A 78-year-old gentleman was found by his son approximately one hour after accidental ingestion of 30-50 ml of 35% hydrogen peroxide; this was taken in error instead of intended gastrograffin in preparation for abdominal CT. The patient was confused, vomiting and was rushed to hospital.

Examination by ICU staff at the time of admission disclosed a fluctuating level of consciousness. Initial examination by the consulting neurologist the day following ingestion showed stupor, a left sixth nerve palsy, absence of left sided movement and bilateral Babinski's. MRI at this point showed multiple foci of restricted diffusion and abnormal T2 signal throughout the anterior and posterior circulations including the right occipital lobe as well as multiple foci in the parietal lobes, right greater than left (Figures 1,2,3). After discussion with the family, level 3 care was instituted (no code, comfort measures only).

However, by the next day, the patient demonstrated dramatic improvement and re-examination by the neurology service five days after ingestion demonstrated a normal neurologic exam, including the patient’s recall of solution ingestion. Investigations at this point showed normal carotid Doppler flow studies, normal cardiac ventricular function, moderate aortic regurgitation and no evidence of an intracardiac shunt, although the echocardiographic study was described as limited. Repeat MRI 2 weeks later showed resolution of most of the previously described ischemic areas, including the right occipital focus (Figure 4).

The patient was discharged home from hospital.

Ingestion of hydrogen peroxide resulting in cerebral infarction has been described in the literature. Typically, this ingestion is in error, but it is sometimes intentional for perceived health benefits. The pediatric population is particularly vulnerable to accidental ingestion. Multiple mechanisms of action have been proposed to account for cerebral infarction. These include hypoxemia as a result of bronchospasm or pulmonary aspiration produced by direct ingestion; undissociated hydrogen peroxide may undergo dissociation in the venous system and right heart with resultant embolization across an existing right to left shunt; the hydrogen peroxide may undergo catalysis only in the arterial supply, then producing embolization. This is similar to iatrogenic air embolism during invasive procedures (ie, arch angiography).

Recovery in case descriptions reported is variable, ranging
from death to residual deficits, with improved outcome following hyperbaric oxygen therapy. Given outcome in described case reports, our patient has shown an excellent course.

REFERENCES