Z Rechtsmed (1988) 100:265–270

Zeitschrift für
Rechtsmedizin

Kasuistiken / Casuistics

A Delayed Drowning Death with Histological Findings of Shock

N. Ikeda, K. Umetsu, H. Takahashi, and T. Suzuki

Department of Forensic Medicine, Yamagata University School of Medicine, Yamagata 990-23, Japan

Summary. A delayed drowning death case with histological findings of shock was described. The person was sustained by continuous positive-pressure respiration and died 2 days after resuscitation from drowning. The histological findings were intravascular microthrombi, hyaline bodies and fibrin thrombi in the brain, multiple megakaryocytes in the pulmonary capillaries, hyaline membranes of the lung, multiple small hyaline bodies in the liver sinusoids, and erosion of the mucous membrane of the stomach as well as histological findings of shock kidney. Drowning and systemic hypotension during resuscitation seemed to cause irreversible oxygen debt of the organs and the tissues to lead to shock.

Key words: Drowning, delayed death – Delayed death, drowning – Drowning, shock

Zusammenfassung. Spättod nach Ertrinken mit histologischen Befunden von Schock. Der Patient wurde ununterbrochen bei Positiv-Druck beatmet und starb 2 Tage nach Rettung vom Ertrinken. Histologisch wurden sowohl intravaskuläre Mikrothromben, hyaline Körper und Fibrinthromben im Gehirn, Megakaryozyten in den Lungenkapillaren und hyaline Membranen in der Lunge, multiple hyaline Körper in den Lebersinusoiden und Erosionen der Magenschleimhaut als auch die histologischen Befunde einer Schock-Niere gesehen. Ertrinken und systemischer Blutdruckverlust während der Wiederbelebung haben einen unersetzbaren Sauerstoffmangel in Organen und Geweben verursacht, der in den Schockzustand führte.

Schlüsselwörter: Ertrinken, Spättod – Spättod, Ertrinken – Ertrinken, Schock

Introduction

State of shock is usually caused by diverse injuries, such as traumatization, burns, blood loss, intoxication, and infection. The essential feature in the

Offprint requests to: T. Suzuki

pathogenesis of shock, however, is the reduction of blood flow with reduced supply of oxygen of tissues to the point where metabolic needs are no longer met [1, 2]. The state of shock may therefore occur if the anoxic state of the tissue is permitted to continue regardless of its cause.

In the present paper we report a delayed drowning death in which histological findings of shock were pronounced in varying degrees in brain, lung, liver, and stomach as well as kidney.

Case

A boy aged 13 years, swimming with his friends in a river, suddenly sank into the water. He was found several minutes later and was promptly removed by his friends. He was apneic and comatose when rescued. He was administered immediate artificial respiration and cardiac massage. Much water was noted to run from his mouth. A rescue team was summoned and he received treatment in an respirator and was transported to a hospital.

On admission to the hospital, he was deeply comatose. No respiratory movements occurred when the patient was disconnected from the mechanical ventilator. The pupils were fixed in diameter and did not respond to sharp change in the intensity of incident light. No motor responses could be elicited by adequate stimulation of any somatic area. There was no reflex response to bronchial stimulation by a suction catheter passed down the trachea. A large amount of water was removed by tracheal suction. His life was sustained for 2 days by continuous positive pressure respiration until his death. During the positive pressure respiration, his tension returned to normal but he was oliguric, and blood urea nitrogen rose steadily.

Autopsy Findings

The decedent was 152 cm tall and weighed 42 kg. Examination of the external surface revealed no evidence of blunt force trauma. The brain was swollen, edematous, and weighed 1700 g. The brain gyri were large, and their crests were flat with compression of the sulci. The sectioned surface was pale, dry, and bulged slightly. On section, there were small petechiae in the superficial layers of cerebral cortex, most marked in the parietal cortex. Petechiae were present in the pericardium. Both lungs were grossly edematous and weighed 915 g each. The general appearance of the lungs was pallor, but there was mottling with red areas amongst those of grey color. Some rather large hemorrhages were seen immediately beneath the pleurae. The kidneys were of normal size. On section, the renal cortex was pale and mottled, and in the deep parts, the medullary pyramids were darkly congested. The mucosa of the stomach was congested and edematous, and contained multiple small hemorrhagic erosions. No important change was seen in the other organs.

