Multiple Organ Infarctions Following Disseminated Intravascular Coagulation Precipitated by Sepsis in a Healthy Infant: A Case Report

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Multiple organ infarctions are a very rare clinical event in children. We report a 3-month-old infant with sepsis and disseminated intravascular coagulation, who was diagnosed with cerebral ischemic stroke associated with middle cerebral artery stenosis and subsequent retinal infarction by magnetic resonance imaging, fundoscopy and magnetic resonance angiography. In addition, he suffered from renal infarction with hypertension and was treated until 1 year of age. We emphasize the importance of early recognition of organ infarctions, prophylaxis of risk factors and of optimized therapy of the underlying etiology.

Key Words: disseminated intravascular coagulation, infant, multiple organ infarction
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Disseminated intravascular coagulation (DIC) is characterized by inappropriate widespread activation of coagulation that leads to extensive microvascular thrombosis and hemorrhage. It is not a rare disorder, and has a frequency of 1.12% in hospitalized children, especially in those with sepsis [1]. Septic patients with severe DIC might present with manifestations of thromboembolic disease or with clinically less apparent microvascular fibrin deposition, which presents predominantly as multiple organ dysfunction [2]. Bleeding is the leading clinical symptom [1]. However, macrocirculatory thrombotic events are rare in this setting, except in babies who have a central catheter *in situ*. We report on the case of a 3-month-old previously healthy Taiwanese infant, who presented with multiple organ infarctions, including brain, retina, kidney and skin, following septic shock and DIC.

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Case Presentation

A previously healthy 3-month-old male infant presented to our emergency department with altered consciousness, and was admitted to the pediatric intensive care unit under suspicion of sepsis. Five days earlier, fever and poor feeding, with clinical signs of upper respiratory tract infection, were noted, followed by diarrhea with muddy and mucus-coated stool 2 days later, when plain abdominal X-ray and abdominal echography revealed no specific findings. On the following 2 days, he was noted to have multiple petechial rashes over the chest and abdomen, with bloody stools.

On admission, the infant appeared irritable and ill. He had dyspnea and unstable vital signs, with a respiratory rate of 40 breaths/min, pulse rate of 220 beats/min and blood pressure of 36/17 mmHg. On physical examination, he showed lip cyanosis, dry oral mucosa

and mottled skin. Respiratory distress with subcostal retraction and nasal flaring was evident. His extremities were cold, with a prolonged capillary refilling time. Laboratory examination revealed a white blood cell count of 31,000/μL; platelet count of 623,000/μL; serum creatinine, 1.0 mg/dL; blood urea nitrogen, 23.8 mg/dL; C-reactive protein, 9.8 mg/L; prothrombin time, 16.3 seconds (normal range, 8.0–12.0 sec); activated partial thromboplastin time, 38.5 seconds (normal range, 24.0–36.8 sec); and D-dimer, 5,777 μg/L (normal range, <324 μg/L). Liver enzymes were elevated [aspartate aminotransferase = 4,047 IU/L (normal range, 10–42 IU/L); alanine aminotransferase = 1,424 IU/L (normal range, 10–40 IU/L)] and blood lactate level was 12.9 mmol/L (normal range, 0.5–2.2 mmol/L). Stool routine showed 2+ occult blood; urine analysis revealed hematuria (5–10 red blood cells/high power field) and 4+ proteinuria. Chest radiographs were normal. Septic shock with multiple organ dysfunction (liver dysfunction, coagulopathy and prerenal azotemia) and metabolic acidosis (venous blood gas = pH 7.2; partial pressure of CO_2 = 11 mmHg; HCO_3^- = 6.3 mmol/L) were observed. Blood cultures were obtained, and intravenous cefotaxime and ampicillin were initiated. The patient was managed with fresh frozen plasma on day 1 and mechanical ventilation and the vasopressor dopamine for 1 day.

On day 2, however, right focal seizure occurred and hypertension (150/90 mmHg) and frequent apnea were noticed. Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) indicated acute infarction in the right fronto-temporo-parietal lobe and basal ganglia in the right middle cerebral artery (MCA) territory, and complete occlusion of the internal carotid artery (Figures 1 and 2A). Lumbar puncture showed clear cerebrospinal fluid without pleocytosis.

On day 3, enlarged, well-demarcated, purplish black areas of hemorrhagic cutaneous necrosis and discoloration of the patient's left toes and fingers were noticed (Figure 3). Prothrombotic status was indicated, based on protein C and protein S deficiency, with serum levels of 30% (normal range, 80–132%) and 88% (normal range, 55–130%), respectively; prothrombin time of 19.7 seconds with international normalized ratio = 1.99; activated partial thromboplastin time of 36.6 seconds; fibrinogen, 141 mg/dL (normal range, 170–410 mg/dL); and antithrombin III, 44% (normal range, 85–118%). Platelet count continuously dropped in the following 3 days from 124,000/μL to 33,000/μL. Transthoracic echocardiography showed no vegetation or embolism. Vancomycin was initiated in addition to cefotaxime due to progression of DIC and sepsis.

On day 5, anisocoria occurred. On fundoscopic examination, whitening and edematous retina with exudate and hemorrhage, as well as signs of the retina folding over the macular area, were found, which were compatible with retinal infarction caused by central retina artery occlusion (CRAO). Furthermore, hypertension (120/80 mmHg) was noted when dopamine was tapered off. In addition, proteinuria and hematuria persisted. Elevated plasma renin level was detected at

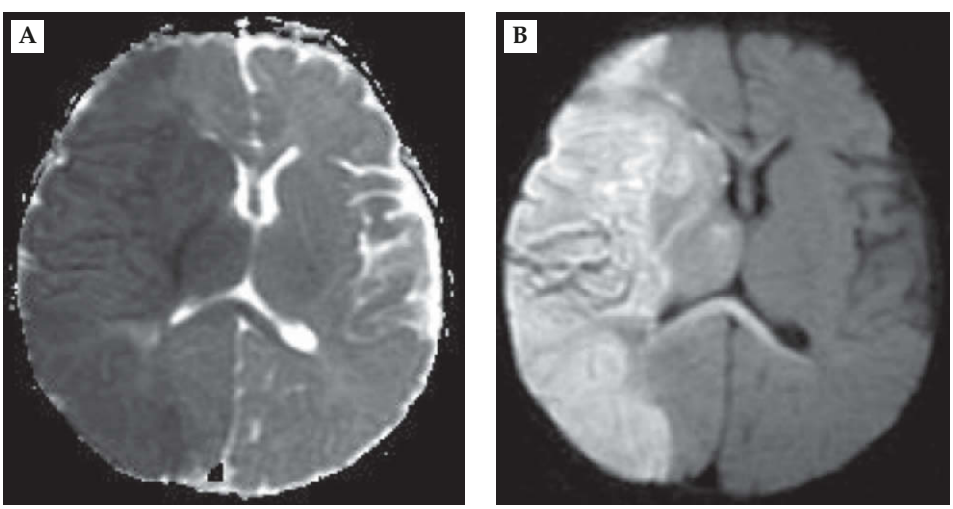

Figure 1. *Age 3 months. (A) Apparent-diffusion-coefficient image and (B) diffusion-weighted axial magnetic resonance image demonstrated low and high signals in the right middle cerebral artery and partial posterior cerebral artery territory, with midline shift to the left side, which suggested acute infarction.*