

Acute Massive Cerebral Infarctions Treated with Hemodialysis

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We report here a 19-year-old woman with Down syndrome and end-stage renal disease who presented with left-sided weakness and fever. She had a massive pericardial effusion of unclear origin that required daily hemodialysis (HD) and cardiac intervention. She developed an acute right middle cerebral artery infarction with severe edema; her cerebral edema significantly improved with daily HD. Later in her hospitalization, she developed seizures and new onset of multiple acute embolic infarctions in left middle cerebral artery, left anterior cerebral artery (ACA), and right posterior cerebral artery (PCA) distributions with midline shift. However, we again noticed a dramatic decrease in cerebral edema with frequent HD. Although there is controversy about the use of dialysis in patients with stroke, our case suggests that daily HD may provide an alternate strategy for treating massive cerebral infarction. More studies are needed in these patients. **Key Words:** Hemodialysis—cerebral edema—stroke.

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A 19-year-old woman with Down syndrome and end-stage renal disease on hemodialysis (HD) was admitted for sudden onset of left hemiparesis and fever after HD. She had been on HD for 2 years. She had no family history of stroke or other thromboembolic disorders. Initial physical examination revealed mild left hemiparesis without facial weakness. Blood pressure was 150/90 mm Hg, pulse 76/min, respirations 22/min, and temperature

38.9°C. Her initial head computed tomography (CT) scan revealed normal findings. However, transthoracic echocardiogram showed a large pericardial effusion with tamponade. She required pericardiocentesis on day 4 and pericardiectomy on day 9. Daily dialysis was initiated for possible uremic pericardial effusion from days 9 to 14. On day 12, she progressed neurologically and developed speech arrest, left hemiplegia, and left homonymous hemianopia. Head CT demonstrated an acute right middle cerebral artery (MCA) infarction with extensive edema and mass effect (Fig 1, A). A total of 5 L of fluid was removed by HD on days 12 through 14. CT on day 15 showed decreased mass effect (Fig 1, B). She improved neurologically and regained the ability to follow simple commands, but her left hemiparesis persisted.

She was medically stable and awaiting nursing home transfer until day 37 when she developed fever to 38.5°C. On day 46, she developed status epilepticus and left gaze deviation. She was treated with fosphenytoin and intubated. She opened her eyes spontaneously but did not follow commands. She also developed a flaccid quadriplegia and a decorticate response to painful stimuli. CT on that day showed no acute infarction, but follow-up CT on day 49 showed two acute embolic infarctions in

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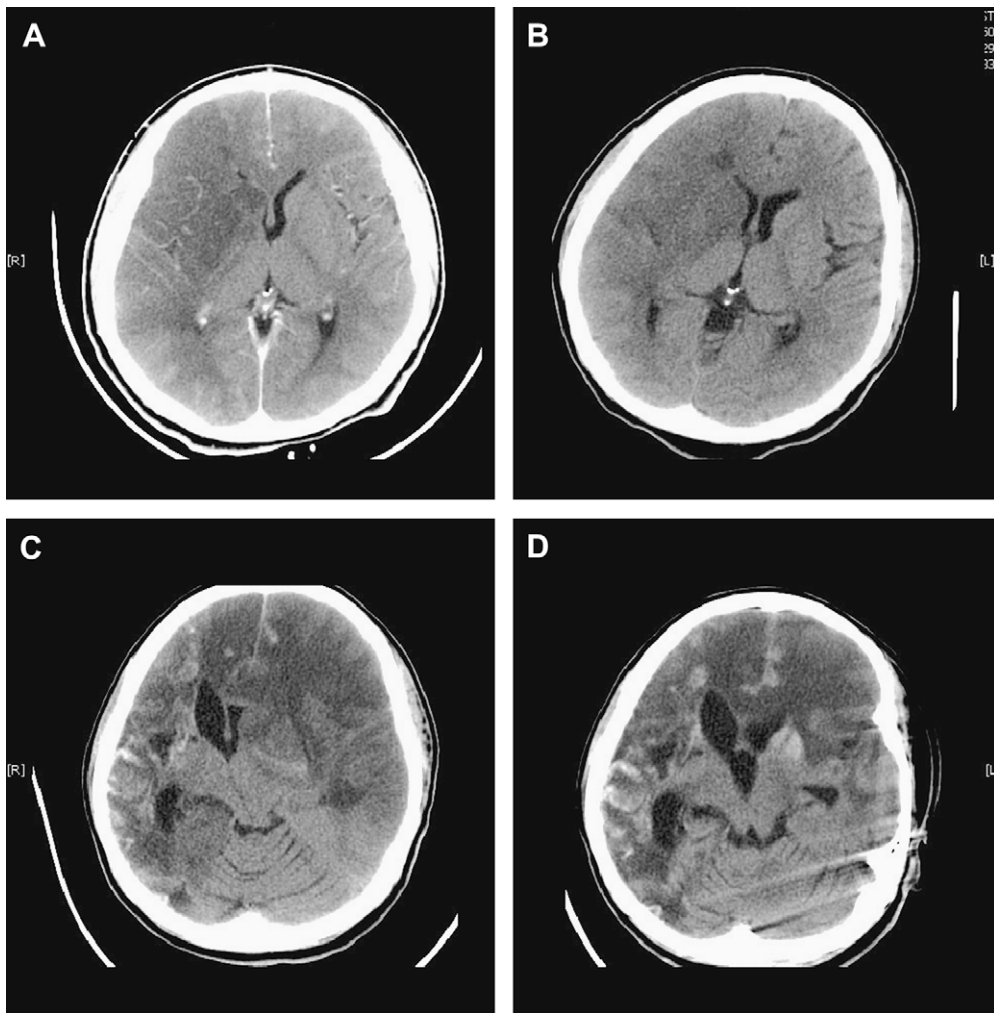


