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ORIGINAL ARTICLE

Forensic aspects of post-mortem histological detection of amniotic fluid embolism

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Abstract Amniotic fluid embolism (AFE) continues to be one of the most feared and devastating complications of pregnancy. A reliable diagnosis can be made only upon histological examination. A detection of AFE every now and then has a relevant implication on medico-legal aspects of intrapartum or post-partum maternal death. However, there are only isolated reports in the literature concerning the detection interval of amniotic fluid elements after their transfer into the lungs. The objective of this study was to determine how long after the onset of clinical symptoms the elements of amniotic fluid may be detectable in the pulmonary circulation. An autopsy, as well as a histological and toxicological examination of 29 women, who died intrapartum or post-partum were performed. AFE was diagnosed in seven women (25%). The maximum survival time of the women with AFE and also the detection interval of AF in the pulmonary vasculature was 36 h. In the lungs of the women who did not die of AFE, amniotic fluid components were not found. Thus, there is no evidence for a physiologic occurrence of AFE. In women who die some days or even weeks after delivery as a consequence of a haemorrhagic shock following post-partum genital bleeding ensuing from uterine atony, AFE should be considered as a cause of a coagulopathy.

Keywords Amniotic fluid embolism · Immunohistology · Survival interval

Dedicated to Prof. Dr. W. Eisenmenger, my teacher, on the occasion of his 65th birthday

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Introduction

Amniotic fluid embolism (AFE) continues to be one of the leading causes of maternal death [1]. The entry of amniotic fluid into the maternal circulation was first described by Meyer in 1926 [2]. However, the clinical importance of this entity was not appreciated until 1941 [3]. Two recent large population-based cohort studies have demonstrated the rate of AFE to be 14.8 and 6.0 per 100,000 multiparous and primigravida deliveries, respectively [1, 4]. The reported maternal mortality rates for AFE range from 37% to 61% [5, 6].

Classic presenting symptoms of AFE include respiratory distress, altered mental status, profound hypotension, coagulopathy and death. Over 50% of post-partum patients who had AFE exhibited a profuse haemorrhaging as the first symptom of an AFE leading to a clinical diagnosis of atonic uterine bleeding [7, 8].

There is no routine clinical diagnostic scheme to confirm AFE. Thus, the histological detection of the amniotic fluid elements in the pulmonary vascular bed is essential for confirmation of the diagnosis [7]. The histological compo-

nents of amniotic fluid emboli have been identified as follows:

1. Epithelial squames shed from the foetal skin
2. Lanugo hairs (these increase in number with the length of gestation period)
3. Fatty material—vernix caseosa
4. Mucin—generally derived from the foetal intestine and associated with passage of meconium
5. Bile pigments derived from the meconium.

The three most common routes of entry are the endocervical veins, the placental site and a uterine trauma site.

For a clinical pathologist, the sole detection of AFE suffices as a rule to explain the clinical course and to close the case. In contrast, it is of paramount importance in forensic practise to assess the dimension of AFE and to know how long the components of amniotic fluid may be detectable in the pulmonary vasculature, as life support systems allow death to be delayed by days or even weeks after the primary catastrophic event. The diagnosis of AFE may explain an unfavourable course of events which led to the death and discharge the medical staff from the accusations by relatives, who often suspect medical malpractice in a case of sudden and unexpected death during parturition.

The literature concerning the histological detection of amniotic fluid over the course of time is scanty and is based entirely on case reports [8–11]. These in turn vary considerably. In 1957, Tuller [8] described foetal epithelial cells in the pulmonary circulation using conventional histology 8 days after delivery. Keeling and Gray stated that the components of amniotic fluid can be histologically visualised several weeks after birth and cited Attwood and Delprado [12]. On account of this, they recommended looking for amniotic fluid embolism even in cases of late maternal death (>42 days up to 1 year after delivery). Attwood and Delprado were actually able to demonstrate epithelial cells in the maternal lungs 5 weeks after birth but the lung area they investigated was infarcted [13]. A similar report was made by Kaptanoglu who found foetal squamous cells in the lung 3 months after the delivery but also in the infarcted lung area [14]. The reported maximal time frames are also not applicable for a professional judgment of the medical treatment in the case of a lawsuit.

