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Sudden Death in Hemodialysis Patients

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ABSTRACT: Hemodialysis patients may die suddenly and unexpectedly from a number of causes. These may be divided into those deaths due directly to and occurring during hemodialysis, those deaths occurring while the patient is not undergoing dialysis, and those deaths that may occur at any time. The first group includes brain herniation, air embolism, acute hemorrhage as a result of machine malfunction or fistula rupture, electrocution, cardiac arrhythmia caused by hypokalemia, complications of subclavian intravenous catheter insertion, third-degree heart block as a result of triglyceride emulsion, and disseminated intravascular coagulation (DIC) or hyperkalemia caused by overheated dialysate. The second group includes deaths due to pericardial tamponade because of effusion and suicidal causes of death (exsanguination, electrolyte imbalance as a result of excessive intake of salt, fluid, or potassium) as well as more conventional methods of suicide. The last category includes people dying of arteriosclerotic cardiovascular disease, hypertensive cardiovascular disease, and internal hemorrhage. Investigation of these deaths, including pertinent historical, laboratory, and autopsy data and investigation of dialysis equipment, is discussed.

KEYWORDS: pathology and biology, dialysis, death

There are 50 000 patients per year undergoing renal dialysis in the United States. An additional 5000 annually enter a dialysis program [*1*]. Cardiovascular disease and infection (especially *Staphylococcus aureus* sepsis) are the leading causes of death [*2-4*]. Hemodialysis patients have three times the cardiovascular mortality as the general population, primarily because of hypertension, which is present in 80 to 90% of patients before they begin dialysis [*2*]. There are many types of life-threatening complications that may occur during, or as a result of, hemodialysis. The medical examiner or forensic pathologist charged with the investigation of the sudden, unexpected death of a hemodialysis patient faces analysis of procedures, equipment, and diseases that are not encountered in his usual practice.

A review of the medical literature on this topic has yielded reports of numerous fatal or potentially fatal complications. They may be divided into deaths occurring during dialysis, deaths while the patient is not undergoing dialysis, and deaths that can occur at any time (Table 1).

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TABLE 1—*Sudden death in hemodialysis patients.*

Deaths During Dialysis	Deaths Off Dialysis	Deaths During or Off Dialysis
Hypokalemia	cardiac tamponade	internal hemorrhage
Anaphylactic reactions or bronchospasm	suicide	arteriosclerotic cardiovascular disease
Brain herniation	external hemorrhage	hyperkalemia
Overheated dialysate		
Hemopericardium caused by subclavian catheter		
Electrocution		
Third degree heart block caused by triglyceride emulsion		
Contaminated dialysate		
Acute hemorrhage		
Air embolism		

Deaths During Dialysis

Patients who are hypokalemic are at risk to develop cardiac arrythmias, including premature ventricular contractions and ventricular fibrillation. Predisposing to this complication is the presence of preexisting conduction system abnormalities, coronary artery disease, left ventricular hypertrophy, and treatment with digitalis preparations and diuretics. In patients with arrythmias caused by hypokalemia, there is often a history of nausea and vomiting, diarrhea, and nasal suctioning. The initiating event is often dialysis against a very low potassium concentration, or potassium-free bath, causing intracellular potassium depletion. Since abrupt and large changes in serum potassium can induce cardiac arrythmias, this diagnosis must be considered even in the patient with predialysis hyperkalemia [*5,6*].

Hypokalemic deaths may be documented by vitreous potassium determination as shown by Sturner and Dempsey [*7*], who documented low vitreous in an infant 4 h after death. If blood could be obtained within minutes after death, an accurate serum potassium concentration might be obtained.[3] A low vitreous or blood potassium level is strongly indicative of antemortem hypokalemia since serum potassium increases after death.

Anaphylaxis or exacerbation of preexisting asthma after the onset of hemodialysis is a potential cause of sudden death. Aljama et al [*8*] reported the case of a 40-year-old woman with intrinsic asthma who had episodes of wheezing and shortness of breath within 1 h of the start of dialysis. She improved after substitution of the Cupruphan® membrane with one made of polyacrylonitrile. Her respiratory distress was felt to be due to pulmonary leukostasis, caused by the Cupruphan, resulting in release of bronchoconstrictor substances from impacted neutrophils in the lungs. Nicholls and Platts [*9*] observed four severe anaphylactic reactions characterized by bronchospasm and cardiovascular collapse occurring within 1 min of the time the patient's blood began flowing through a dialysis filter. The cause was felt to be an allergic reaction to the ethylene oxide (acting as a hapten) with which the dialyzers were sterilized. The dialyzers in question had been improperly primed before usage, resulting in probable failure to remove all of the ethylene oxide.

