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A Dialysis Patient Died from A Subdural Hematoma After a Traffic Accident

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Abstract

Hemodialysis (HD) is a common treatment method for maintaining the life of some patients with uremia, renal failure, etc. At present, with the increase in the number of patients undergoing long-term dialysis, it is inevitable that they will be injured due to accidents and events in production and life. In addition, medical technologies such as hemodialysis have certain risks. Patients may be intertwined with complications, trauma, underlying diseases and other factors, which can easily lead to liability disputes between the parties. For such cases, forensic workers should comprehensively consider the case, analyze the relationship between disease, injury and death, and make scientific and reasonable judgments based on sufficient materials. This article summarizes and reports a case in which a dialysis patient died of subdural hematoma (SDH) and brain herniation after a traffic accident. The causal relationship between traffic accident trauma, disease, hemodialysis and SDH is comprehensively analyzed, providing a reference for forensic identification of such cases.

Keywords

Subdural hematoma; Traffic accident; Hemodialysis; Uremia.

1. INTRODUCTION

Long-term HD patients are inevitably injured due to accidents and incidents in production and life. At this time, the patient's injuries, underlying diseases and dialysis complications are often intertwined. Once the patient dies or other serious consequences occur, it is easy to cause liability disputes among the relevant parties. At present, HD has become a commonly used clinical treatment method. Patients with renal failure, uremia, etc. can survive for a long time by maintaining HD. However, this extracorporeal blood purification technology has both obvious effects and many complications. Among them, cerebrovascular complications, such as cerebral infarction and cerebral hemorrhage, are important causes of disability and death in HD patients [1-3]. Therefore, this article summarizes and reports a case of differential diagnosis practice of a dialysis patient who died of SDH and brain herniation after a traffic accident.

2. CASE

2.1. Background

An 55-year-old woman who had a traffic accident one morning. She was admitted to the hospital that day and the cranial CT scan showed no abnormality in the brain parenchyma and

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no displacement of the midline structure. The cranial CT on the second day showed patchy slightly high-density shadows in the right frontal lobe, and brain contusion and laceration were not excluded. The coagulation function test before hemodialysis on the third day: prothrombin time 23.36s, fibrinogen 5.42g/L, activated partial thromboplastin 48.68s, thrombin time 24.32s and D-dimer 1.22mg/L, but after hemodialysis treatment, she developed impaired consciousness, unequal pupils on both sides, and lost light reflex. The head CT showed SDH and brain herniation in the right frontal, temporal and parietal lobes. The deceased died five days later after failed rescue. The medical record shows that the deceased started HD three years ago, twice a week, had a cerebral hemorrhage a year ago, and had type 2 diabetes (more than 20 years), uremia and hypertension grade 3.

2.2. Forensic examination

Surface examination of the corpse:No swelling was felt on the scalp, no bone friction was felt on the skull, an arteriovenous fistula was visible on the radial side of the left forearm, and a 3.5cm×3cm skin cyanosis was seen around it. Three pinholes were seen 6cm below the left cubital fossa, and a 6.5cm×4.5cm skin cyanosis was seen around it. No other special findings were found.

Anatomical examination: A 10.2cm×11.5cm blood clot weighing 137g was found under the dura mater on the right frontal and temporal parietal (Fig. 1A), the right hippocampal sulcus was protruding (Fig.1B), and focal hemorrhage was seen on the middle cerebellar peduncle and pons section(Fig. 1C). No vascular rupture or hemangioma rupture was found in the cerebrovascular examination. The weight of the left and right kidneys was 130g and 129g, respectively, and the thickness of the renal cortex was 0.6cm and 0.5cm, respectively. No obvious abnormalities were found in the remaining organs and tissues.