The purpose of the present study was therefore to determine how long amniotic fluid elements may be detectable in the maternal pulmonary circulation by means of histology. In a further step, the concept of the physiologic nature of AFE was to be verified, since some authors assume that small amounts of amniotic fluid often gain access to the pulmonary circulation intrapartum [15, 16].

Materials and methods

Between 1996 and 2008, post-mortem examinations were performed on 29 women who died intrapartum or postpartum. The mean age of the women was 32 years (median 32 years). Cases of late maternal death (> 42 days up to 1 year after delivery) were not included in the study.

The medical records were studied for gestational age, the mode and complications of delivery, as well as a cause of death in every case of maternal death. In some cases, information about the clinical presentation, for example, the narrations of relatives with regard to the onset of symptoms, was extracted from the investigation files.

A toxicological examination and alcoholometry were performed to identify a potential intoxication. Samples of lung, brain, heart, liver, kidney, uterus and placenta from every woman were examined histologically. Multiple sections were prepared from the lungs (n=200 from 29 autopsies), as AFE is usually not uniformly distributed in the pulmonary vasculature. Sections (5 µm thick) were prepared from the formalin-fixed paraffin-embedded tissues, which were routinely stained with haematoxylin and eosin (H&E), elastic van Gieson, Prussian blue staining, periodic acid–Schiff (PAS) and luxol fast blue/periodic acid–Schiff. Immunohistochemical staining was performed for the optimal visualisation of the amniotic fluid components and their localisation. The peroxidase–anti-peroxidase technique was performed using primary antibodies against cytokeratin (pan-cytokeratin Lu-5, monoclonal, mouse anti-human 1:200, BMA Biomedicals, Augst, Switzerland), vimentin (anti-vimentin, V9, monoclonal, mouse anti-human, 1:100, DAKO, Glostrup Denmark) and human chorionic gonadotropin (polyclonal, rabbit anti-human, 1:200 DAKO, Glostrup Denmark). Sections from the product of conception served as positive control. All sections were counterstained with Mayer's hämalaun (mixture of haematoxylin and alum) and mounted in a solution of polymers in xylene (Entellan®, Electron microscopy sciences, Hatfield, PA, USA).

The histological examination was performed by three persons who did not possess information about the clinical history and the assumed cause of death. The extent of AFE was assessed as none "–", slight "+", moderate "++" and severe "+++" [3].

Results

Of the 29 women included in the study, 26 were 9 months pregnant and three were 8 months pregnant at the time of delivery; two women died intrapartum and 27 women postpartum. The mean survival interval between childbirth and

Table 1 Results of the autopsy, the toxicological and histological investigation as well as alcoholometry (n=29)