Fatal brain herniation occurred in an 18-year-old male 3 h after the start of dialysis. There was no evidence of a preexisting lesion, such as cerebral hemorrhage or subdural hematoma. The reason for the herniation was felt to be a decrease in cerbrospinal fluid pH, probably brought about by rapid dialysis. The increase in brain osmolarity because of the increase in hydrogen ion concentration causes cerebral edema [*10*]. Another postulated mechanism for

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cerebral edema occurring during dialysis is the rapid lowering of serum urea. The cerebrospinal fluid urea concentrations do not diminish as rapidly as in the serum because of slow transport of urea across the blood brain barrier. This produces an osmotic gradient, with water moving from the blood and extracellular fluid into the central nervous system (CNS) [*11*].

Overheated dialysate can cause death by massive hemolysis which may be accompanied by disseminated intravascular coagulation (DIC) or hyperkalemia. In one dialysis center, three people died and four others suffered nonfatal cardiovascular collapse while undergoing dialysis because of improper wiring in a heater alarm system and corrosion of an on-off control which caused an auxiliary heater to turn on. Serum potassium levels were not elevated in the decedent and one survivor tested. No significant anatomic changes were seen at autopsy.[4] Tietlemans et al [*12*] reported the case of a patient who, having been dialyzed against a 52°C dialysate for about 100 min, complained of sudden pain in her arteriovenous fistula. She developed DIC, probably as a result of thromboplastic substances released from lysed red blood cells, and subsequently recovered. An investigation showed that the mechanical thermostat of the heating system allowed wide temperature swings between the on and off settings, and the adjustable alarm had not been set at the 35 to 42°C range. This type of temperature monitor and alarm is not now in common use. Another patient cited by Tietlemans et al [*12*] was dialyzed against a bath at 55°C, developed massive hemolysis, and suffered a fatal cardiopulmonary arrest, presumably because of hyperkalemia. Temperature alarms must be set manually on dialysis machines currently used. When the alarm is activated, the dialysate is shunted around the dialyzer.

In patients in urgent need of dialysis, rapid access may be achieved through a subclavian vein. The catheter is significantly stiffer than those used for nondialysis purposes. A 60-year-old woman collapsed with severe hypotension 35 min after the start of dialysis and died 15 min later. At autopsy there was a hemopericardium (300 mL) caused by perforation of the anterior wall of the superior vena cava. The perforation was ascribed to a stiff catheter tip [*13*]. Barton et al [*14*] reported three cases of nonfatal atrial or vena-caval perforations in adults with subclavian catheters. The first patient sustained a perforation at the junction of the right atrium and inferior vena cava 3 h after the start of hemodialysis. The second patient had a perforation of the superior vena cava at the junction of the right atrium and the third had a perforation at the border of the right atrum and inferior vena cava. Both had falls in blood pressure immediately after beginning hemodialysis. The perforations were thought to be due to tenting of the caval wall by the stiff catheter as a result of suboptimal positioning in the superior vena cava. Bleeding was accelerated by the forceful returning of blood through the catheter secondary to the pumping action of the dialysis machine. Re-examination of anteroposterior (AP) or posteroanterior (PA) chest X-rays showed a lack of caudal curvature of the catheter tip toward the right atrium. Barton et al [*14*] also mention two reported instances of hemothorax due to vena-caval or subclavian vein perforation caused by the repositioning of catheters thought to have slipped out of the vena cava. Other complications of subclavian catheter insertion include pneumothorax and hemothorax.

Gotloib and Servadio [*15*] have the only reported case of sudden death during dialysis as a result of electrocution. A 41-year-old patient had a cardiac arrest 45 min after the start of hemodialysis. The autopsy failed to show a cause of death, but examination of the dialysis machine revealed a short-circuited, ungrounded thermostat. Using a microammeter, current leaks of 42 μA into the dialysis machine and 37 μA into the dialysis bath were found. Using saline, a current of 35 to 37 μA was found, which was reduced to 4 to 7 μA with grounding. The authors point out that electrical leaks in a dialysis bath through either the thermostat or heating element can easily pass through a thin, low-resistance cellophane

[4]N. Hollander, medical examiner, Southwestern Institute of Forensic Sciences, Dallas, TX, personal communication, 1984.

membrane traveling to the heart through the blood column. They state that an alternating current of 60 Hz at 20 μA can cause ventricular fibrillation when applied directly to the heart. In home dialysis, the dialysis equipment may act as a sink for nonmedical equipment (for example, a television set) not built to medical standards [*16*]. The main factors constituting a primary electrical hazard are failure of grounding and insulation. Grounding failure, the most common electrical hazard, may cause current to flow through the patient. Electrocution could result when the patient touches an improperly grounded component.

