

Pathogenesis of conjunctival petechiae

Gabriele Lasczkowski^{a,*}, Manfred Riße^a, Ulrike Gamerdinger^b, Günter Weiler^a

^a*Institut für Rechtsmedizin, Justus-Liebig-Universität, Frankfurter Street 58, 35392 Giessen, Germany*

^b*Institut für Pathologie, Justus-Liebig-Universität, Langhansstr. 10, 35392 Giessen, Germany*

Received 7 March 2003; accepted 9 March 2004

Available online 19 June 2004

Abstract

Conjunctival petechiae from 15 cases (cause of death: different natural and unnatural) were analysed using confocal laser scanning microscopy (CLSM) in order to visualize the kind of the damage within the vessel wall (diapedesis versus rhexis). The pathomorphological findings with multiple ruptures of vessels appearing to be filled to bursting point define the conjunctival petechiae as a rhexis-haemorrhage.

© 2004 Elsevier Ireland Ltd. All rights reserved.

Keywords: Conjunctival petechiae; Pathogenesis; Confocal laser scanning microscopy; Rhexis

1. Introduction

The occurrence of petechial haemorrhaging in the conjunctiva can be observed in cases of external suffocation as well as in deaths due to other unnatural causes (e.g. electrocution, cranio cerebral trauma, intoxication) or natural (e.g. cardiac, CNS, infectious) causes. They may also occur in situations of altered intra-thoracic or -abdominal pressure resulting in inflow congestion and increased pressure to veins and capillaries of the head and neck region (e.g. epileptic fit, asthma attack, sneezing, coughing, vomiting, giving birth, valsalva) [1]. Previously, Tardieu classed these petechiae as haemorrhages resulting from asphyxiation. From a pathogenetic perspective, the causes are now regarded as being purely mechanical as well as bi-/multi-factorial. The purely mechanical explanation sees the petechiae as the result of elevated venous/capillary pressure [2]. Bschor [2] suggests that the development of conjunctival haemorrhages occurs after at least 10–20 s of complete venous compression, i.e. blockage. Other authors propose that it would take several minutes but in a “Perthes-blockage”, they may form within seconds or milliseconds (literature in [3]). Multi-causal explanations include elevated

intra-vessel pressures and other factors such as age, constitution, cardiac weight [4,5] and damage to capillary walls stemming from hypoxia (literature in [6]). As a result of prone position and/or the head lying lower than the rest of the body, petechiae may also develop post mortem [7]. Lignitz et al. [8] report the occurrence of petechial haemorrhages after death, which originated from the effects of exposure to heat. After critical evaluation of several publications (e.g. [9]), it was concluded that resuscitation attempts alone do not lead to the formation of such haemorrhages. Henn et al. [10] tried to explain the pathogenesis of such petechiae using the technique of serial histological sections, but the pathomorphological changes, the bleeding source within the vessel wall, could not be made visible with conventional light microscopy. As there was no evidence for rupturing and because they thought that erythrocytes had been caught whilst passing through the wall of the blood vessels, they assumed they were dealing with a haemorrhage related to diapedesis. Using a confocal laser scanning microscope (CLSM), we tried to establish whether conjunctival petechiae had been caused by diapedesis or by rhexis haemorrhaging.

2. Materials and methods

Conjunctival petechiae from 15 corpses where death had occurred from different causes, either natural or unnatural,

* Corresponding author. Tel.: +49 641 99 41412;

fax: +49 641 99 41419.

E-mail address: gabriele.lasczkowski@forens.med.uni-giessen.de (G. Lasczkowski).

