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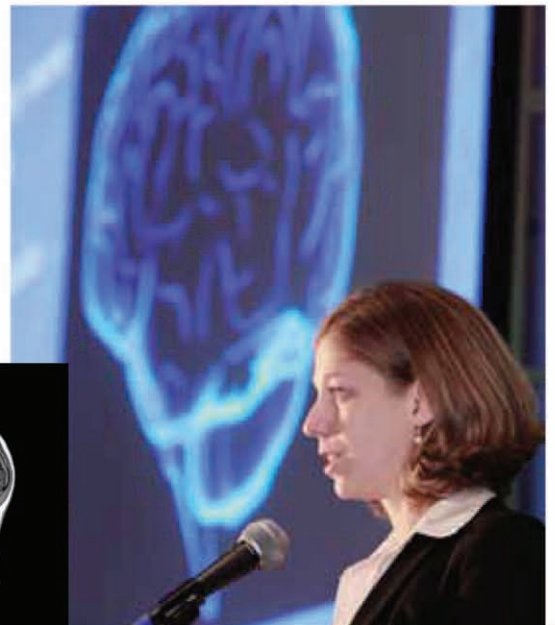
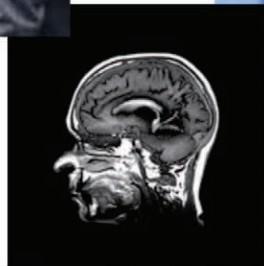
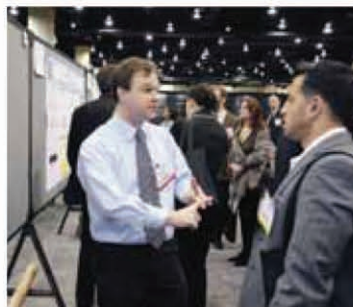
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Abstracts



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patients died: 1 in dosage tier 1 and 1 in dosage tier 2. In both instances death was attributed to aSAH. Delayed cerebral ischemia was present in 2 patients in dose tier 1 and 1 patient in dose tier 2. The odds in favor of good outcome defined as GOS of 1 were: ALISAH dose tier 2 vs. IHASt (all cohort): 3.1 (95%CI: 0.92 - 10.8); ALISAH dose tier 1 vs. IHASt (all cohort): 1.0 (95%CI: 0.4 - 2.6); ALISAH dose tier 2 vs. ALISAH dose tier 1: 3.0 (95%CI: 0.67 - 14.1). **Conclusion:** Although a limited sample size, our data provide preliminary evidence that high-dose HA therapy may be neuroprotective after aSAH. These results have led to the design of a multicenter, randomized, placebo-controlled feasibility study of HA in aSAH—the ALISAH II Phase II Trial. This trial will further evaluate safety and the potential treatment effects of HA. This will be necessary prior to the execution of a Phase III efficacy trial.

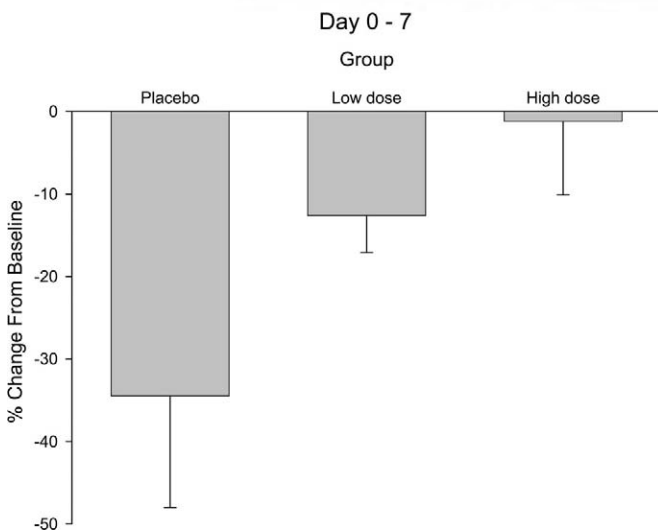
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192

Effect of Site-Specific, Sustained-Release Nimodipine on Vasospasm After Subarachnoid Hemorrhage in Dogs