Histological Findings

Kidneys. Marked dilation of the capsules of Bowman and collapse, swellings of endothelial and surface cells, broadening of the basal membrane and impairment of the loops were found in the glomeruli. Masses of granular material, somewhat resembling dead cell bodies occurred in many of the capsules of Bowman. Marked dilatation of the tubuli with flattening and necrosis of the epi-

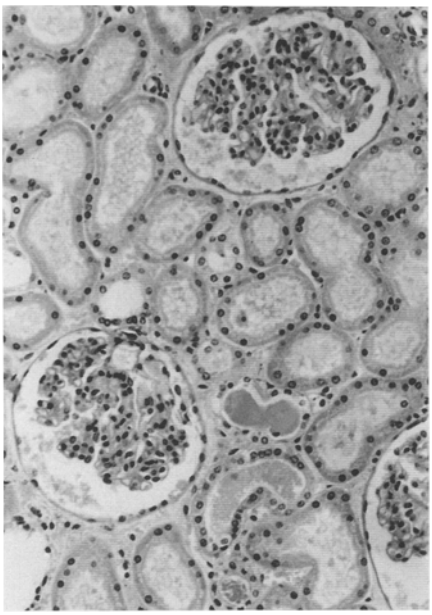

Fig. 1. Kidney with marked dilatation of the capsules of Bowman and the contorted tubules, flattening and necrosis of the tubular epithelia. Masses of granular material are seen in the capsules of Bowman. Distal tubules contain fine granular casts. × 240

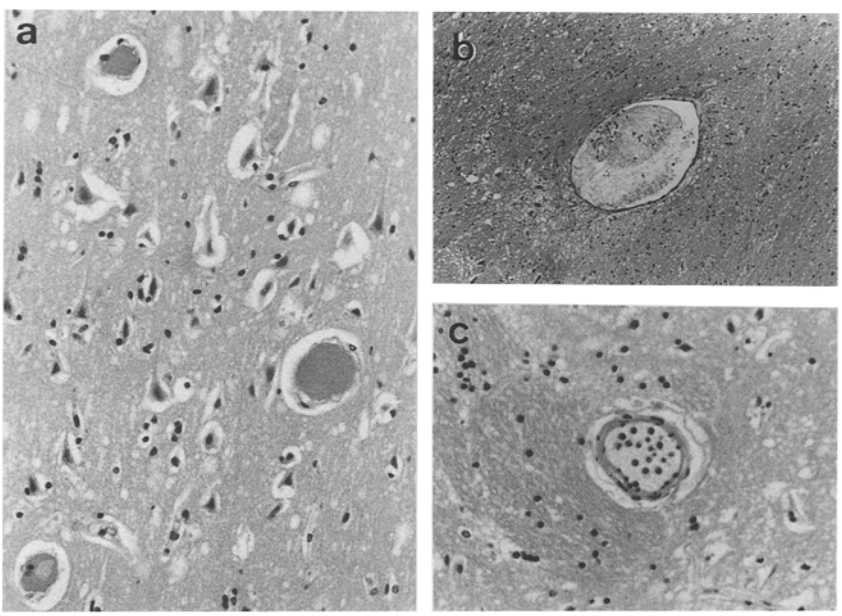

Fig. 2. a Dilated cerebral capillaries with hyaline body. Perivascular edema and swelling of the endothelial cells are noted; × 210. **b** Dilated cerebral arteriole with fibrin thrombus; × 76. **c** Cerebral capillary with microthrombus. Perivascular edema and swelling of endothelial cells are noted; × 190