Figure 1. Head CT with contrast demonstrates right middle cerebral artery (MCA) infarction with extensive cytotoxic edema with mass effect (A). Head CT without contrast shows improvement after 5 days of dialysis with 6 L of fluid removed (B). Head CT without contrast demonstrates acute left anterior cerebral artery (ACA), MCA, and right posterior cerebral artery (PCA) infarctions with subfalcine and transtentorial herniation (C). Dramatic improvement after 4 days of dialysis with 12.3 L of fluid removed (D).

the left MCA distribution with mass effect and midline shift and in the right posterior cerebral artery (PCA) distribution (Fig 1, C). She subsequently developed acute left anterior cerebral artery (ACA) infarction and extension of the left MCA infarctions with subfalcine herniation and transtentorial herniation on day 54. She received one bolus of hypertonic saline infusion (75 mL) and HD for reducing cerebral edema. She had 12.3 L of fluid removed in 4 days with eventual resolution of midline shift and uncal hernia, although her neurologic status remained unchanged (Fig 1, D). Her intracranial pressure (ICP) was not measured during dialysis. Her hospital course was complicated by acute myocardial infarction, pneumonia, catheter-related infection, and deep vein thrombosis. A transesophageal echocardiogram produced negative results for vegetations and clots. There was a mild increase in lupus anticoagulant of 50.8 (27-41), but other factors, such as factor V Leiden, prothrombin mutation, anticardiolipin antibody, and antinuclear antibody

(ANA), were negative. Anti-neutrophil cytoplasmic antibodies (ANCA), rheumatoid factor (RF), complement, serum protein electrophoresis, homocysteine, VDRL, and cytomegalovirus (CMV) titers were within normal limits.

Discussion

Massive MCA infarction is associated with an 80% mortality. No medical intervention has been established as effective in controlling cerebral edema or improving outcomes after stroke, but surgical decompression within 48 hours of stroke onset reduces mortality and increases the likelihood of a favorable functional outcome.¹ We noted improvement in our patient's cerebral edema with daily HD after her first stroke. Although it is possible that her initial improvement may have been spontaneous, she also had a dramatic decrease in cerebral edema after her second stroke with HD. The advantage of using HD in such cases is the ability to remove fluid in a relatively

short period of time and thereby significantly reduce cerebral edema and mass effect and limit electrolyte abnormalities. However, previous studies have shown that intermittent HD may worsen cerebral infarction, either from reduction in cerebral perfusion pressure or because of increased cerebral edema secondary from the diffusion gradient of urea and osmolality between plasma and CSF and brain.² These changes in cerebral edema and perfusion can be minimized by carefully modifying dialysis prescription, including daily HD, using less bicarbonate concentration, or a high-sodium dialysate.³ Complex cases may require ICP monitoring. Daily HD can reduce the osmotic gradient across the blood-brain barrier and, when combined with fluid removal, has a net effect of decreasing cerebral edema such as the result seen in our patient.

Another factor that may contribute to reducing cerebral edema is the elimination of inflammatory cytokines, such as interleukin (IL)-1 β , tumor necrosis factor (TNF- α), and IL-8, associated with ischemic injury. Both animal and human experiments have demonstrated that inflammatory mediators can be cleared by hemofiltration and hemodiafiltration. This interest has led to the use of continuous renal replacement therapy as adjunctive therapy for burn patients with sepsis.⁴ However, the use of anticoagulation with continuous renal replacement therapy in patients with acute stroke is contraindicated because of possible hemorrhage.³ Although the standard HD membrane does not remove inflammatory cytokines associated with ischemic injury, the combined use of a high-performance membrane (e.g., polymethylmethacrylate and polysulfone used in our case) and high-flux dialysis has been shown to remove and absorb midsize molecules up to 30 kDa such as β -2microglobulin, IL-8, and IL-1b.⁵⁻⁷ Aggres-

sive removal of humoral factors may reduce cerebral edema, especially when dialysis is initiated immediately and repeatedly after an acute inflammatory process.

In summary, this case suggests that HD may represent a novel strategy for treating massive cerebral infarction, but more study is needed.

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