Number	Year	Age/time of death/ gestational age	Cause of death	Survival time	Cause of death (autopsy)	Cause of death (histology and medical charts)	Toxicology	Alcohol
1	1996	18, pp, 8 months	Suicide/hanging	6 days	Suicide/hanging	Unremarkable	Negative	0.04‰
2	1996	33, pp, 9 months	Haemorrhage	7 days	Not identified	Haemorrhage	nd	nd
3	1997	28, pp, 9 months	Pelvic abscess	7 days	Pelvic abscess		nd	0.02‰
4	1997	25, pp, 9 months	Ruptured aortic aneurysm	9 days	Ruptured aortic aneurysm	Unremarkable	nd	0.02‰
5	1997	35, pp, 39 weeks	Haemorrhage	11 h	Not identified	Haemorrhage	nd	nd
6	1997	41, pp, 9 months	Coronary dissection	6 days	Coronary dissection	Unremarkable	nd	0.03‰
7	1998	37, pp, 9 months	Childbed fever	8 days	Not identified	Childbed fever	Negative	0.06‰
8	1999	33, pp, 9 months	Haemorrhage	2.3 h	Not identified	Haemorrhage	nd	nd
9	1999	33, pp, 9 months	Anaesthetic-related incident	2.5 h	Not identified	Anaesthetic-related incident	nd	nd
10	2000	34, ip, 9 months	Not identified	ip	Not identified	Not identified	Negative	0.00‰
11	2000	29, pp, 9 months	Ruptured aortic aneurysm	5 days	Ruptured aortic aneurysm	Unremarkable	nd	nd
12	2000	29, pp, 9 months	Haemorrhage	9 days	Not identified	Haemorrhage	nd	nd
13	2001	33, pp, 9 months	Epileptic seizure	11 days	Not identified	Epileptic seizure	Negative	nd
14	2001	31, pp, 9 months	AFE	14 h	Not identified	AFE	nd	nd
15	2002	31, pp, 39 weeks	Haemorrhage	4 h, 37 min	Not identified	Haemorrhage	nd	nd
16	2002	26, pp, 9 months	Allergic reaction	5.5 days	Not identified	Allergic reaction	Negative	nd
17	2003	42, pp, 9 months	AFE	3 h	Not identified	AFE	nd	nd
18	2003	31, pp, 33 weeks	Ruptured aortic aneurysm	5 h	Ruptured aortic aneurysm	Unremarkable	nd	nd
19	2004	40, pp, 39 weeks	Haemorrhage	29 days	Not identified	Hypoxic brain damage, pneumonia as consequence of haemorrhage	Benzodiazepine/ morphine	nd
20	2005	41, pp, 9 months	AFE	12 h	Not identified	AFE	nd	0.02‰
21	2005	32, pp, 9 months	AFE	8 h	Not identified	AFE	nd	nd
22	2006	29, pp, 34 weeks	Haemorrhage	2 h, 40 min	Not identified	Haemorrhage	nd	nd
23	2006	34, pp, 9 months	AFE	15 min	Not identified	AFE	nd	0.00‰
24	2007	29, pp, 36 weeks	Haemorrhage	2 days	Not identified	Haemorrhage	nd	nd
25	2007	31, pp, 33 weeks	Sepsis	30 days	Not identified	Sepsis	nd	nd
26	2007	30, ip, 9 months	Ruptured splenic artery aneurysm	ip	Ruptured splenic artery aneurysm	Unremarkable	nd	0.01‰
27	2007	36, pp, 9 months	AFE	9 h, 30 min	Not identified	AFE	Atracurium, ethomidate	nd
28	2007	38, pp, 9 months	AFE	36 h	Not identified	AFE	nd	nd
29	2008	25, pp, 9 months	Myocarditis	13 days	Not identified	Myocarditis	Negative	0.00‰

Survival time after delivery

pp post-partum, *ip* intrapartum, *np* not performed

death was 5.5 days (median 5 days, minimum 15 min, maximum 30 days).

The causes of death established at the autopsy, upon histological and toxicological examination, as well as upon the study of medical charts and investigation files were AFE (n=7, 25%), haemorrhagic shock (n=8, 28%) and others (n=13). In one case, the cause of death could not be clarified (Table 1, electronic supplementary material). Most of the pregnant women died in the hospital several days after delivery. For this reason, toxicological analysis was run on eight out of 29 cases, and the alcoholometry in ten out of 29 cases (mean 0.02‰, median 0.02‰). In two cases, the toxicological analysis showed the presence of substances consistent with iatrogenic administration for anaesthesia and pain control. One death was attributed to AFE as a consequence of traumatic uterus rupture without life-threatening blood loss following a vehicle accident.

The mean survival interval between childbirth and death in the group with haemorrhagic shock (n=8) was 6 days (median 19 h), and three women died 7, 9 and 29 days after the delivery, respectively. The survival interval in the group with AFE varied from 15 min to 36 h after presentation of the first symptoms (median 9.5 h, mean 12 h). All women with AFE were 9 months pregnant and the mean age was 36 years as well as the median age (n=3, 31–34 years; n=4, 36–42 years; Table 2). Most patients had several components of amniotic fluid in the pulmonary vasculature of every lung section simultaneously. In all cases, innumerable squamous cells and even conglomerates consisting of squamous cells were seen. Mucous strands were found in three cases, and meconium was present in two cases. In a case of traumatic uterus rupture following a vehicle accident, decidual cells and even whole chorionic villi were also observed in the small pulmonary arteries (Figs. 1, 2, 3, 4, 5 and 6, Table 2).

