A Contribution to the Controversy over Dimethyl Sulfoxide Toxicity: Anesthesia Monitoring Results in Patients Treated with Onyx Embolization for Intracranial Aneurysms

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LETTER TO THE EDITOR

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A contribution to the controversy over dimethyl sulfoxide toxicity: anesthesia monitoring results in patients treated with Onyx embolization for intracranial aneurysms

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Sir,

I read the article about using Onyx and the monitoring results [1] with great interest. There are some questions and comments concerning the anesthesiologic treatment.

The recommended dose rate of vecuronium for intubation is 0.1 mg/kg. Higher doses result in a much longer duration of relaxation—for example, 86 min after 0.3 mg/kg. Furthermore, a dose of 1 mg/kg may cause heart failure and a much longer recovery time. The induction dose of fentanyl is at least 1 μg/kg, and to achieve a good hemodynamic situation even in patients with subarachnoid hemorrhage (SAH), we use much higher doses (at least 3 μg/kg). It is not very helpful to use high doses of vecuronium to save opioids because the anesthesiologist risks increases in both blood pressure and intracranial pressure (ICP).

Anesthesia was maintained with isoflurane and nitrous oxide. The use of nitrous oxide in the seven patients with SAH is very dangerous because of the increase in ICP; a total intravenous anesthesia technique is recommended for patients with disturbed intracranial compliance. The monitoring of the ICP in these patients is obligatory. Using nitrous oxide risks cerebral ischemia even if the neuroradiologic result is perfect. Nitrous oxide is preferable for the interventional neuroradiologist because of the dilatation of the cerebral vessel; however, many studies have shown an increase in intracranial volume [2].

Keeping the patients "hyperdynamic" is recommended in SAH to prevent a decrease in cerebral perfusion pressure (if "hyperdynamic" means "maintain a preferable cerebral perfusion pressure"). In healthy patients there is no need to reach a "hyperdynamic" state; to reach this goal in patients with SAH, it would be helpful to infuse the fluid before the anesthesia induction. Propofol decreases the peripheral vessel resistance; in our patients with hypertension taking beta-blocking drugs or ACE inhibitors, we infuse 500 ml colloidal fluid before the induction with propofol. Thus, a decrease in cerebral perfusion pressure can be prevented in many patients. Bradycardia is often seen even after anesthesia induction with opioids and propofol; we see it much more often in patients taking beta-blocking drugs. Bradycardia is no sign for a "hypodynamic" situation or hypotension. In patients with ischemic heart disease the prevention of bradycardia can cause myocardial infarction.

The author suggests that all patients need oxygen after treatment because of the DMSO metabolized to dimethyl sulfide. For example, 1–3% of 10 ml DMSO is metabolized to 100–300 mg dimethyl sulfide which is exhaled over

several days (with a peak at 12 h after injection). So the amount of dimethyl sulfide cannot be responsible for a decrease in oxygen saturation immediately following the treatment. All patients have a reduction of functional residual lung capacity during and after general anesthesia which results in low oxygen saturation. After a few minutes to hours of spontaneous breathing, it normalizes.

DMSO has been used in many human therapeutic situations. Some side effects have been reported. Significant pulmonary side effects are pulmonary edema and bronchospasm [3], but neither was seen by the authors. From the anesthesiologic point of view, there is no evidence for frequent pulmonary disturbances when using DMSO for the treatment of intracranial aneurysms.

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