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Unusual finding in a water-logged corpse – hyperchylomicronemia or pulmonary fat embolism?

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Summary. During excavation work at the bank of the River Inn the corpse of a 32-year-old alcoholic male was recovered. Head injuries suggested a crime of violence. Postmortem examination demonstrated conspicuous milky turbidity of the blood, which was found by laboratory testing to be due to hyperchylomicronemia. The findings are interpreted and their relevance to the determination of postmortal head injuries is discussed. The inadequacy of double-edged knife specimens and frozen-section biopsies for the estimation of pulmonary fat embolism as a vital reaction is considered.

Key words: Hyperchylomicronemia – Pulmonary fat embolism – Double-edged knife specimens – Vital reaction – Water-logged corpse

Zusammenfassung. Bei Baggerarbeiten wurde die Leiche eines 32-jährigen Alkoholikers aus dem Inn geborgen. Die festgestellten Kopfverletzungen des Leichnams erweckten den Verdacht auf eine Fremdtötung. Im Rahmen der Obduktion zeigte sich eine auffällige, milchige Trübung des Leichenblutes, welche sich aufgrund der laborchemischen Untersuchungen als Hyperchylomikronämie herausstellte. In Zusammenhang mit der Interpretation dieses Befundes wird versucht, eine pulmonale Fettembolie als vitale Reaktion von einer bloßen Fettphanerose differentialdiagnostisch abzugrenzen. Mögliche Unzulänglichkeiten der Doppelmesserschnittmethode gegenüber der Anfertigung von Sudan III-gefärbten Gefrierschnittpräparaten zur Beurteilung einer pulmonalen Fettembolie werden aufgezeigt.

Schlüsselwörter: Hyperlipidämie – Hyperchylomikronämie – Pulmonale Fettembolie – Doppelmesserschnittpräparate – Vitale Reaktion – Wasserleiche

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Introduction

Evidence of vital reactions of the organism has always been essential in medicolegal examinations, particularly for the reconstruction of events and criminal acts. Pulmonary fat embolism has been much discussed in the relevant literature with regard to its etiology and diagnostic value. Theories are numerous, sometimes controversial, sometimes overlapping. Some authors suggest that mechanical injury causes the embolic dispersion of lipids from traumatized tissues; others discuss the mobilization of depot fat caused by trauma and stress and subsequent de-emulsification of plasma lipids or direct transport to the right heart and the pulmonary circulation via the thoracic duct. Another theory attributes pulmonary fat embolism to a disturbance in microcirculation and associated clotting disorders [2, 6, 13, 26]. Some authors have reported so-called "atraumatic" pulmonary fat embolisms [2, 7], whereas other investigators strongly doubt these findings and ascribe them to confusion with hyperlipemia and hyperchylomicronemia [8, 17–19]. Bschor and Haasch have shown that aggregated chylomicrons have to be considered in histological examinations [10]. In cases of alcoholics with fatty degeneration of the liver hyperlipemia has been discussed as one of the factors that promote pulmonary fat embolism [6, 9].

The most commonly used histological techniques for the identification of pulmonary fat embolism are frozen-section biopsy with Sudan red G staining and twin-edged knife techniques. The latter method is based on taking thin slices of tissue from both lobes of the lungs using Valentin's twin-edged knife. These specimens are immersed in water. After hemolysis of blood they are placed between two slides for microscopical examination [14, 22].

Sigrist [22] advocates the use of native twin-edged knife sections, since this particular method of specimen collecting permits the easier detection of intravascular