The extent of AFE was assessed as “moderate” in two cases and as “severe” in the remaining four cases. There was no correlation between the severity of AFE and the survival interval (Table 2). For example, in a lung of a 38-year-old woman (primigravida and primipara), massive AFE was evident 36 h after the onset of symptoms. In some cases, bone marrow emboli were found in the pulmonary vasculature consistent with resuscitation.

The components of AF could be visualised by means of H&E in every case. However, the assessment of the severity and the reliable localisation of AF components within a special organ were markedly improved and facilitated through the usage of the immunohistochemistry.

Genital bleeding as the first symptom of AFE was present in four cases. Two women displayed primarily profound hypotension. An altered mental status was

Table 2 Cases of amniotic fluid embolism (n=7)

Age/ GA	AF elements/ microthrombi	AFE extent	ST	First signs of AFE	Genital lesions	Mode of delivery
31 J G2P2	Squamous cells Microthrombi	++	14	Genital bleeding	Episiotomy	Vaginal, extirpation of the uterus
42 J G4P4	Squamous cells Microthrombi	++	4	Hypotonia, genital bleeding	Bruises of the cervix	Caesarian section (section in moribund)
41 J G1P1	Squamous cells meconium Mucous strands Chorionic villi Decidual cells Microthrombi	+++	12	Cardiovascular collapse	Traumatic uterus rupture placental separation	Caesarian section
33 J G1P1	Squamous cells Microthrombi	+++	8	Genital bleeding	No lesions	Vaginal, extirpation of the uterus
34 J G3P3	Squamous cells microthrombi	++	15	Hypotonia, genital bleeding	Disruption of the mucous membrane in cervix, perineal rupture	Vaginal (polyhydramnion)
36 JG1P1	Squamous cells Mucous strands Microthrombi	+++	91/ 2	Genital bleeding	Vaginal rupture	Vaginal (vacuum extractor), extirpation of the uterus
38 J G1P1	Squamous cells Meconium Mucous strands Microthrombi	+++	36	Neurologic symptoms, genital bleeding	Massive bruises of the cervix	Vaginal (vacuum extractor), extirpation of the uterus

GA gestational age, *ST* survival time in hours, *AF* amniotic fluid, *G* gravida, *P* para

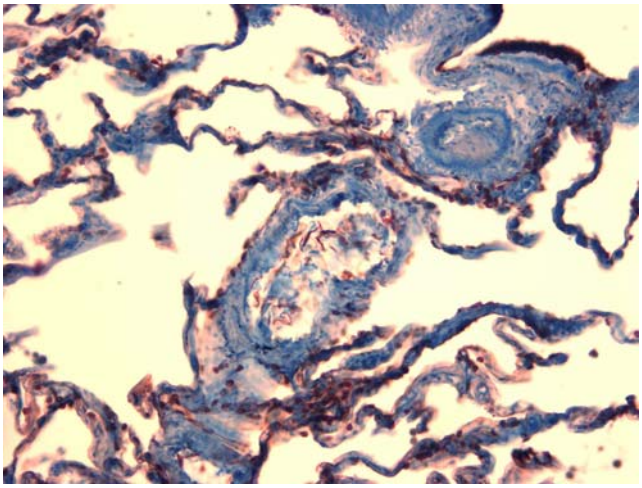

Fig. 1 Squamous cells in a lung artery, pan-cytokeratin (clone Lu-5), ×100

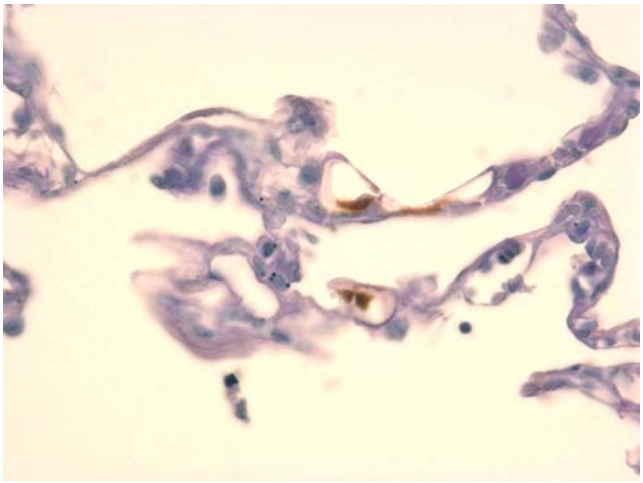

Fig. 3 Meconium in lung capillaries (PAS, ×400)

observed in only one woman, and shortly thereafter the patient went into shock. By computed tomography (CT), an intracerebral bleeding was diagnosed and assumed as a cause of death; AFE was not suspected at all. The CT findings were confirmed at the autopsy (Fig. 7). However, the histological examination revealed severe AFE as the cause of coagulopathy and resulting intracerebral haemorrhage.