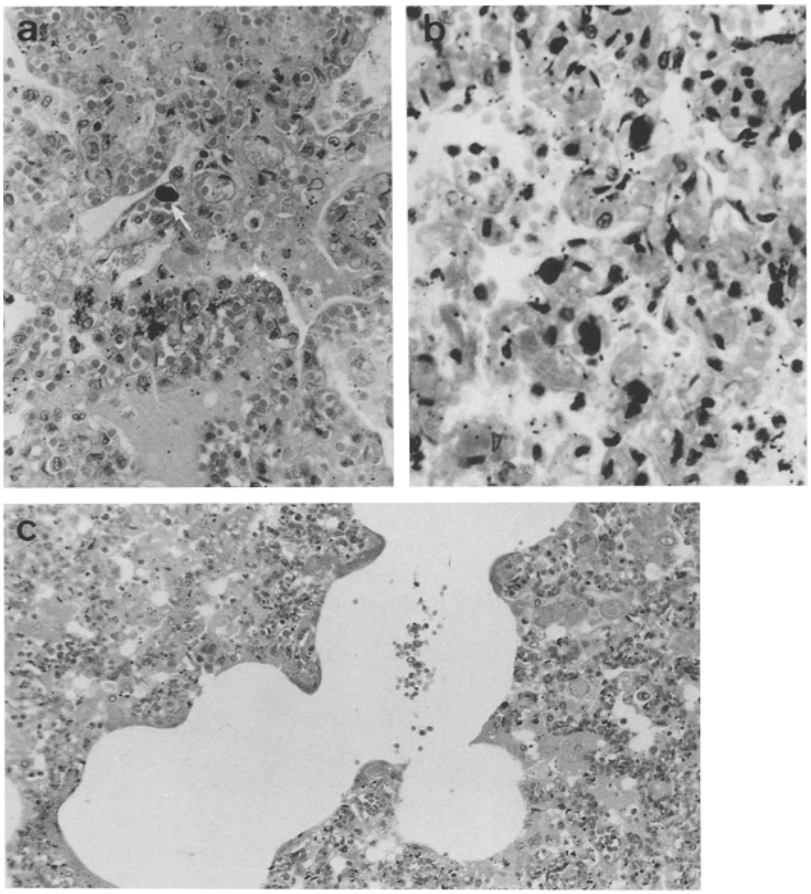

Fig. 3. **a** Pronounced interstitial and intra-alveolar edema with high-grade vascular congestion. A megakaryocyte (↑) is noted; × 143. **b** Atelectasis and multiple megakaryocytes in the pulmonary capillaries of the alveolar septa; × 240. **c** Hyaline membrane of the alveolar wall; × 95

thelia was seen. Fine granular reddish or brownish casts were seen in the distal tubules or collecting ducts. Interstitial edema separating the tubules was not prominent (Fig. 1).

Brain. Microthrombi, hyaline bodies, and fibrin thrombi were seen in the dilated arterioles and capillaries. The perivascular spaces were enlarged and swelling of the capillary endothelial cells were noted (Fig. 2).

Lungs. A pronounced interstitial and intra-alveolar edema occurred with widening of the inter-alveolar connective tissue. There was high-grade vascular congestion, circumscribed escape of erythrocytes, and massive leukostasis of polymorphonuclear granulocytes in the pulmonary circulation. Evidences of atelectasis and multiple megakaryocytes with a naked nucleus in the capillaries of the alveolar septa were seen. The hyaline membranes were formed on the surface of alveolar wall (Fig. 3).

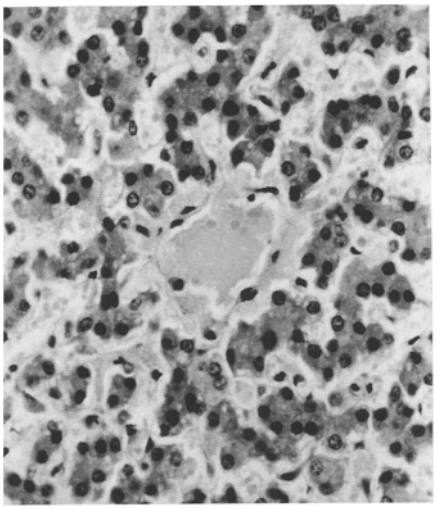

Fig. 4. Liver with severely dilated central acinal sinusoid with multiple small shock bodies. Necroses of some parenchymal cells are seen; × 266

Liver. Multiple small hyaline bodies were found in the dilated central acinal sinusoids. The cell necroses occurred in a few parenchymal cells (Fig. 4).

Stomach. The mucosa of stomach showed shock erosions with hemorrhages and fibrin precipitation. They were circumscribed and sharply demarcated from their surroundings.

