Successful Transplantation of Organs from a Donor with Bacterial Meningitis Caused by *Streptococcus pneumoniae* 

— A Case Report —

Eunjung Park, M.D., Sang-Cheon Choi, M.D., Youngjoo Lee, M.D.*, Yoonseok Jung, M.D. and Younggi Min, M.D.

Departments of Emergency Medicine, *Anesthesiology and Critical Care Medicine, Ajou University School of Medicine, Suwon, Korea

The number of organs transplanted worldwide is increasing each year, creating the need for increased organ procurement.[1] The number of organ transplantations has been increasing in South Korea.[2,3] The availability of cadaveric organs continues to be the most important factor limiting the number of transplantations performed.[4] Compared to other developed countries, the organ donation rate from brain dead donors in South Korea very low.[2] The scarcity of organs available for transplantation has led to the progressive broadening of criteria for accepting organs from donors who have died due to infections such as endocarditis and bacterial meningitis.[2] We report a case of a patient with bacterial meningitis caused by *Streptococcus pneumoniae*, who met clinical criteria for brain death and whose liver and kidney were transplanted successfully.

**Key Words:** donor selection, meningitis, organ transplantation.

CASE REPORT

A 55-year old man visited the emergency room with loss of consciousness. He had complained of back pain for 10 days, which prompted visits to an orthopedic clinic and pain clinic. During the previous admission, he had received several intravenous injections of ketorolac, acupuncture, and intrathecal injection of opioid. Without any aura, he suddenly lost consciousness and breathing. At the time of the emergency room visit, carotid pulse was not palpated and cardiopulmonary resuscitation (CPR) was started. The initial rhythm was pulseless electrical activity. During CPR, chest compression was continued with minimal interruption. Simultaneous artificial ventilation following the insertion of an endotracheal tube and epinephrine injection following the introduction of an intravenous line was performed. After CPR for 4 minutes, return of spontaneous circulation was detected. Initial vital signs were blood pressure 90/54 mmHg, pulse rate 112 bpm, respiratory rate 18 bpm, and body temperature 36.4°C. His mental status was comatose. Fluid resuscitation and norepinephrine was infused and titrated to optimize blood pressure. Post cardiac arrest care including mechanical ventilation and therapeutic hypothermia was continued. The patient was admitted to the emergency intensive care unit. Initial laboratory results were white blood
cell (WBC) count 33,800/μl, hemoglobin 11.1 g/dl, hematocrit 33.6%, platelet count 325,000/μl, blood urine nitrogen 22.8 mg/μl, creatinine 1.0 mg/μl, sodium 144 mmol/L, potassium 3.5 mmol/L, total bilirubin 1.2 mg/dl, alanine aminotransferase 107 U/L, aspartate aminotransferase 60 U/L, creatine kinase 268 U/L, creatine kinase MB fraction 4.3 μg/L, troponine I <0.006 ng/ml, and S100 0.214 μg/L. Arterial blood gas analysis results were pH 6.955, pCO₂ 92.2 mmHg, pO₂ 122.9 mmHg, and base excess −14.0 mmol/L. Electrical cardiogram showed normal sinus rhythm without any ST segment or T wave abnormality. To search for the reason of cardiac arrest, brain computed tomography angiogram (CTA), transthoracic echocardiography (TTE), and coronary angiography were performed. TTE showed hypokinesia of mid-left-ventricle without concordance with coronary territories and coronary angiography showed normal coronary artery. Brain CTA showed the effacement of cortical sulci with severe ventricular enlargement that was consistent with meningoencephalitis (Fig. 1). To reduce intracranial pressure, an external ventricular drain was inserted.

Cerebrospinal fluid (CSF) analyses revealed protein 447 mg/dl, glucose <10.0 mg/dl, WBC 100/μl (neutrophils 86%, lymphocytes 5%, monocytes 9%), and red blood cell count 490/μl. To manage the meningoencephalitis, ceftriaxone, vancomycin, and ampicillin were administered. A CSF culture was positive for S. pneumoniae while two blood cultures, a urine culture, and sputum culture were negative for any pathogen. On the second hospital day, the mental status was comatose. Post cardiac arrest care of mechanical ventilation, norepinephrine infusion, and therapeutic hypothermia with rewarming phase was continued. The neurological exams including pupil light reflex, corneal reflex, and motor response were lost. Polyuria, hypernatremia (sodium 172 mmol/L) and low urine sodium level showed the development of diabetes insipidus. To control diabetes insipidus, vasopressin was infused and titrated. A brain CT taken at the second hospital day showed diffuse brain swelling with decreased hydrocephalus. Electroencephalogram showed severe diffuse encephalopathy. At the eighth hospital day, brain magnetic resonance imaging showed aggravated meningoencephalitis with hypoxic brain injury (Fig. 2). CSF at the ninth hospital day showed pus and the cellular examination could not be taken due to cellular degeneration. CSF culture did not show any growth of pathogen. Electroencephalogram at the twelfth hospital day showed severe diffuse encephalopathy. At day 24 of hospitalization, he was pronounced brain dead. The family consented to organ donation. Donor’s renal and liv-

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**Fig. 1.** Brain computed tomography angiogram taken at admission shows the effacement of cortical sulci with severe ventricular enlargement that was consistent with meningoencephalitis.

**Fig. 2.** Brain magnetic resonance imaging at the eighth hospital day shows aggravated meningoencephalitis with hypoxic brain injury. T2-weighted image (A) and fluid-attenuated inversion recovery sequence (B) shows hyperintense signals in the cortex of both cerebral hemispheres, basal ganglia and thalamus.