Int J Legal Med (2008) 122:157–161
DOI 10.1007/s00414-007-0185-3

ORIGINAL ARTICLE

Clotted blood as sign of alcohol intoxication: a retrospective study

T. Fracasso · B. Brinkmann · J. Beike · H. Pfeiffer

Received: 20 December 2006 / Accepted: 13 June 2007 / Published online: 19 July 2007

Abstract A total of 138 autopsies performed at the Institute of Legal Medicine of the University of Münster between 1994 and 2006 were subdivided into two groups: (1) 69 asphyxial deaths with a blood alcohol level (BAL)>1‰ and (2) 69 asphyxial deaths with a BAL of 0.00‰. The coagulation state in the central vessels was registered in all cases as fluid, compactly clotted or loosely clotted, and the post-mortem interval was recorded. Histology investigations were performed on the liver to analyze the incidence of hepatic fibrosis/cirrhosis. Fisher's exact test was performed to check for statistical significance. The blood was found to be clotted in 49.3% of the cases of group (1) and in 5.8% of group (2) ($p<0.01$). The post-mortem interval did not have any influence on the coagulation state as observed in both groups. Liver fibrosis/cirrhosis was a rare finding detected in three cases in group 1 and in two cases in the control group 2 and, therefore, not relative to our observations. A distinctly positive BAL is often associated with heavy stages of blood coagulation as observed during autopsy. Distinctly positive alcohol concentrations have an influence on the fibrinolytic process and, hence, on the coagulation status.

Keywords Post-mortem blood · Clotted blood · Alcohol intoxication

T. Fracasso (✉) · B. Brinkmann · J. Beike · H. Pfeiffer
Institute of Legal Medicine, University of Münster,
Röntgenstraße 23,
48149 Münster, Germany
e-mail: Tony.Fracasso@ukmuenster.de

Introduction

The pathophysiology of alcohol intoxication is a classical topic in legal medicine that still interests many examiners [1–4]. Unfortunately, the post-mortem findings are not characterised by any specific features.

In some recent autopsy cases in which the cause of death was acute alcohol intoxication, we observed an unusual degree of clotted blood in the central vessels and in the heart. The textbooks of legal medicine do not really deal with the post-mortem blood clotting in relation to alcohol intoxication. Whilst several authors have reported that the blood remains fluid [5–7], others have discussed an influence of alcohol on coagulation [8–10].

We have, therefore, re-examined cases of asphyxial death with and without BAL to investigate this issue.

Materials and methods

The material consisted of medico-legal autopsies performed between 1994 and 2006 in the Institute of Legal Medicine of the University of Münster. In a first approach, all asphyxial deaths, i.e., by strangulation, hanging and drowning were extracted. In a second approach, all asphyxiation deaths with visible signs of putrefaction and those with BAL>0<1‰ were sorted out. Because the BAL-positive group consisted of 69 cases, we have randomly attributed and composed a comparable number in the BAL negative control group, aiming at an ideal match according to gender and ages. In each case, a complete autopsy was performed together with a full histology; toxicology screening was performed if there existed any indication and BAL was determined from

blood of the femoral veins. All autopsy records contained a statement regarding the coagulation state.

The cases (Table 1) were divided into two groups that were matched for gender and age as follows:

- Group 1: asphyxial deaths with BAL>1‰ (N=69, 18 females, 51 males; mean age, 46.5 years); in all cases, the cause of death was asphyxia. The minimum BAL was 1.04‰, the highest 4.11‰ with a mean value of 2.37‰.
- Group 2: asphyxial deaths not associated with positive BAL (BAL=0.00‰; N=69, 18 females, 51 males; mean age, 44.6 years).

The coagulation state of the blood in the central vessels and the heart was described as:

- Fluid: If the blood was completely fluid or if only a few millilitres were coagulated;
- Compactly clotted: If a compact clot with a considerable consistency filled the heart and the central vessels and only a small quantity of blood remained fluid (Fig. 1);
- Loosely clotted: if a considerable proportion of the blood was loosely coagulated and mixed with an equal proportion of fluid blood.