Two patients given a triglyceride emulsion for alimentary purposes died of third-degree heart block while undergoing hemodialysis [*17*]. At autopsy one patient had multiple subendocardial infarcts and the other had left ventricular hypertrophy. One patient had a serum free fatty acid level of 2463 μmol/L (normal 200 to 800). The other patient's free fatty acids were not measured. Both patients were treated with heparin, which increases the level of free fatty acids. Free fatty acids have caused ventricular arrythmias in patients with apparently normal hearts who were undergoing dialysis.

There are several unusual causes of sudden death during dialysis [*5*]. Dialysate contaminated with aluminum, calcium, and fluoride can cause sudden death. Acute hemorrhage can result from line separation on the dialysis machine. Merrill [*11*] points out that air embolus can result from a low level of blood in the drip chamber and leakage of air into blood connections on the negative pressure side of the line. Modern dialysis machines have air detectors that must be set manually; when a leak is detected, the machine stops.

Deaths Occurring Off Dialysis

Pericardial tamponade, suicides, and external hemorrhage generally occur in the patient who is off dialysis. Watson et al [*18*] described two hemodialysis patients who suddenly became severely hypotensive. Pericardiocentesis, performed after ultrasonography, yielded 1000 mL (hematocrit 20%) and 900 mL (hematocrit 17%) of fluid, respectively. Tamponade (other than that caused by hemorrhage) in hemodialysis patients has two causes: uremic and dialysis pericarditis. Uremic pericarditis, with an incidence of 20%, occurs in underdialyzed or undialyzed patients, who are azotemic. Dialysis pericarditis, which occurs in 2 to 27% of dialysis patients, usually is seen within three months of the start of dialysis. It is associated with treatment with heparin and accumulation of fluid caused by the osmotic gradient between pericardial fluid and blood. In 20% of patients with dialysis pericarditis the effusion progresses to tamponade. Dialysis tamponade may not be suspected clinically because the pulsus paradoxus typically associated with tamponade is absent.

Suicide accounts for approximately 1% of deaths in hemodialysis patients [*19*]. Hemodialysis patients have a suicide rate of from ten [*19*] to one hundred [*20*] times the suicide rate of the general population. Traditional methods of suicide may be utilized; methods peculiar to dialysis patients include exsanguination by disconnecting or otherwise interrupting the shunt and excessive intake of salt, fluid, or potassium.

Acute external hemorrhage can result from a disconnected shunt or infected arteriovenous fistula. Two such cases are presented.

Case 1

The body of a 67-year-old diabetic chronic dialysis patient was found lying in a large pool of blood on his bedroom floor. A belt was tied around the left upper arm and a bloody paper towel was found next to the body. Vascular access for dialysis was provided by a subcutaneous arteriovenous fistula (Gortex graft) in the left antecubital area. Severe peripheral vascular disease caused difficulty in maintaining graft patency, requiring numerous surgical explorations, that most recent occurring two weeks before death at which time a thrombectomy was performed. Examination of the body identified the source of the fatal hemorrhage as a

perforation of the shunt (Fig. 1, upper). A hemorrhagic track led from the 2-cm site of skin perforation (Fig. 1, lower) to a 10-by-7-mm defect in the anterior wall of the graft. The graft defect was located in an area where multiple needle punctures overlapped. The intact suture line from the recent surgical procedure was located lateral to the site of rupture. Microscopically, the subcutaneous tissue in the area of the graft was extensively infiltrated by acute inflammatory cells. *Staphylococcus aureus* was isolated from this area. Three factors appear to have contributed to the occurence and severity of the hemorrhage. The numerous closely spaced needle punctures and the staphylococcal infection were local factors operative in the incident. The deceased may also have had a systemic coagulopathy caused by his renal failure (vitreous urea nitrogen 131 mg/dL, vitreous creatinine 8.3 mg/dL).

Case 2

A 47-year-old woman who was undergoing chronic dialysis because of analgesic nephropathy was found lying on her bed with her feet resting on the floor. A large pool of blood was present on the floor around her feet. An external dialysis shunt catheter had been placed in the right leg was covered with a blood-soaked bandage. She had awakened her mother, who noted profuse bleeding from the site of the catheter. An ambulance was summoned but the daughter was dead at the time of their arrival. Examination of the right leg demonstrated that the arterial end of the catheter was in place but the venous portion was displaced.