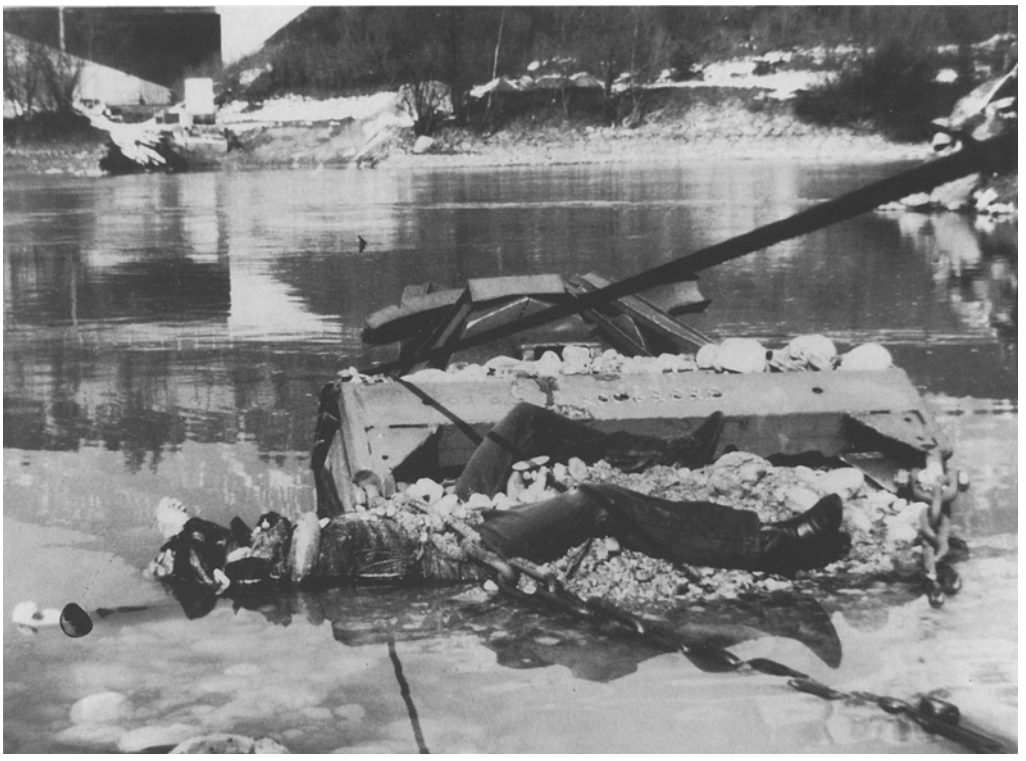

Fig. 1. Position of the corpse at the site of discovery

fatty infiltrations because they are more common and are present in higher concentrations in the "tree-like" vascular ramifications.

Case report

In February 1989 a male corpse was found during excavation work at the bank of the river Inn. The corpse was recovered by the shovel of a cable dredger together with shingle. The police were called immediately. As the officers found scalp wounds indicative of external force, the investigating magistrate ordered a medicolegal inspection and postmortem examination of the corpse.

When the public medicolegal officer arrived, the corpse was still in the original position, i.e., lying supine among excavated material and the dredger chains with the upper part of the body partly in the water (Fig. 1). The surrounding water was milky, and the turbidity increased as the riverbed was stirred up during the recovery of the corpse. The corpse could soon be identified as a 32-year-old industrial worker who had been dismissed some days before. He had last been seen alive in a nearby pub on the evening before the discovery of the body.

Autopsy and laboratory findings

Postmortem examination revealed that the scalp wounds had been caused by the dredger shovel. The underlying skull bone was partly depressed, the dura mater was open and superficial areas of the cerebral cortex were traumatized. Moreover, a few pontine veins were ruptured near the superior sagittal sinus. The subdural space of the cranial cavity contained abundant milky-bloody liquid, which was initially attributed to the inflow of river water. Finally milky-bloody liquid also drained out mainly from the open veins and white streaks became evident; we suspected an embolic dispersion of the water.

Other fresh injuries included minor hematomas on both thighs and on the right side, superficial ruptures of the liver and some cracked ribs.

Specimens from both lobes of the lung were examined using the twin-edged knife method and demonstrated moderate fat embolism, which seemed to confirm the genesis of the injuries intra vitam.

However, the first doubts were raised during the histological evaluation of the frozen section biopsies stained with Sudan red G. It was observed that the fatty substance was finely dispersed and partly permeated by cellular blood (Fig. 2).

Homogenous infiltrations of fat, which are characteristic of post-traumatic fat embolism, were scarce and mostly localized in the pre-capillary parts of the pulmonary artery, indicating that the injuries had occurred during the agonal phase.

Moreover, small amounts of sudanophilic granular material were detected in all other organs, affecting both venous and arterial vessels.

Microscopical examination of the hemolyzed blood with the polarizing microscope gave no evidence of crystalline substances comparable with those found in the river water.

Laboratory findings of the blood plasma demonstrated an elevation of triglycerides to 1075 mg%, which is at least a sixfold increase over the normal concentration. Lipid electrophoresis showed almost pure chylomicron bands. All other parameters of fat metabolism were within the normal range.