Histological sections of the liver were stained with H&E. The fibrosis of the liver was classified according to the Metavir classification [11]. Advanced bridging fibrosis or cirrhosis (Metavir grades F3 and F4) were considered reliable markers of hepatic dysfunction.

Information about the post-mortem interval (PMI) was available in 50 cases of group 1 and in 60 of group 2.

Fisher's exact test was performed for statistical analysis.

Results

In approximately 50% of the asphyxial deaths of group 1, considerable degrees of blood coagulation were observed, whilst the corresponding percentage in the control group was 6% (Table 2). The differences are highly significant (p<0.01).

In group 1 the mean BAL for females was 2.58‰ and for males 2.44‰. Compactly or loosely coagulated blood was found more frequently in males (55%) than in females (33%). This difference is, however, not statistically significant (p=0.12).

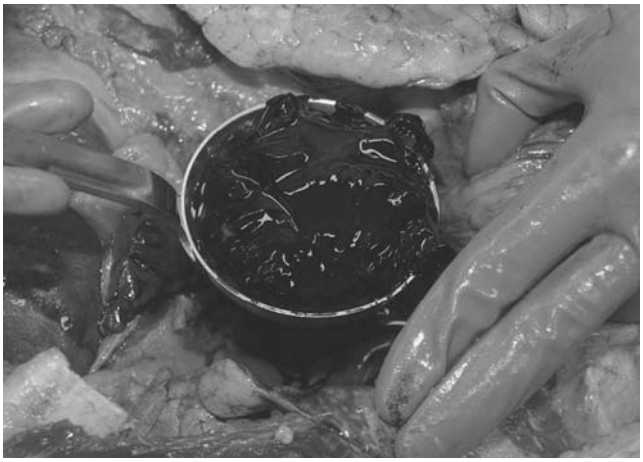

Fig. 1 Clotted blood in the heart and central vessels

In group 1, the BAL was not grossly different between the subgroups with fluid, loosely or completely coagulated blood. Also, intermediate BALs (i.e. between 1.0 and 1.99‰) were not more often associated with clotting than high (2–3‰) and very high (>3‰) BALs.

The mean PMI was 34 h in group 1 (range, 3–113 h) and 28 h in group 2 (range, 5–89 h h). The difference is not significant. The cases were further subdivided into two subgroups (<or=24 and >24 h; Table 3). There was no difference in the frequency of blood clots observed in both groups.

The histological investigation revealed strong hepatic fibrosis in three cases in the first group and in two cases in the control group. In all these cases, the blood was fluid.

Discussion

This investigation has shown that asphyxial deaths of individuals with distinctly positive BALs are much more often associated with advanced stages of blood clotting than non-alcoholised victims dying from the same causes. The ratio difference between both groups is 1:9. Moreover, 50% of asphyxial deaths of "drunken" persons are associated with advanced stages of blood clotting as observed during autopsy. Also, the most advanced stage of post-mortem blood clotting was exclusively associated with distinctly

Table 1 Causes of death in the two groups

	Hanging	Drowning	Strangulation by ligature	Manual strangulation	Choking
Group 1, BAL>1‰	16	47	2	1	3
Group 2, BAL=0‰	33	26	9	1	0

Table 2 Frequencies of clotted blood in the investigated groups

Coagulation state	Group 1, BAL>1‰	Group 2, BAL=0‰
Loosely clotted	15 (21.7%)	4 (5.8%)
Compactly clotted	19 (27.5%)	0
Fluid	35 (50.7%)	65 (94.2%)

positive BALs (n=19 cases) and never found in the control group.

Weiler et al. [12] have pointed out this phenomenon in 1980 and found similar results. However, there exists a major difference: in our study, we observed an extreme degree of coagulation (compactly clotting or "en bloc" coagulation) in 27.5% of the cases with BAL. This type was not described by Weiler et al. [12], and we cannot evaluate whether this depends on differences in the standard autopsy protocol or reflects different drinking habits. Theoretically, this can also have originated from a very different composition of the study group of Weiler at al. with regard to age and gender. Unfortunately, this possible influencing factor cannot be derived from the manuscript.