From the remaining six cases, AFE was diagnosed or suspected by clinicians in three women, and haemorrhagic shock following genital bleeding without any connexion to AFE was assumed in the other three patients.

Amniotic fluid in the uterine veins was found only in the case of traumatic uterus rupture and placental separation (Fig. 8, Table 2). In five out of seven patients, lesions of the cervix with tears in the mucous membranes were found on macroscopic examination.

In 22 women who showed no clinical symptoms of AFE at all or the cause of death was in no way related to pregnancy, no components of amniotic fluid were found in the lung sections.

Discussion

Amniotic fluid embolism is a potentially devastating complication of pregnancy that often results in poor obstetric outcome, which in turn is often equated with malpractice especially by relatives. In a publication "Medico-legal nightmare: a tragic case, a needless trial" a Canadian anaesthetist gives an impressive example of "medical experts", who without having any experience with AFE caused an absurd legal battle over 4 years, accusing several doctors of substandard care [17].

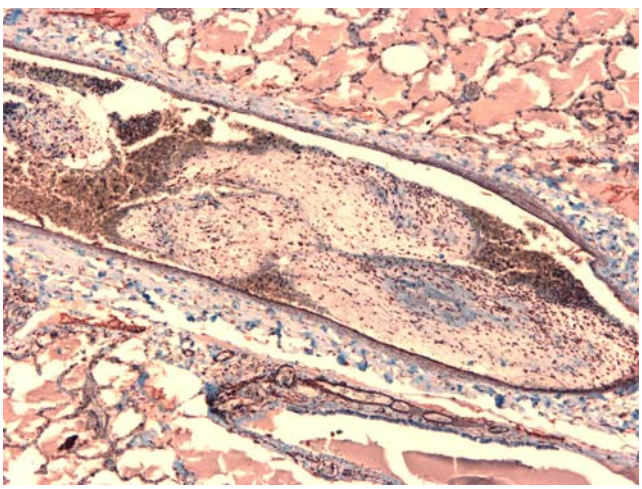

Fig. 2 Amniotic fluid embolism consisting largely of mucin within a pulmonary artery. Vimentin, ×40

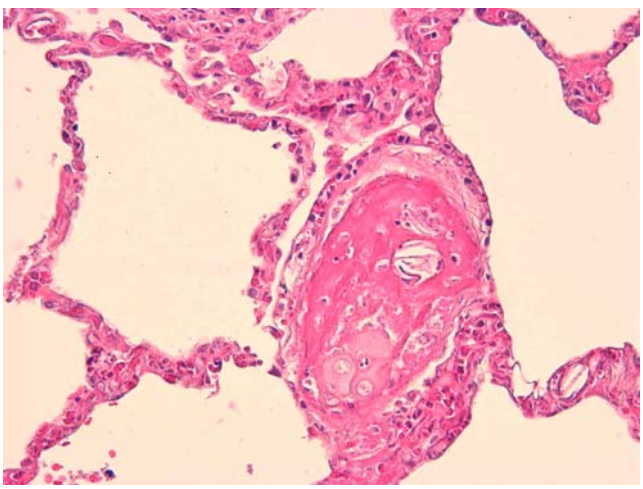

Fig. 4 Squamous cells and decidual cells embedded in a microthrombus. Pulmonary artery (200x, H&E)