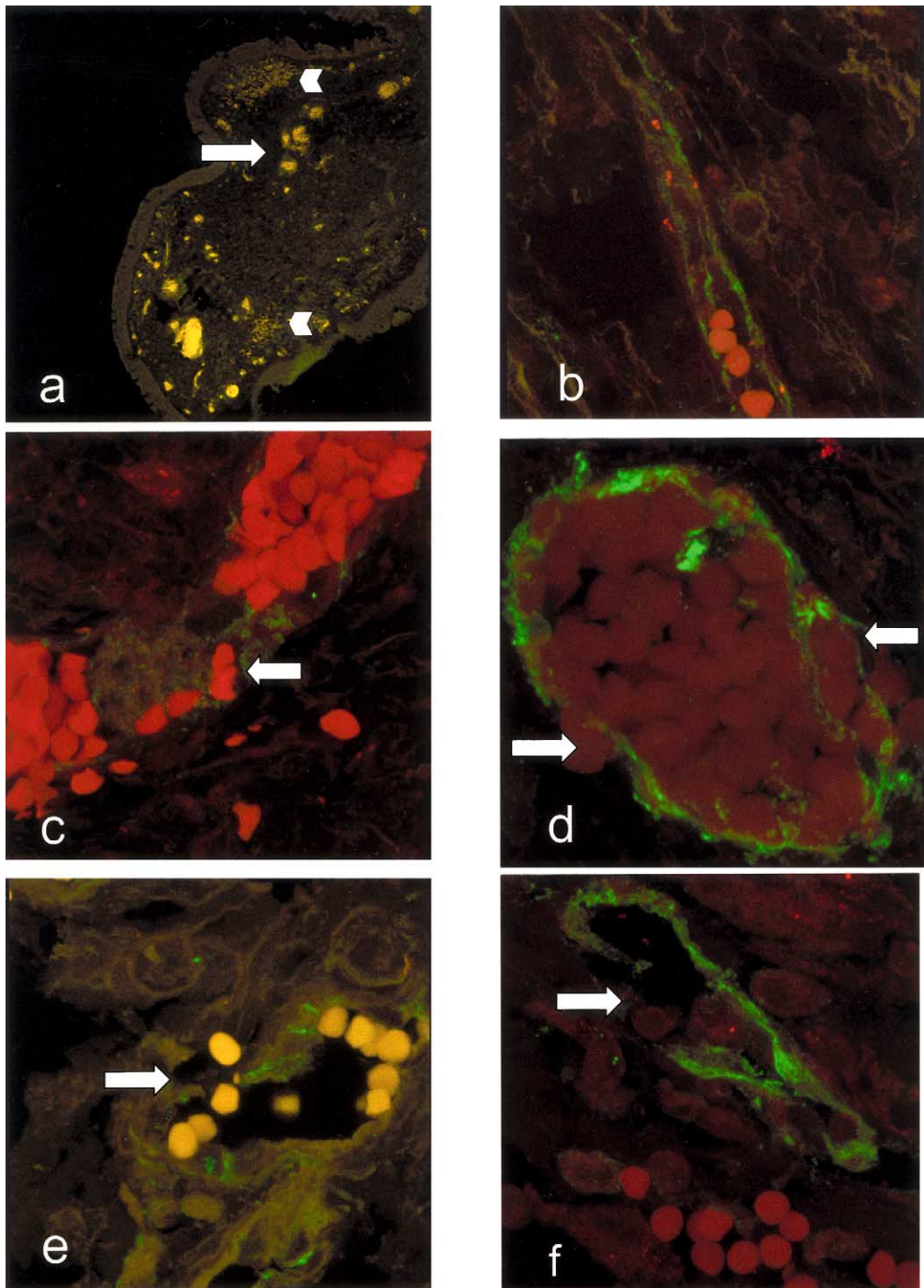


Fig. 1. Projection images (overlayed series of optical sections) of conjunctival petechiae. CLSM (generates optical sections of a defined plane in a sample by collecting light from this specified single plane). (a) General sight of conjunctival petechiae. Dilation and filling of terminal vessels (▶), blood extravasation (◊). A 78-year-old female died of compression of trachea in helpless position: HL PL FLUOTAR, 10.0×0.30 ; zoom, 1; image size, about $100,000,000 \mu\text{m} \times 100,000,000 \mu\text{m} \times 9.77 \mu\text{m}$; step size, about $0.46 \mu\text{m}$; sections, 21. (b) Conjunctiva, capillary vessel ($8, 26 \mu\text{m}$), normal finding. An 82-year-old woman died of multiple trauma: PL APO, 100.0×1.40 OIL; zoom, about 1.48; image size, about $67.70 \mu\text{m} \times 67.70 \mu\text{m} \times 6.19 \mu\text{m}$; step size, about $0.16 \mu\text{m}$; sections, 39. (c) Terminal vessel, loop ($25, 79 \mu\text{m}$); congestion; rupture with extravasation of erythrocytes (▶). A 78-year-old female died of compression of trachea in helpless position: PL APO, 100.0×1.40 OIL; zoom, about 1.40; image size, about $71.36 \mu\text{m} \times 71.36 \mu\text{m} \times 9.77 \mu\text{m}$; step size, about $0.16 \mu\text{m}$; sections, 49. (d) Terminal vessel,

Table 1
Material analysed ($n = 15$)