Discussion

Irreversible renal injury leading to anuria and death as a complication of secondary shock has attracted general attention since the description by Bywaters and Beall [3] and Maitland [4]. It was believed at first that toxic agents from damaged skeletal muscle were responsible for producing selective damage in the renal tubular segments [5]. Subsequent studies, however, indicated that the essential damage to the kidney in such circumstances was probably attributable primarily and principally to the renal ischemia that occurs during the post-traumatic episode of systemic hypotension [1]. Any injury that is followed by secondary shock is capable of causing renal damage if the state of shock is sufficiently severe or protracted. Eeles and Sevitt [6] found microthrombi in the lungs and kidneys of injured and burned subjects, and postulated that microthrombosis might mediate irreversible shock in injured and burned patients. If the reduction of blood flow with reduced delivery of oxygen to the tissue to arise anoxic state is permitted to continue, it aggravates the circulatory deficiency and causes progress to a vicious cycle. Ikeda et al. [7] observed the typical findings of shock in the vulnerable viscera of the rabbits led to death by tourniquet shock without crushing of the skeletal muscle. They considered that the hypoxic metabolites derived from the distal limb to the tourniquet location caused impairment of microcirculation, alteration of blood coagulability, and

disturbance of the tissue oxygen supply, and these pathologic conditions formed a vicious cycle to lead to shock.

Fuller [8] studied the autopsy material from 20 delayed drowning deaths, and indicated that although they had aspirated significant amounts of water, profound blood volume changes, and electrolyte disturbances were corrected too rapidly to be detected clinically. He found acute renal tubular necrosis in one of these patients who had had a 12-h period of hypotension to shock levels while on positive-pressure breathing and died 3 days after immersion, and he suggested that the hypotensive shock and hypoxia secondary to the injury to the lung parenchyma damage the vulnerable viscera including kidney. However, any histological investigation of vulnerable viscera other than kidney was not carried out.

The histological findings that were determined in our delayed drowning death case were microthrombi, hyaline bodies (shock bodies), and fibrin thrombi in the dilated arterioles and capillaries of the brain, multiple megakaryocytes in the pulmonary capillaries, typical hyaline membranes of the alveolar wall intravascular hyaline bodies in the dilated central acinal sinusoid and hepatic cell necrosis, acute erosions of the mucous membrane of the stomach as well as histological findings of shock kidney.

Northway et al. [9] pointed out the connections between hyaline membranes and changes in the lungs following artificial respiration. Typical hyaline membranes, however, have been detected in the lungs of adult following variously induced states of shock [10].

These histological findings of shock of the present delayed drowning death case seemed to be caused by severe tissue hypoxia following drowning and systemic hypotension during resuscitation.

References

1. Anderson WAD, Kissane JM (1977) Pathology, 7th edn. Mosby, Saint Louis, pp 218–219
2. Janssen W (1984) Forensic histopathology. Springer, Berlin Heidelberg New York Tokyo, pp 156–173
3. Bywaters EGL, Beall D (1941) Crush injuries with impairment of renal function. Br Med J 1:427–432
4. Maitland AIL (1941) Case of crush injury with recovery. Lancet 1:446–448
5. Dunn JS, Gillespie M, Niven JSF (1941) Renal lesions in two cases of crush syndrome. Lancet 2:549–552
6. Eeles GH, Sevitt S (1967) Microthombi in injured and burned patients. J Pathol Bacteriol 93:275–293
7. Ikeda N, Takahashi H, Umetsu K, Suzuki T (1987) Histopathological studies on tourniquet shock death. Jap J Legal Med 41:15–20
8. Fuller RH (1963) The clinical pathology of human near-drowning. Proc R Soc Med 56: 33–38
9. Northway WH, Jr, Rosan RC, Porter DY (1967) Pulmonary disease following respirator therapy of hyaline membrane disease. Bronchopulmonary dysplasia. New Engl J Med 276:357–368
10. Bleyl U (1970) Vergleichende Untersuchungen an Erwachsenen und Neugeborenen zur Entstehung pulmonaler hyaliner Membranen. Verh Dtsch Ges Pathol 54:340–348

Received October 15, 1987