the cause of death:

| | *N* | Data of death (h) | | Age median (yr) |
|---|---|---|---|---|
| | | Mean | SD | |
| Multiple trauma | 10 | 12.82 | 7.32 | 41.70 |
| Craniocerebral trauma | 9 | 14.25 | 5.07 | 52.80 |
| Other violent deaths | 15 | 20.32 | 17.32 | 35.70 |
| Myocardial infarction | 7 | 13.37 | 9.32 | 52.60 |
| Pulmonary embolism | 6 | 16.32 | 7.82 | 54.20 |
| Other natural deaths | 12 | 15.32 | 9.83 | 34.20 |
| Hanging | 11 | 18.30 | 9.22 | 52.40 |

The group of 'other violent deaths' is composed of:

| | N | | N |
|---|---|---|---|
| Suffocation | 1 | Intoxication by CO | 2 |
| Electrocution | 1 | Intoxication by CNH | 1 |
| Drowning | 1 | Intoxication by I.P.O. | 4 |
| Burns | 2 | Stab wounds | 3 |

The group of 'other natural deaths' is composed of:

| | N | | N |
|---|---|---|---|
| Infant sudden death | 3 | Abortion + D.I.C. | 1 |
| Gastric cancer | 1 | Renal failure | 1 |
| Hepatic cancer | 1 | Peritonitis + cancer | 1 |
| Septic shock | 2 | Cirrhosis of the liver | 2 |

The average volume of the pericardial fluid obtained for examination has been 14.58 ± 9.463 ml.

## Results

The results were initially arranged in four groups, according to the cellular predominance:
Group 1: Isolated mesothelial cells
Group 2: Isolated and plated mesothelial cells
Group 3: Inflammatory cells and plated and isolated mesothelial cells
Group 4: Inflammatory cells and isolated mesothelial cells.

In Table 1 appear the figures of the different causes of death.

For the statistical analysis, determined with the $\chi^2$ the data were organized in two groups:

Group A.... without inflammatory cells
Group B.... with inflammatory cells

The instance of isolated erythrocytes was not taken into account. In Table 4, it is possible to observe two distinct groups, distinguished by the characteristic of the inflammatory cells present.

**Table 1.** Distribution of different group cells. Group A: Without inflammatory cells. Group B: With inflammatory cells. Group 1: Isolated mesothelial cells. Group 2: Isolated and plated mesothelial cells. Group 3: Inflammatory cells and plated and isolated mesothelial cells. Group 4: Inflammatory cells and isolated mesothelial cells

| | Group A | | Group B | |
|---|---|---|---|---|
| | Group 1 | Group 2 | Group 3 | Group 4 |
| Hanging | 0 | 3 | 5 | 3 |
| Multiple trauma | 1 | 5 | 3 | 1 |
| Craniocerebral trauma | 0 | 7 | 1 | 1 |
| Other violent deaths | 0 | 6 | 7 | 2 |
| Myocarcial infarction | 0 | 4 | 2 | 1 |
| Pulmonary embolism | 0 | 2 | 4 | 0 |
| Other natural deaths | 2 | 0 | 8 | 2 |

**Table 2.** Statistical results of the two groups with different causes of death in relation to different group cells present in pericardial fluid sediment

| | Group A | Group B |
|---|---|---|
| Craniocerebral trauma | 7 | 2 |
| Other causes of death | 23 | 38 |

$r = 5.14$
$P \leq 0.05$

**Table 3.** Statistical results of the two groups with different causes of death, in relation to different group cells present in pericardial fluid sediment

| | Group A | Group B |
|---|---|---|
| Other causes of death | 26 | 26 |
| Other natural deaths | 4 | 14 |

$r = 4.21$
$P \leq 0.05$

**Table 4.** Profiles of different inflammatory cells present in different causes of death

| | P | M | L | L + P | L + M | M + P | L + P + M |
|---|---|---|---|---|---|---|---|
| Hanging | 1 | 2 | 3 | — | 2 | — | — |
| Multiple trauma | 1 | 1 | 2 | — | — | — | — |
| Craniocerebral trauma | — | — | 1 | — | 1 | — | — |
| Other violent deaths | 2 | — | 3 | 3 | — | 1 | — |
| Myocardial infarction | 2 | — | 1 | — | — | — | — |
| Pulmonary embolism | — | — | 4 | — | — | — | — |
| Other natural deaths | 3 | — | 3 | 2 | — | — | 2 |