Alcohol can positively and negatively influence the process of coagulation and, hence, the coagulation status as found during autopsy.

Chronic alcohol consumption can affect coagulation by damaging the synthesising capacity of the hepatocytes, both directly and through metabolites [13–16]. The prolonged clotting times are attributed to a deficiency of vitamin K and a subsequent varying degrees of the production of coagulation factors [17]. Also acetaldehyde (ACH), the primary metabolic product of ethanol can contribute to this failure by inactivating the coagulation system [18–22] through the interaction with coagulation proteins at high concentrations [23].

More acute types of alcohol consumption seem to have an adversarial effect, especially in relation to the clinical risk for a stroke or coronary heart disease [24–27]. Heavy binge drinking results in a marked elevation of plasminogen activator inhibitor (PAI-1) followed by a transient decrease of fibrinolysis [28, 29]. This type of drinking is also associated with an increase of urinary excretion of 2.3-dinor-thromboxane B2 reflecting platelet activation [30, 31]. Also other studies have confirmed that the platelets are activated, and fibrinolysis is inhibited [32, 33].

Also alcohol-induced diuresis can play a role [34]. With increasing alcohol concentrations, alcohol-induced diuresis occurs [34] with subsequent dehydration [35] but with no marked effects on the plasma sodium and potassium concentrations [36]. A decrease in BAL is conversely accompanied by anti-diuresis [35] and an increase of water intake [36]. Moreover, recent experiments have shown that chronic alcohol consumption inhibits the diuresis [35]. We would tentatively suggest that the aforementioned effect cannot explain the extreme stages of dehydration that are sometimes found in alcoholics. Among the other factors responsible, e.g. diarrhoea, vomiting and reduced fluid intake, as well as diuresis in diabetics, need to be considered as well.

In studies performed in the post-mortem period, the time of recording the coagulation status may be important as well: according to Berg [37, 38] and Tacheiki et al. [39–41], the processes of coagulation and fibrinolysis take place in a very early post-mortem phase, i.e. the primary coagulation is followed (or not) by fibrinolysis, this process being terminated after roughly 4–8 h.

In our study, very few autopsies were performed in an early post-mortem phase, i.e. 6–8 h. Thus, the processes of coagulation and decoagulation were no longer of influence. Also the subdivision into an early (<24 h) and a late (>24 h) phase showed no influence by the PMI. Thus, the observation made could not have been influenced by the PMI.

Chronic alcohol abuse does not seem to make a major contribution via influencing the coagulation process in our study. There were only very few cases with obvious sequelae of this type of abuse. Also as these cases have shown and as expected from the literature, fluid blood status would be expected and not the converse. Alcohol-induced dehydration could of course have influenced the findings in a positive way, i.e. inducing stronger coagulation, but we would suggest that such actions are minimal if ever existing: the coagulation group under the influence of alcohol had a mean blood water content of 73.3%. The water concentration was 73.7% in the cases with complete-

Table 3 Early and late PMI in the investigated groups

	PMI< or =24 h		PMI>24 h		Average PMI	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
Loosely clotted	5	1	7	1	32.5 (N=25)	24.8 (N=2)
Compactly clotted	8	0	5	0		
Fluid	16	38	9	20	26.6 (N=25)	20 (N=58)
Total	29	39	21	21		

ly clotted blood and 73.3% in cases with fluid blood. In the control group, data were only available for three cases, and the mean water concentration was 71.3%. Moreover, there was no description of dehydration signs at autopsy. Therefore, to explain our findings, there only remain the published influences on the fibrinolysis process. In other words, the inhibition of fibrinolysis by intermediate and high alcohol concentrations is responsible for advanced degrees of post-mortem coagulation even in groups where this is normally rare or extremely rare.

Our results show that a BAL>1‰ is approximately nine times more often associated with post-mortem clotted blood in the heart and central vessels in cases of asphyxial deaths. These results are consistent with those published by Weiler et al. [12].

In conclusion, alcohol intoxication can be taken into account if clotted blood is seen during autopsy.