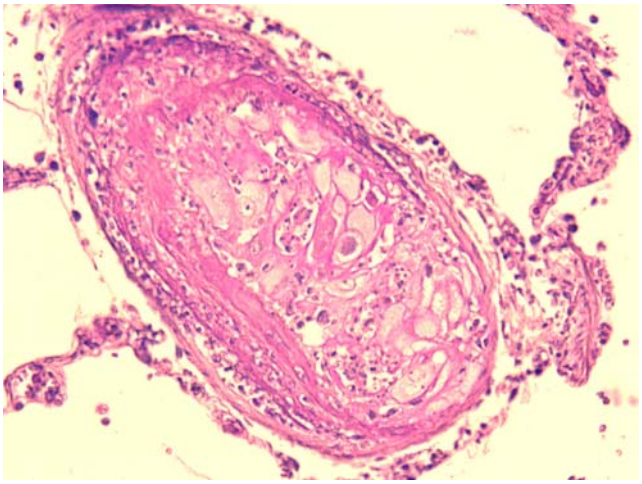

Fig. 5 Decidual cells in a pulmonary artery. Note infiltration of the vessel wall by granulocytes. Survival interval 12 h. H&E, ×40

The diagnosis of AFE can only be made by means of histological examination, which is usually post-mortem, because, in the living, AFE remains a diagnosis of exclusion dependent on rapid bedside evaluation and judgment. Promising results in the diagnosis of AFE could be achieved by the measurement of serum mast cell tryptase which is elevated in some cases of AFE [10, 18]. Fineschi et al. [19] demonstrated an increase of the number of pulmonary mast cells in post-mortem cases of AFE. These findings indicate an anaphylactic reaction to foetal antigens and could explain a fatal course in cases with a minimal extent of AFE in which a physical block of the pulmonary vasculature cannot be assumed.

The correct diagnosis may present particular difficulties to the forensic pathologist as deaths occur in hospital patients who, following the acute catastrophe, may have been maintained on a life support system for a considerable time. On post-mortem examination, the original pathology has then often resolved or altered.

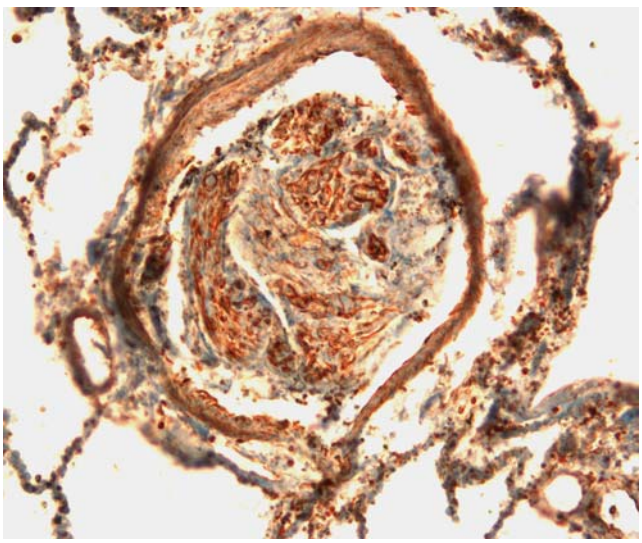

Fig. 6 Chorionic villi within pulmonary artery. Vimentin, ×40

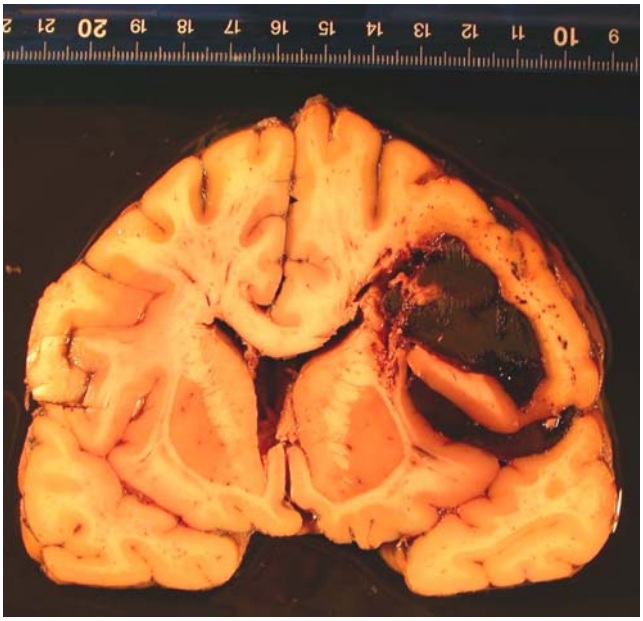

Fig. 7 Expansive subarachnoid haemorrhage in the left fissura sylvii and also expansive haemorrhage in the left fronto-parietal centrum semiovale