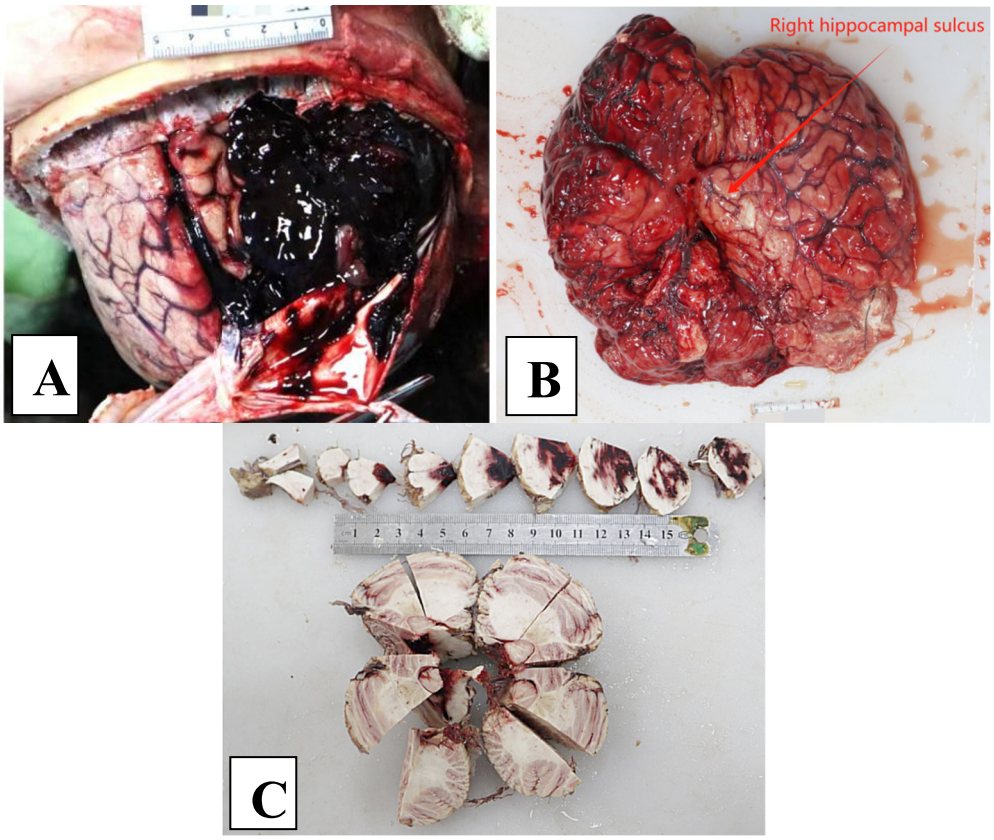

Figure 1. Right fronto-temporo-parietal SDH (A). The right side of the brain tissue was compressed by the hematoma, causing depression, and the right hippocampal sulcus was convex to the left (B). Bleeding was seen in the pons and middle cerebellar peduncle(C).

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Histopathological examination: A large number of red blood cells were found in the left frontal subdural area, accompanied by inflammatory cell infiltration (Fig. 2A), dilation and congestion of the meninges and cerebral blood vessels, leakage of red blood cells from the subarachnoid space of the left frontal lobe, widening of the spaces around cortical neurons and capillaries, increased number of microglia in the white matter, plaque hemorrhage in the pons and autolysis of brain tissue, inflammatory cell infiltration, and aggregation of inflammatory cells in the venule cavity (Fig. 2B). No obvious pathological changes such as aneurysms were found in the cerebral blood vessels. The left anterior descending coronary artery was atherosclerotic, with a stenosis of the lumen of grades I to II; glomerular fibrosis, focal infiltration of lymphocytes, renal tubular casts, dilation of interstitial venules, thickening of arteriolar walls, and autolysis of renal tubular epithelial cells and glomeruli.