References

1. Schloegl H, Dresen S, Spaczynski K, Stoertzel M, Wurst FM, Weinmann W (2006) Stability of ethyl glucuronide in urine, post-mortem tissue and blood samples. Int J Legal Med 120:83–88
2. Koski A, Vuori E, Newer I (2005) Antidepressants: evaluation of fatal toxicity index and interaction with alcohol based on Finnish postmortem data. Int J Legal Med 119:244–248
3. Hoiseth G, Kristoffersen L, Larssen B, Arnestad M, Hermansen NO, Morland J (2007) In vitro formation of ethanol in autopsy samples containing fluoride ions. Int J Legal Med 121:85–89
4. Boumba VA, Ziavrou KS, Vougiouklakis T (2007) Biochemical pathways generating post-mortem volatile compounds co-detected during forensic ethanol analyses. Forensic Sci Int (in press). DOI 10.1016/j.forsciint.2007.03.018
5. Huckenbeck W, Bonte W (2003) Sektionsbefund bei Alkoholvergiftung und chronischem Missbrauch. In: Madea B, Brinkmann B (Hrsg) (ed) Handbuch Gerichtliche Medizin, Band II. Springer, Berlin Heidelberg New York, p 427
6. Mueller B (1953) Tödliche Alkoholvergiftung. In: Mueller B (Hrsg) Gerichtliche Medizin. Springer, Berlin Heidelberg New York, pp 773–774
7. Brettel H-F (1986) Die tödliche Alkoholvergiftung. In: Forster B (Hrsg) Praxis der Rechtsmedizin. Georg Thieme, Stuttgart, Germany, pp 466–467
8. Prokop O (1976) Die tödliche Alkoholvergiftung und plötzliche Todesfälle bei Alkoholikern. In: Prokop O, Göhler W (Hrsg) Forensische Medizin. Fischer, Stuttgart, Germany, pp 369–370
9. Maxeiner H (2003) Gewaltsame Erstickung. In: Madea B (ed) Praxis rechtsmedizin. Springer, Berlin Heidelberg New York, pp 147–152
10. Adebahr G (1969) Der forensische Beweiswert von Befunden an der Leiche. Beitr Gerichtl Med 25:44–50
11. Desmet VJ, Gerber M, Hoofnagle JH, Manns M, Scheuer PJ (1994) Classification of chronic hepatitis: diagnosis, grading and staging. Hepatology 19:1513–1520
12. Weiler G, Adebahr G, Klöppel A (1980) Zum diagnostischen Wert von "geronnenem" Herzblut bei akutem Erstickungstod. Z Rechtsmed 85:23–27
13. Mukamal KJ, Jadhav PP, D'Agostino RB et al (2001) Alcohol consumption and hemostatic factors: analysis of the Framingham offspring cohort. Circulation 104:1367–1373
14. Mukamal KJ, Massaro JM, Ault KA et al (2005) Alcohol consumption and platelet activation and aggregation among women and men: the Framingham offspring study. Alcohol Clin Exp Res 29:1906–1912
15. Dakeishi M, Iwata T, Ishii N, Murata K (2004) Effects of alcohol consumption on hepatocellular injury in Japanese men. Tohoku J Exp Med 202:31–39
16. Baysan O, Kaptan K, Erinç K et al (2005) Chronic heavy ethanol consumption is associated with decreased platelet aggregation in rats. Tohoku J Exp Med 206:85–90
17. Brecher AS, Adamu MT (2001) Coagulation protein function: enhancement of the anticoagulant effect of acetaldehyde by sulfated glycosaminoglycans. Dig Dis Sci 46:2033–2042
18. Basista MH, Joseph A, Smolen S, Koterba A, Brecher AS (1994) Acetaldehyde alters coagulation protein function. Dig Dis Sci 39:2421–2425
19. Brecher A, Koterba AP, Basista MH (1996) Coagulation protein function. III. Effect of acetaldehyde upon the activation of prothrombin. Alcohol 13:423–429
20. Brecher AS, Koterba AP, Basista MH (1996) Coagulation protein function. IV. Effect of acetaldehyde upon factor X and Xa, the proteins at the gateway to the common coagulation pathway. Alcohol 13:539–545
21. Brecher AS, Hellman K, Dulin C, Basista MH (1998) Coagulation protein function. V. Diminution of antithrombin III function by acetaldehyde. Dig Dis Sci 43:1746–1751
22. Sabol DA, Basista MH, Brecher AS, Haider K, Kleshinski J (1999) Coagulation protein function. VII. Diametric effects of acetaldehyde on factor VII and factor IX function. Dig Dis Sci 44:2564–2567
23. Brecher AS, Adamu (2002) MT short- and long-term effects of acetaldehyde on plasma. Alcohol 26:49–53
24. Hansagi H, Romelsjö A, Gerhardsson de Verdier M, Andreasson S, Leifman A (1995) Alcohol consumption and stroke mortality. Stroke 26:1768–1773
25. Booyse FM, Parks DA (2001) Moderate wine and alcohol consumption: beneficial effects on cardiovascular disease. Thromb Haemost 86:517–528
26. Murray RP, Connett JE, Tyas SL, Bond R, Ekuma O, Silversides CK, Barnes GE (2002) Alcohol volume, drinking pattern and cardiovascular disease morbidity and mortality: is there a U-shaped function? Am J Epidemiol 155:242–248
27. Davidson DM (1989) Cardiovascular effects of alcohol. West J Med 151:430–439
28. Numminen H, Syrjälä M, Benthin G, Kaste M, Hillbom M (2000) The effect of acute ingestion of a large dose of alcohol on the hemostatic system and its circadian variation. Stroke 31: 1269–1273
29. Sasaki A, Kurisu A, Ohno M, Ikeda Y (2001) Overweight/obesity, smoking and heavy alcohol consumption are important determinants of plasma PAI-1 levels in healthy men. Am J Med Sci 322:19–23
30. Patrono C, Ciabattoni G, Pugliese F, Pierucci A, Blair IA, FitzGerald GA (1986) Estimated rate of thoromboxane secretion into the circulation of normal humans. J Clin Invest 77:590–594
31. Catella F, FitzGerald GA (1987) Paired analysis of urinary thromboxane B2 metabolites in humans. Thromb Res 47:647–656
32. Van de Wiel A, van Golde PM, Kraaijenhagen RJ, von dem Borne PAK, Bouma BN, Hart HC (2001) Acute inhibitory effect of alcohol on fibrinolysis. Eur J Clin Invest 31:164–170
33. De Lange DW, Hijmering ML, Lorsheyd A, Scholman WLG, Kraaijenhagen RJ, Akkerman JWN, van de Wiel A (2004) Rapid