In the present study, a post-mortem examination was performed on 29 women who died intrapartum or post-partum. Upon histological examination, moderate or massive AFE was found in seven patients (25%). As some authors assume a physiologic nature of AFE, the medical charts as well as investigation files were studied but the clinical course left no doubt in the diagnosis of fatal AFE

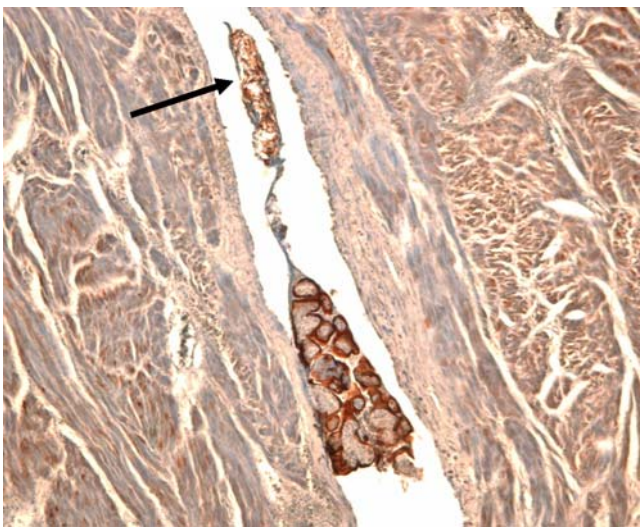

Fig. 8 Traumatic uterus rupture and placental separation: chorionic villi and amniotic fluid elements (*arrow*) in a large uterine vein (×40, pan-cytokeratin)

[7, 16]. Most of the patients presented with uncontrollable genital bleeding as a first clinical sign of AFE. The longest detection interval of pulmonary AFE was 36 h. In eight women who died as a consequence of haemorrhagic shock, elements of amniotic fluid were not found, but in patients who die many days or even weeks after delivery as a consequence of early post-partum haemorrhagic shock AFE should be considered as the cause of an underlying coagulopathy.

The components of amniotic fluid could be identified in routine haematoxylin-and-eosin-stained sections. However, the results of immunohistochemistry showed quite plainly that they might also be easily overlooked. In particular, small emboli require careful searching of sections stained with special techniques [20]. In our opinion, the use of immunohistochemistry permits a more reliable assessment of the dimension of AFE.

The mean survival time in AFE cases in our study was 12 h (minimum 15 min, maximum 36 h, median 9.5 h). The survival rate was similar to that reported by Högberg and Joelsson [21], who determined a survival interval of 12 h. There was no correlation between the survival time and the extent of the AFE. Fatal AFE is most commonly associated with relatively small tears in the uterus, cervix or vagina which have not totally disrupted the wall [11]. The histological visualisation of amniotic fluid debris in the uterine veins was only seldom successful [22–24] although it is obvious that amniotic fluid can gain entry into the maternal circulation only via uterine veins. In our study, the components of AF, decidual cells, as well as whole chorionic villi, were only detected in the uterine veins and in the pulmonary circulation in a woman with traumatic uterine rupture and placental separation [24, 25].

Throughout the literature, there are also references to the concept that amniotic fluid routinely enters the maternal circulation at the time point of delivery. This concept is based on the detection of AF components in blood samples from pulmonary arteries [14, 15]. These results have been never confirmed by a histological examination of lung sections. In our study, squamous cells, mucin or lanugo hairs were not found in any of the tissue sections of 22 cases of maternal death other than AFE or in deaths in no way related to pregnancy. Similar results were achieved by Roche and Norris, who compared lung specimens from 20 toxaemic patients with an equal number of patients with clinical evidence of AFE [26]. None of the sections from the toxaemic patients stained positive for the components of amniotic fluid. Using immunohistochemical methods, which allow a more precise search for amniotic fluid debris, we could confirm the statement of Roche and Norris nearly 35 years later. Thus, there is no conclusive evidence to support the suggestion that AFE is a common physiological occurrence.

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