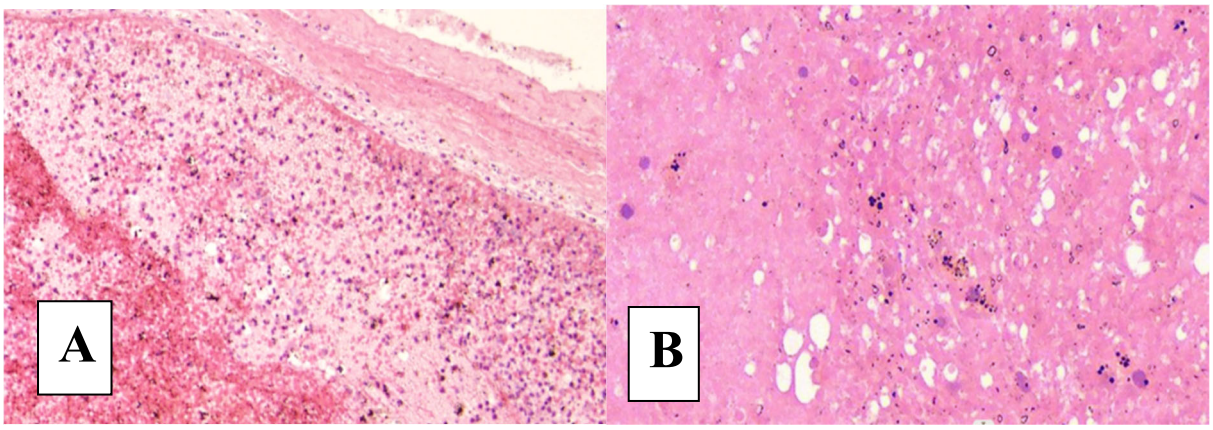

Figure 2. Right frontal subdural hemorrhage with inflammatory cell infiltration (A). plaque hemorrhages were seen in the pons with inflammatory cell infiltration (B).

Toxicological analysis: No common toxic (drug) components such as ethanol, sleeping pills, organophosphorus pesticides and their metabolites were detected in the deceased's heart blood, liver and stomach contents.

2.3. Identification of the cause of death

The deceased had chronic diseases (hypertension, diabetes, renal failure, uremia, etc.). After undergoing HD following the traffic accident, she developed SDH and secondary hippocampal sulcus herniation, which compressed the middle cerebellar peduncle and pons and caused bleeding, leading to dysfunction of the body's central nervous system. Ultimately, the deceased died of respiratory and circulatory failure. The deceased developed SDH and secondary hippocampal sulcus herniation, and her ultimate cause of death was the result of the combined effects of the fall in the traffic accident and her own diseases (including complications).

3. DISCUSS

The deceased in this case suffered from chronic diseases such as hypertension, diabetes, and renal failure. She had been on hemodialysis for 3 years and had a cerebral hemorrhage 1 year ago. The SDH occurred after hemodialysis on the same day, but she had a clear history of craniocerebral trauma 2 days before. The cause of death may be related to the deceased's own disease, complications of medical measures, and traffic accident injuries. The deceased's death may be mainly caused by a certain reason, or it may be the result of multiple factors. On the basis of a comprehensive autopsy, combined with the history of injuries and diseases, a systematic analysis of injuries and diseases, and the relationship between the two and the

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occurrence and development of fatal pathological changes should be conducted to clarify the cause and mechanism of the death of the deceased.

3.1. Trauma and SDH

Traumatic SDH is a common type of craniocerebral trauma, accounting for about 40% of intracranial hematomas. It often occurs after a road traffic accident, fall, blow, or other severe external force has been applied to the brain [4]. Patients generally have a clear history of head trauma, which may be accompanied by other craniocerebral injuries such as skull fractures and cerebral contusions. Typical SDH is caused by ruptured bleeding of bridging veins or blood vessels on the surface of the cortex. The hematoma compresses the brain tissue, which can easily lead to brain herniation and death. According to the speed of hematoma formation and the time of symptom onset, SDH can be divided into three types: acute, subacute, and chronic. Among them, those who develop symptoms within 3 days after injury are acute, accounting for about 70% of traumatic SDH [5]. SDH often occurs at or near the impact point of craniocerebral trauma, or on the opposite side of the impact point. It is most common in the frontal and temporal regions. The typical CT sign is a crescent-shaped or semilunar high-density shadow under the inner plate of the skull [6]. Due to the space-occupying effect of the hematoma, patients mainly experience symptoms and signs of increased intracranial pressure and brain herniation, such as headache, dizziness, and hemiplegia. The mortality rate of acute cases can be as high as 50% to 80% [7].