P = polynuclears; M = macrophages; L = lymphocytes

**Table 5.** Distribution of polynuclears in different causes of death

| | Without polynuclears | With polynuclears |
|---|---|---|
| Hanging | 7 (30.50%) | 1 (5.90%) |
| Multiple trauma | 3 (13.00%) | 1 (5.90%) |
| Craniocerebral trauma | 2 (8.70%) | — — |
| Other violent deaths | 3 (13,00%) | 6 (35.30%) |
| Myocardial infarction | 1 (4.40%) | 2 (11.80%) |
| Pulmonary embolism | 4 (17.40%) | — — |
| Other natural deaths | 3 (13.00%) | 7 (41.10%) |
| Total | 23 (100%) | 17 (100%) |

The cases with inflammatory cells were grouped according to the presence or absence of polinuclears, as markers of acute inflammatory reaction. The results expressed in Table 5 are reflective of inflammatory cells only. In two cases (p.f. no. 173 and no. 180) those of hanging and pulmonary embolism, respectively, reagent multinucleated giant cells were found.

## Discussion

The most striking factor, from our perspective, is how the data is distributed into two distinct categories, according to the nature and characteristics of the cause of death. In the majority of those cases of fulminant death, with a short or nonexistent survival period, there is a significant statistical tendency toward the absence of inflammatory cells. In this group, no cardiac affection is evident, as is demonstrated by the absence or significant reduction of cellular response. In contrast, the group of "other natural deaths" shows a significant statistical tendency toward a predominance of inflammatory cells.

It is important to note that the cellular elements present in the pericardial fluid may not necessarily correspond directly to the cause of death. Rather, they may be present as a result of a previous state (e.g., an inflammatory pericardial effusion in a polyarthritic subject who committed suicide by hanging). This issue is fundamental to a critical analysis of our data. Various overriding factors, such as the previous condition, duration, and intensity of the agony, and the nature and cause of death, however, be confirmed by our findings, inasmuch as the data reveal an absence of inflammatory reaction in a cranio-cerebral trauma or

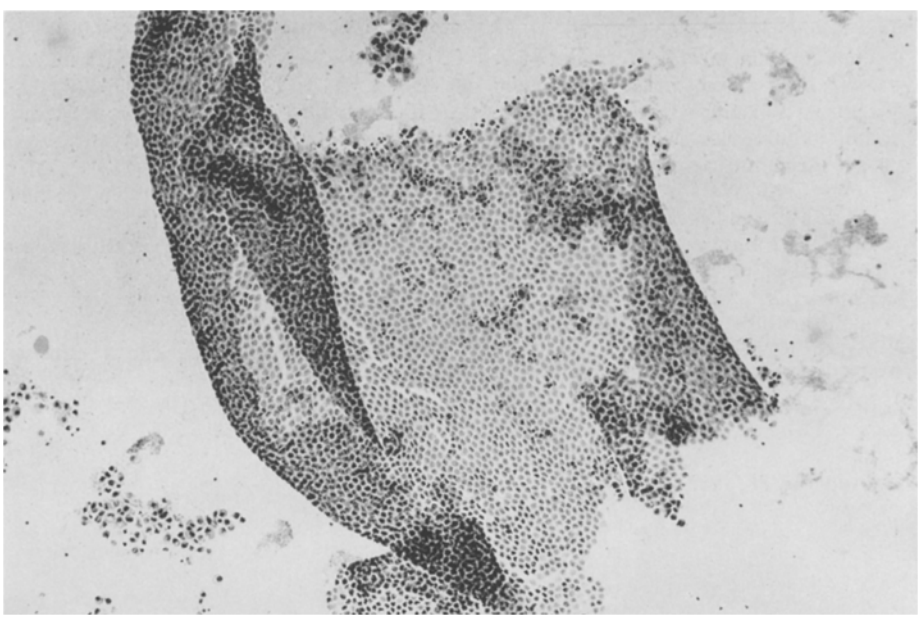

**Fig. 1.** Plated mesothelial cells in pericardial fluid sediment

when discrepancies are not presented by other factors (such as secondary pericarditis reacting to a myocardial infarction).

The data in Table 5 confirm the tendency previously described of the cellular elements characteristic of an acute inflammatory reaction (polynuclears) to be found in both the groups of significant cardiac affectation (myocardial infarction and other violent deaths) and in the pathologic groups including secondary pericardial affectation, in which the presence of pericardial effusion can be observed with relative frequency.

With the reservations already mentioned, it is evident that the presence of inflammatory cells expresses a response of the visceral flap to intense myocardial activity. The accompanying increase in permeability is made evident not only by the increase of biochemical markers, as demonstrated in previous works, but also by the cellular elements.

Finally, there is the presence of plated mesothelial cells (Fig. 1), an important characteristic of the sediments procurred from the cadavers. The origin of these cells can be accounted for by the instance of agonic suffering. Nevertheless, a specific study would be necessary to determine for certain the mechanism of their production.

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Received May 18, 1987