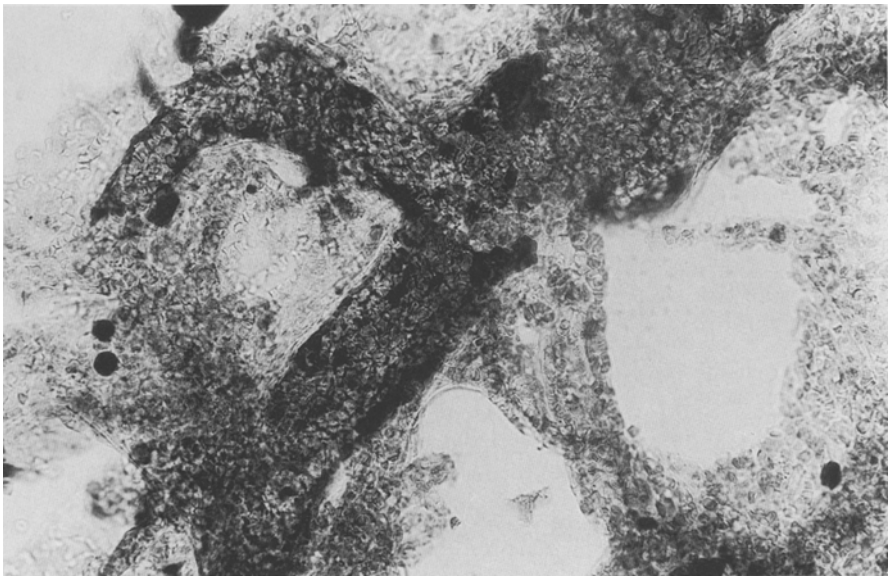

Fig. 2. Precapillary branching in the lung: fine granular intravascular sudanophilic material with infiltrations of corpuscular blood elements and marginal aggregation. Sudan red G; × 250

Discussion

Chylomicrons are the water-soluble form of triglycerides. Following food intake, they are synthesized in the epithelial cells of the intestinal mucosa and, in combination with fatty acids, transported to the venous blood via the lymphatic system and the thoracic duct. After distribution in the organism they are absorbed, split and incorporated by intra- and extrahepatic lipoprotein lipases in the endothelial cells [24]

Several different exogenous and endogenous factors may induce an isolated elevation of the concentration of chylomicrons [21].

A familial lack of lipoprotein lipase (hyperlipoproteinemia type I according to Frederickson) is transferred by recessive autosomal heredity and most commonly becomes manifest during childhood [4, 11, 16]. The most important exogenous factor causing hyperchylomicronemia is the intake of high-fat food. The peak of the digestive hyperlipidemia is achieved 2–7 h after food intake; its intensity is subject to wide inter- and intra-individual variations.

In the case of chronic alcoholism, clinical investigations have also demonstrated hypertriglyceridemia, and electrophoretic lipoprotein splitting showed a prevalence of the chylomicron and pre-β fractions [3, 5, 12, 20].

According to Reh [17, 18], severe forms of protracted hypoxemic conditions and other protracted conditions caused by hypoxemia may also cause an increase in blood fats. As Amelotti [1] observed earlier, rare cases of these phenomena could also be found in water-logged corpses. Another factor that should not be neglected, and may have been of significance in the case presented, is the effect of cold on the organism. If the body temperature falls to 26°C or less, sequestration of the intravascular fluid into the intracellular space occurs, thus producing a reduction in plasma volume. As a consequence, hematocrit and blood viscosity increase, as a result of which the relative content of blood fat is also increased.

The case presented was a chronic alcoholic with fatty degeneration of the liver caused by alcohol. The constellation of findings, particularly the histological findings, indicated protracted asphyxia due to the prolonged drowning process. Since at the time of the event the water temperature of the River Inn was considerably below 10°C, terminal hypothermia is also highly probable. However, hypothermic bleeding of the gastric mucosa (Wischnevsky spots) had not yet occurred.

There was no evidence of a congenital lipometabolic disturbance in the family history, neither were there any signs of diabetes mellitus or of any form of dysglobulinemia, such as plasmacytoma or macroglobulinemia, hypothyroidism, or status epilepticus. Pancreatitis and bronchial asthma could be ruled out.

At the final evaluation, the findings were therefore interpreted as hyperchylomicronemia of mainly exogenous origin. The protracted drowning process was considered to be the cause of death. Injuries of the head and extremities were caused during agonal floating.

The vascular occlusions detected in the double-edged knife specimens were interpreted as fat embolism and, as could be seen from the Sudan-stained frozen section biopsies, consisted mainly of fine granular condensed chylomicrons. Similar fine granular sudanophilic intravascular substances have also been described after postmortem burns and charring [23]; in these cases no typical fat embolism had developed and the chemical structure was similar to that of triglycerides in depot fat [15]. Weiler [25] observed this kind of fat phanerosis (confluence of fatty substances) in a case of Zieve's syndrome.

It can therefore be concluded that native double-edged knife specimens are not sufficient for the identification and evaluation of traumatic fat embolism. The method has several advantages: it is fast and simple to apply and sections are thicker [22]; however, as is shown by the case presented, for the assessment of fat embolism as a vital reaction the additional staining of fat in frozen-section specimens is indispensable.

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