The deceased in this case had a clear history of craniocerebral trauma caused by a traffic accident. SDH occurred in the right frontal, temporal and parietal regions within 3 days after the injury. Although no skull fracture or vascular rupture was found during the autopsy, traumatic SDH cannot be ruled out. Anandasivam et al. [5] conducted a big data analysis based on 92,030 cases of SDH in the trauma database and showed that less than 1/3 of SDH patients had skull fractures. Kazuhisa et al. [8] also believed that the magnitude of external force and skull fracture were not necessary conditions for identifying traumatic SDH after analyzing traumatic SDH reported in the literature, and they also found that only about 1/6 of traumatic SDH cases could be found with vascular ruptures. Similarly, although bridging vein laceration is a typical feature of traumatic SDH, not all cases can be found with clear vascular ruptures during autopsy [9-11]. Therefore, the cause of death in this case should be comprehensively judged based on a systematic analysis of injuries, diseases and other factors. In the absence of other sufficient explanations for SDH, the possibility of traumatic SDH caused by a traffic accident cannot be ruled out.

3.2. Disease and SDH

Compared with traumatic SDH, spontaneous SDH caused by the body's own diseases and lesions is relatively rare. The main diseases and risk factors include vascular abnormalities (such as arteriovenous malformations and aneurysms), diabetes, hypertension, tumors, use of anticoagulants, and coagulation diseases [11,12]. The most common cause is aneurysm rupture, which leads to rapid bleeding, large hematoma, and high mortality [13]. Chronic diseases such as uremia, diabetes, and hypertension, as well as their secondary vascular lesions and coagulation abnormalities, often play an important role in the occurrence and development of SDH. Uremia can cause damage to the vascular wall, abnormal coagulation and fibrinolysis, and uremic factors in the plasma inhibit platelet adhesion and aggregation, which promote bleeding. At the same time, the use of anticoagulants will further aggravate the bleeding tendency of such patients [14]. With the development of the disease, diabetic patients can lead to the gradual decline of brain, kidney and heart functions and brain atrophy, which increases the risk of SDH. It has been reported that the 30-day mortality rate of SDH in diabetic patients is as high as 8.94% [12]. Long-term hypertension increases the fragility of the intracranial blood vessel walls,

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which can easily cause brain tissue to soften during vasospasm. This causes the blood vessels in the softened areas to lose brain tissue support and are prone to rupture and bleeding when blood pressure suddenly rises [15].

The autopsy of this deceased did not reveal obvious pathological changes such as cerebral vascular aneurysms, but the deceased had multiple chronic diseases such as hypertension, diabetes, and renal failure, and was a high-risk group for SDH. At the same time, the deceased had renal anemia, which would reduce blood viscosity, prevent platelets from contacting the blood vessel wall, and further increase the risk of bleeding [16]. Histopathological examination of this case showed atherosclerosis of the left anterior descending coronary artery, with a stenosis of the lumen reaching grades I to II; glomerular fibrosis, focal lymphocyte infiltration, renal tubular casts, dilatation of interstitial venules, and thickening of arteriolar walls. The pathological changes were consistent with the medical history and background. Comprehensive analysis shows that in this case, it cannot be ruled out that the deceased had spontaneous SDH due to his own diseases (hypertension, diabetes, uremia, etc.). Similarly, it cannot be ruled out that trauma induced and aggravated spontaneous SDH.