Deaths Occurring At Any Time

Hemorrhage is a common potentially fatal complication in hemodialysis patients [*21*]. Subdural hematomas, gastrointestinal hemorrhage, hyphema, retroperitoneal hemorrhage, mediastinal hemorrhage, hemorrhagic pleural effusions, ovarian hemorrhage [*22*], and subcapsular liver hematomas have been reported. Glass et al [*22*] reported the case of a 21-year-

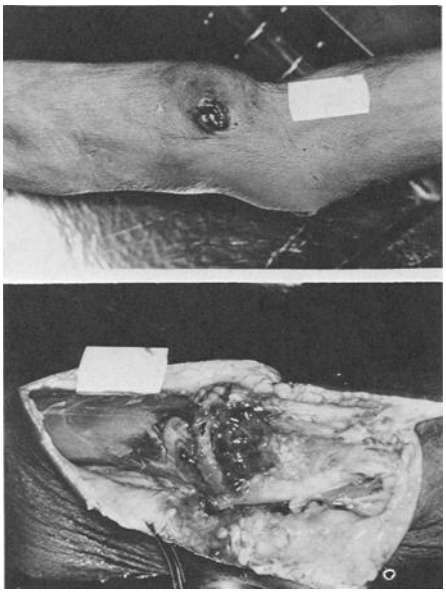

FIG. 1—(upper) *Defect in left antecubital fossa connected by hemorrhagic track to graft and* (lower) *defect in graft, in an area containing multiple needle punctures, and surrounded by* S. aureus *infection.*

old woman who had been undergoing hemodialysis for six months and was treated with anticoagulants. One night, 25 days after her last menstrual period, she became severely hypotensive. At laparotomy, hemoperitoneum (800 mL) caused by a ruptured corpus luteum was found and the patient died shortly thereafter. The authors felt that the normal blood loss from the corpus luteum was magnified by anticoagulant therapy. Subdural hemorrhage occurred in 3.3%[3] of Leonard and Shapiro's [*23*] 394 patients, only 2 of which survived. Most of the subdural hemorrhages were bilateral, caused localized signs, and occurred after an average of 13.5 months of dialysis. Factors involved in the development of subdural hemorrhage included anticoagulant therapy and hypertonic dialysis (producing shrinkage of the brain with resultant tension on bridging veins). A history of head trauma was present in four of the thirteen cases. Common factors causing hemorrhage location in hemodialysis patients are anticoagulant therapy and platelet dysfunction as a result of uremia.

Hemodialysis patients have three times the cardiovascular mortality as the general population [*2*], and thus, may die suddenly and unexpectedly at any time, including during dialysis. Risk factors particularly common in hemodialysis patients are hypertriglyceridemia, which occurs in 50 to 93%, and hypertension. To certify the cause of death as arteriosclerotic cardiovascular disease or hypertensive cardiovascular disease of course one must exclude the causes of death listed above.

Hyperkalemia occurs in patients who have not adhered to their diets. Such individuals may be found dead, or die shortly after commencement of dialysis. When seen before death, the chief complaint is weakness.[5] Electrocardiographic findings are characteristic.

Conclusion

In the major dialysis center serving western Michigan (St. Mary's Hospital in Grand Rapids, MI) blood urea nitrogen (BUN), creatinine, potassium sodium, and hematocrit are routinely assayed predialysis and postdialysis in new patients. Chronic hemodialysis patients have electrolytes, a complete blood count, hepatitus B surface antigen (HBsAg), and a SMAC (Sequential Modular Analysis Computerized) battery performed once monthly predialysis. A potassium level is determined at every dialysis in unstable patients. Patients who are markedly hyperkalemic and who are dialyzed against a potassium-free bath undergo electrocardiographic monitoring during dialysis. Electrocardiograms (EKGs) are obtained at least every six months. Patients receiving a triglyceride emulsion or a nutritional supplement must be dialyzed daily. Chest X-rays are taken after insertion of a subclavian catheter. The softer polyurethane catheter has been substituted for Teflon®.

In the St. Mary's Dialysis Center, dialysis machines have a routine safety check every six months by specially trained technicians. The conductivity of the bath and grounds in the machine are checked every three months. Occasionally faulty grounding had been found but there have been no electrocutions. Cupruphan membranes are used, but in case of an asthmatic episode, a plate dialyzer is substituted. Saline recirculation through the system for 10 min or reuse of a dialyzer is usually sufficient to rid the system of ethylene oxide. Devices used to prevent air embolism include an air detector, bubble trap, and an alarm that must be set manually. Newer heaters on the Gambro dialysis machines used at this center are controlled by a microprocesser which shuts off the flow of dialysate at temperatures above 40°C.