intake of alcohol (binge drinking) inhibits platelet adhesion to fibrinogen under flow. Alcohol Clin Exp Res 28:1562–1568
34. Collins GB, Brosnihan KB, Zuti RA, Messina M, Gupta MK (1992) Neuroendocrine, fluid balance, and thirst response to alcohol in alcoholics. Alcohol Clin Exp Res 16:229–233
35. Parlesak A, Pohl C, Bode JC, Bode C (2004) Water metabolism in rats subjected to chronic alcohol administration. Nephron Physiol 97:9–15
36. Marsano L, McClain CJ (1989) Effects of alcohol on electrolytes and minerals. Alcohol Health and Research World, US Government Printing Office, USA
37. Berg SP (1950) Das postmortale Verhalten des Blutes. Z Gerichtl Med 40:1–75
38. Berg S (1963) Physiologisch-chemische Befunde im Leichenblut als Ausdruck des Todesgeschehens. Z Gerichtl Med 54:136–149
39. Tacheiki S, Wakasugi C, Shikata I (1984) Fluidity of the blood after sudden death: Part I. Am J Forensic Med Pathol 5:223–227
40. Tacheiki S, Wakasugi C, Shikata I (1985) Fluidity of the blood after sudden death: Part II. Am J Forensic Med Pathol 6:25–29
41. Tacheiki S, Tokunaga I, Hayakumo K, Maeiwa M (1986) Fluidity of the blood after sudden death: Part III. Am J Forensic Med Pathol 7:35–38