[NM-173] Seizure After Cardiac Transplantation: A Case of Cerebral Hyperperfusion?

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Seizures are a known postoperative phenomenon following cardiac transplantation. Neurologic complications occur in up to 60% of cardiac transplant recipients, with seizures being the most common(3,4). With improving survival rates, morbidity and mortality associated with neurologic sequelae have become increasingly more relevant. Common etiologies of these seizures include cyclosporine related neurotoxicity and cerebral infarction. We present a case of a pediatric patient who developed seizures postoperatively, however, due to the utilization of cerebral oximetry intraoperatively, we argue that his seizures may have been caused by cerebral hyperperfusion.

The patient is a 13 year old male, born with transposition of the great arteries, hypoplastic tricuspid valve, right ventricular hypoplasia, and an interrupted aortic arch. During the year prior to his transplant, his ventricular function and resultant symptoms had worsened. He was listed I-B for transplantation and was noted to have end stage congestive heart failure and was cyanotic at baseline. Cerebral oximetry was utilized for monitoring throughout the case. After transplantation with the donor heart, his cerebral oximetry was noted to increase dramatically though not unexpectedly. Otherwise, the transplantation proceeded without incident.
In the following postoperative course, the patient reported severe headaches and had a seizure. CT imaging revealed diffuse cerebral edema. An external ventricular drain was placed to assist with intracerebral pressure management. The patient eventually required ventriculoperitoneal shunt placement and remains on chronic antiepileptic therapy.

Discussion
Cerebral hyperperfusion is a condition typically described in patients who have undergone repair of severe carotid stenosis. The stenotic carotid vessels chronically limit cerebral perfusion leading to the development of compensatory auto-regulatory mechanisms. With a sudden improvement of flow through the cerebral circulation, the auto-regulation is surmounted and the patient can develop hemorrhage and/or edema which can be devastating.

It is not difficult to surmise that hemodynamic instability during and after cardiac transplantation may result in times of cerebral hypoperfusion that cause infarction. Given advances in monitoring like transcranial doppler and cerebral oximetry, one can now have additional information regarding cerebral flow. This is especially important as improved cardiac output may result in significant increases in cerebral blood flow despite a mostly unchanged systemic blood pressure.

There have been studies demonstrating a correlation between the percentage increase in cardiac index and incidence of neurologic dysfunction after placement of a left ventricle assist device(2). One study shows the effect of cardiac transplantation on cerebral blood flow using transcranial doppler (1). Our case reveals an opportunity for putting the pieces together for the evaluation of cerebral blood flow during cardiac transplantation and the incidence of postoperative neurologic dysfunction.

1. Neurology (2006) 66(1):124-126
2. J Thoracic and Cardiovascular Surgery (2009) 37(4): 1012-1019
3. J Child Neurol (2002) 17(3): 195-199
4. J Neurol (2010) 257:563–568