Cause of death	Age (years)/sex	Resuscitation	Time elapsed before autopsy	Position
Coronarism; aspiration	52/male	None	21 h	Back
Sudden death/heart failure	49/male	Applied	18 h	Back
Myocarditis	32/male	None	46 h	Back
Global cardiovascular arrest	38/male	Applied	41 h	Back
Myocarditis	22/male	None	41 h	Back
Coronary thrombosis	61/male	None	6 days	Back
Pericardial tamponade/myocardial infarction	49/male	Applied	52 h	Back
Pericardial tamponade; ruptured aorta; medionecrosis	28/pregnant female	None	34 h	Back
Epileptic fit	43/male	None	6.5 days	Back
Narcotic intoxication	26/male	None	5 days	Side
Chest injury caused by blunt force	81/male	None	27 h	Back
Compression of trachea; helpless position	78/female	None	76 h	Side
Compression of trachea; coverage of respiratory tract; poisoning (analgesics)	6/male	None	16 h	Back
Strangulation/injuries from blunt and sharp instrument	21/male	None	2 days	Front
Untypical hanging	53/male	None	6 days	Suspended, then back

were analysed using a confocal laser scanning microscope (Table 1). Macroscopically one could see rounded, clearly separated haemorrhages ranging in size from flea bites to 1 mm circular spots (“classical petechial haemorrhages”). The picture of the endothelial cells was gained using CD 31/FITC at 488 nm (em: 500–550 nm). The erythrocytes were visible due to auto-fluorescence (ex: 568 nm; em: 580–630 nm). Petechiae from deaths associated with capillary wall damage as a result of sepsis or a clotting disorder have not, as yet, been considered.

3. Results

In those cases, which showed minute haemorrhages when macroscopically analysed, we observed small erythrocyte extrusions around parts of those blood vessels that were extremely full. Indeed, the vessel wall was ruptured to an extent far exceeding that of the size of the erythrocyte. In the cases of “classical petechial haemorrhages”, we found several full capillary spaces, but also empty ones. Some of the ruptured parts of the vessel were empty. The

walls were covered in ruptures and appeared extremely thin in those areas where the vessels were filled to bursting point. All cases were linked pathomorphologically by a haemorrhage associated with the rupture of the vessel wall (Fig. 1).

The haemorrhages and erythrocyte extrusions were merely found in those regions of the vessels where the walls were of a simple structure consisting of one layer of endothelial cells and a basement membrane. The applied method allowed the display of endothelial cells. The capillary endings and the surrounding areas of the vessel were affected depending on their wall structure and size, thus showing that post-capillary venules were also involved (Fig. 2).

Damage to the blood vessels was practically the same irrespective of cause of death.

4. Discussion

The picture of conjunctival petechiae obtained using CLSM is consistent with a rhexis haemorrhage, as is

postcapillary venule (36 μm), filled to the bursting point. Rupture within vessel wall (►). Erythrocyte extravasation at its beginning. A 22-year-old male died from myocarditis; treatments included cardio-pulmonary resuscitation compressions; death occurred after lying in coma in intensive care unit for 1 week; post-mortem carried out 1.5 days later: PL APO, 100.0 \times 1.40 OIL; zoom, about 2.01; image size, about 49.8 μm \times 49.8 μm \times 8.5 μm ; step size, about 0.20 μm ; sections, 43. (e) Capillary vessel, dilatation (17, 31 μm). Rupture with extravasation of erythrocytes (►). Autolysis: 6 days elapsed before autopsy (weak staining with CD 31/FITC; swollen surrounding tissue). A 53-year-old male died of untypical hanging: PL APO, 100.0 \times 1.40 OIL; zoom, about 1.48; image size, about 67.6 μm \times 67.6 μm \times 5.21 μm ; step size, about 0.16 μm ; sections, 33. (f) Capillary vessel (12, 68 μm), ruptured wall around erythrocyte extrusion (►); 22-year-old male died from myocarditis; treatments included cardio-pulmonary resuscitation compressions; death occurred after lying in coma in intensive care unit for 1 week; post-mortem carried out 1.5 days later: PL APO, 100.0 \times 1.40 OIL; zoom, about 1.48; image size, about 67.65 μm \times 67.65 μm \times 5.49 μm ; step size, about 0.20 μm ; sections, 28.

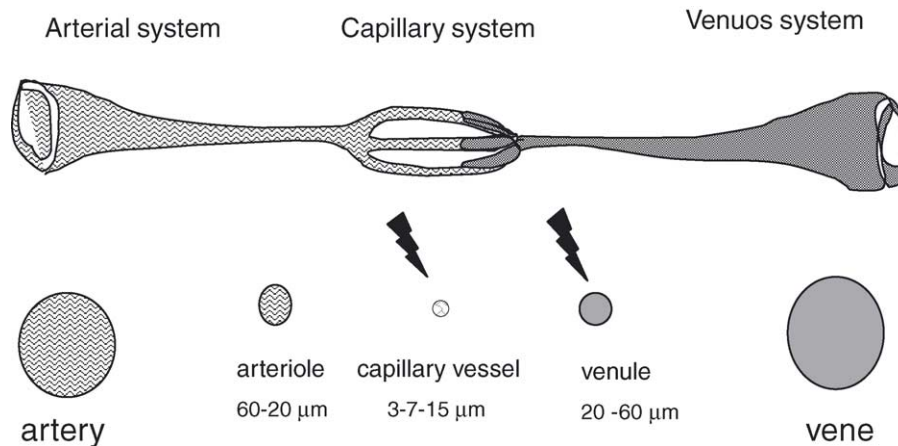


Fig. 2. Localisation of blood vessel damage.

generally suspected [6]. As opposed to a diapedesis-related haemorrhage, a rhexis haemorrhage is caused by damage to the vessel wall by a mechanical force disrupting the wall coherence. A haemorrhage caused by diapedesis can be explained by clotting problems, circulatory disorders or damage to the wall due to toxic substances or hypoxia [7]. This type of haemorrhage involves the movement of blood components through preformed pores and gaps formed by parting endothelial cells.