3.3. HD and SDH

HD mainly drains blood into the dialyzer and removes metabolic waste through diffusion, convection and other methods, thereby maintaining the balance of water electrolysis and pH in the body [17]. Cerebral hemorrhage is one of the main causes of death in hemodialysis patients. According to a study conducted by Fayed A et al. [14] on 1217 long-term hemodialysis patients, 3.37% of them developed SDH, and the mortality rate of patients with SDH within 30 days was 29.27%. Intracranial hemorrhage in HD patients often occurs during or 24 hours after hemodialysis. The main factors include: (1) coagulation dysfunction: toxin accumulation damages the coagulation system, resulting in platelet dysfunction and aggregation inhibition, which leads to bleeding. At the same time, dialysis can activate the coagulation system, which is easy to form thrombi on the dialysis membrane and consume platelets and coagulation factors [18]. (2) use of anticoagulants: uremia patients often need maintenance dialysis. Long-term and repeated use of anticoagulants, coupled with transient thrombocytopenia and volumetric hypotension during dialysis, make hemodynamics unstable, further aggravating the bleeding tendency [19]. (3) vascular sclerosis and calcification: uremia and other end-stage renal disease patients often have hypertension, hyperlipidemia, atherosclerosis, etc. Hyperphosphatemia and accumulation of parathyroid hormone can cause vascular calcification, increase vascular sclerosis and vascular fragility, and further increase the bleeding tendency [14]. According to Diener et al. [20], hyperglycemia, hypertension and hyperlipidemia are all risk factors for intracranial hemorrhage in HD patients .

The deceased in this case was a patient with uremia who started maintenance HD 3 years ago. She also had high-risk factors such as diabetes and hypertension, and had a cerebral hemorrhage one year ago. The SDH occurred on the third day after the traffic accident and after hemodialysis, but the CT scan the day before did not show any signs of SDH. Therefore, the possibility of hemodialysis complicating SDH in the deceased cannot be ruled out.

3.4. Cause of death analysis

As mentioned above, the SDH of the deceased in this case could be traumatic, spontaneous, or a complication of hemodialysis. None of the three can be ruled out. Numerous literature has shown [21] that the development of SDH, as well as death after SDH, is affected by both the degree of neurological damage and the overall health status of the deceased at the time of injury. Minor injuries can induce fatal SDH in patients with fragile blood vessels and coagulation disorders. Underlying diseases may also reduce an individual's tolerance to trauma, aggravate the consequences of injury, and increase the incidence of complications and even death. The

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case facts, injury history, identification data and autopsy findings of this case all support that the deceased died of central nervous system dysfunction caused by SDH and brain herniation, that is, SDH and brain herniation were the direct causes of death of the deceased. As for the cause of SDH and brain herniation, that is, the fundamental cause of death of the deceased, or the causal relationship between traffic accident trauma, the deceased's own disease and hemodialysis and SDH and brain herniation, a comprehensive and systematic analysis should be conducted in combination with the literature and the specific circumstances of this case before a scientific and reasonable judgment can be made. At this stage, this case should be summarized as a combination of injury and disease (including complications) leading to SDH, brain herniation, and even death.

4. SUMMARY

Cases of coexistence of trauma, disease and their complications will also increase in forensic judicial appraisal. With the aging of the population and the development and popularization of medical technology, there will be more and more individuals living with long-term illnesses. Medical technologies such as hemodialysis that maintain the survival of patients with some diseases have certain risks in themselves, and patients may die suddenly due to complications [22,23]. Clinical workers should be vigilant. For patients with underlying diseases, they should comprehensively consider injury and disease information, make early predictions and take preventive measures, adjust treatment strategies, and reduce the incidence of complications or adverse consequences. For such cases, forensic workers should comprehensively consider the case, analyze the relationship between disease, injury and death, and make scientific and reasonable judgments based on sufficient materials.

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