When a patient dies suddenly while on hemodialysis, it is mandatory that the dialysis equipment not be disturbed until the scene can be investigated. All tubes must be left in place and antemortem blood samples must be submitted to the forensic pathologist. A medical history, including the events occurring immediately before death, should be obtained. Postulated mechanisms and suggestions for investigation of these deaths are enumerated in Tables 2 through 4.

[5]J. Galloway, chief nurse, Saint Mary's Hospital, Hemodialysis Unit, personal communication, 1983.

TABLE 2—*Deaths during dialysis.*

Cause of Death	Mechanism of Death	Method of Determination
Hypokalemia.	Cardiac arrythmias, premature ventricular contractions (PVCs), ventricular tachycardia, especially in presence of left ventricular hypertrophy (LVH), digoxin.	1. Predialysis hypokalemia. 2. Dialysis against low potassium dialysate (history). 3. LVH, coronary artery disease, or previous conduction abnormalities. 4. History of nausea and vomiting (N&V), diarrhea, nasal suction, digitalis preparation, diuretic.
Anaphylactic reactions or bronchospasm.	1. Pulmonary leukostasis induced by Cupruphan membrane, with release of bronchoconstrictor substance. 2. Allergy to ethylene oxide used to sterilize dialyzer.	1. Determine type of membrane used in dialysis. 2. Determine method used to sterilize equipment. 3. History of intrinsic asthma.
Brain herniation.	1. Increased cerebrospinal fluid (CSF) hydrogen ion concentration, causing increased osmolarity leading to edema. 2. Edema caused by rapid lowering of serum urea.	1. Autopsy and/or clinical evidence of herniation 2. History of rapid dialysis.
Overheated dialysate (defects in manufacture and improper maintenance, thermostat malfunction, failure to set alarm temperature).	Hemolysis with or without DIC (in some cases mechanism unknown).	1. Examination of dialysis machine by engineer, including maintenance records. 2. Autopsy. 3. Postmortem blood or vitreous electrolytes (if obtainable soon after death).
Hemopericardium or hemothorax because of subclavian dialysis catheter.	Perforation of atrium or vena cava by catheter tip.	1. AP chest X-ray. 2. Direct examination at autopsy. 3. History of repositioning catheter.
Electrocution.	Ventricular fibrillation.	1. Machine check by electrical engineer for proper grounding, and insulation breakdown; including check of maintenance records.
Triglyceride emulsion causing three-degree heart block.	Unknown; ? elevated free fatty acids.	1. History of triglyceride administration; high serum free fatty acids. 2. EKG.
Contaminated dialysate.	Unknown.	1. Document lack of use of de-ionizer and reverse osmotic device.
Acute hemorrhage	Exsanguination caused by dialyzer leak.	1. Check dialyzer.
Air embolism.	Air in venous line.	1. Autopsy. 2. Machine check, including maintenance records.

TABLE 3—*Deaths off dialysis.*

Cause of Death	Mechanism of Death	Method of Determination
Cardiac tamponade	1. Hemopericardium caused by administration of heparin. 2. Pericardial fluid accumulation as a result of osmotic gradient between pericardial fluid and blood.	1. Autopsy. 2. History of anticoagulation and reagent.
Suicidal types of death	1. Hyperkalemia. 2. Excessive intake of salt/fluid. 3. Exsanguination. 4. Traditional methods of suicide.	1. Autospy. 2. Postmortem vitreous or blood electrolytes, creatinine, and urea nitrogen. 3. History, scene investigation
External hemorrhage	Exsanguination caused by disconnected shunt or rupture of arteriovenous (A-V) fistula.	Examination of shunt and vascular access site.

TABLE 4—*Deaths occurring during or off dialysis.*

Cause of Death	Mechanism of Death	Method of Determination
Hyperkalemia	1. Cardiac arrhythmias.	1. Dietary history. 2. Serum potassium (antemortem or immediately postmortem).
Internal hemorrhage	1. Uremic functional platelet disorders. 2. Anticoagulant administration. 3. Trauma (subdural hemorrhage). 4. Hemorrhagic corpus luteum.	1. Autopsy. 2. History of anticoagulation. 3. History of trauma.
Arteriosclerotic cardiovascular disease (ASCVD)	Ventricular fibrillation	1. Autopsy. 2. Eliminate other possible causes.

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