In the sample analysed, all the pathomechanisms associated with a venous congestion were to be found; this was demonstrated by a massive dilation and huge amount of blood in the terminal vessels, as shown in the histological pictures from the petechiae. Haemorrhages and erythrocyte extrusions were merely found near vessels with weak walls. Those parts of the vessel with walls formed by several layers were not affected. Therefore, the thickness, i.e. structure of the blood vessels is a factor accounting for the pathogenesis of such haemorrhages. Within and around the haemorrhages, we observed vessels with ruptured walls filled to bursting point but also empty capillary spaces. One must bear in mind that when the endothelial cells are damaged, vaso-active substances are released, which cause slowing or inhibition of the blood flow. This means that an anterograde influx into the vessels does not occur. On the other hand, damage to the terminal vessels results in blood flowing into the loose conjunctival connective tissue. Finally, one should bear in mind the general final collapse of the cardio-vascular system, from which follows the inhibition of the blood flow back to the arteries.

The conjunctiva consists of loose connective tissue within which lie single or clustered white blood cells. The structure of this tissue means it cannot withstand elevated pressure within the blood vessel, and it cannot give a weak vessel wall external support. This may explain why petechiae are first formed in the loose parts of the

conjunctivae (i.e. outside those parts which are connected to the tarsus) and why one cannot find petechiae in cases of drowning in very deep water, where the external pressure is too high for them to be formed [2]. Thus, the second main factor to consider is the structure of the tissue around the blood vessels.

Elevated pressure in the veins and capillaries, continued arterial blood flow and the structural nature of the conjunctiva and the affected parts of blood vessels could be used to explain the observed micro-ruptures and conjunctival petechiae as mechanically caused rhexis haemorrhages. This is confirmed by the fact that such haemorrhages may arise within seconds or minutes, or in extreme cases even in the fraction of a second.

Further studies should concentrate on the exact localisation of the blood vessel damage under specific conditions of conjunctival circulation. They should also investigate the differences in size and distribution of petechiae as well as markers associated with hypoxia and parameters of wound healing.

References

- [1] O. Prokop, R. Wabnitz, Vorkommen von Bindehautblutungen bei Lebenden und Toten, dargestellt in 10 tabellen, *Z Rechtsmedizin, J. Legal Med.* 67 (1970) 249–257.
- [2] F. Bschor, Beurteilung von Stauungsblutaustritten im Kopfbereich bei Strangulation und anderen Todesursachen, *Beitr. Gerichtl. Med.* 25 (1969) 146–152.
- [3] B. Madea, W. Grellner, Vitale Reaktionen I, *Rechtsmedizin* 12 (2002) 378–394.
- [4] H. Maxeiner, Über Kopfstauung, Petechien und “Zyanose” beim Tod durch homizidale Halskompression, *Rechtsmedizin* 7 (1997) 37–44.
- [5] H. Maxeiner, A. Winkelhofer, Petechiale Lidhaut und Konjunktivalblutungen bei verschiedenen Todesursachen—

- Häufigkeiten und Einflußfaktoren, *Rechtsmedizin* 10 (1999) 7–13.
- [6] S.F. Ely, C.S. Hirsch, Asphyxial deaths and petechiae: a review, *J. Forensic Sci.* 45 (2000) 1274–1277.
- [7] W. Jansssen, *Forensische Histologie*, in: H.E. Weinig, S. Berg (Eds.), *Arbeitsmethoden der Medizinischen und Naturwissenschaftlichen Kriminalistik*, Band 10, Verlag Max Schmidt, Römhild, Lübeck, 1977.
- [8] E. Lignitz, O. Prokopp, H. Strauch, Hitzebedingte postmortale Blutaustritte der Lidbindehäute, *Jpn. J. Legal Med.* 35 (1981) 79–81.
- [9] I. Hood, D. Ryan, W.U. Spitz, Resuscitation and petechiae, *Am. J. Forensic Med. Pathol.* 9 (1988) 35–37.
- [10] R.H.E. Henn, E. Liebhardt, W. Haug, Serienschittuntersuchungen an Stauungsblutungen, *Beitr. Gerichtl. Med.* 30 (1973) 146–147.