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Nimodipine is approved for treatment of patients with aneurysmal subarachnoid hemorrhage (SAH). However, it is controversial as to whether nimodipine improved outcome by preventing angiographic vasospasm or by some other mechanism because the dose was low and limited by side effects so there was no effect on vasospasm. Other recognized effects of nimodipine include fibrinolytic activity that might inhibit microthromboembolism after SAH and be another mechanism by which it improves outcome. We tested the hypothesis that high doses of nimodipine delivered in a site-specific, sustained-release formulation would prevent vasospasm in the dog double-hemorrhage model of vasospasm and SAH. Six dogs were randomly allocated to be treated with a placebo or with low (10 mg) or high-dose (30 mg) nimodipine formulated in biodegradable microspheres. Animals underwent baseline angiography and then creation of SAH by injection of blood into the cisterna magna. Placebo or one of the 2 doses of nimodipine was injected into the cisterna magna along with the blood injection. Two days later, the blood injection was repeated. Angiography was repeated 7 and 14 days later. Animals were assessed neurologically every day and blood and cerebrospinal fluid were obtained to measure nimodipine concentrations. Nimodipine decreased angiographic vasospasm in a dose-dependent fashion (Figure, $p = 0.08$, analysis of variance). Seven days after SAH, placebo-treated animals had a $35 \pm 10\%$ reduction in basilar artery diameter compared to $13 \pm 3\%$ in the low-dose and $-1 \pm 6\%$ in the high-dose nimodipine-treated animals. The dogs did not develop systemic side effects such as hypotension or behavior abnormalities, supporting the hypothesis that high doses of nimodipine delivered in a site-specific, sustained-release fashion are safe. Furthermore, no untoward pathological findings were observed in the brains of dogs after 14 days. Site-specific, sustained-release delivery of nimodipine in a biocompatible, biodegradable polymer may be effective at preventing experimental angiographic vasospasm.



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193

Diagnostic Yield of Repeat Catheter Angiography in Patients with Catheter and CT Angiography Negative Subarachnoid Hemorrhage

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Purpose: To determine the yield of repeat catheter angiography for the detection of causative vascular abnormalities in patients with subarachnoid hemorrhage (SAH) who have negative initial catheter and CT angiograms. **Methods:** From January 1st, 2005 until July 31st, 2010, we instituted a prospective protocol in which patients who presented with SAH documented by non-contrast CT (NCCT) or cerebrospinal fluid (CSF) xanthochromia and had negative initial catheter and CT angiograms were also evaluated with repeat catheter angiography 7 days and 3 months after presentation to assess for causative vascular abnormalities. Two neuroradiologists evaluated the NCCTs to determine the pattern of SAH (perimesencephalic or not) with differences in interpretation resolved by consensus. Catheter angiograms were reviewed by experienced interventional neuroradiologists at the time of clinical care. **Results:** 71 patients were included in our study, with a mean age of 53.6 years (median 54 years, range 19-88 years). 45 patients were female (63.4%) and 26 male (36.6%). 68 patients had SAH by NCCT (95.8%) and 3 patients had CSF xanthochromia (4.2%). Among the former, 29 patients had perimesencephalic SAH (42.6%) and 39 had non-perimesencephalic SAH (57.4%, kappa 0.91, 95% CI 0.89-0.93). Repeat catheter angiograms 7 days after presentation were performed in all patients and 3 months after presentation were performed in 38 patients (53.5%). Mean time interval between presentation and the first repeat catheter angiogram was 6.4 days (median 6 days, range 3-12 days), and the second repeat catheter angiogram was 91.9 days (median 89 days, range 35-164 days). The first repeat catheter angiogram demonstrated a causative vascular abnormality in 4 patients (yield of 5.6%), 3 of which had non-perimesencephalic SAH (yield of 7.7%) and 1 had perimesencephalic SAH (yield of 3.4%). The vascular abnormalities were 3 aneurysms and a 2mm arteriovenous malformation at the skull base supplied by the right vertebral artery. Aneurysm locations were 1 right supraclinoid internal carotid artery (blister-like), 1 right superior hypophyseal artery and 1 distal left superior cerebellar artery. Mean aneurysm size was 1.67mm (median 1.5mm, range 1.5-2mm). 3 patients underwent endovascular treatment of the vascular abnormality and 1 patient elected not to undergo any treatment. The second repeat catheter angiograms did not demonstrate any causative vascular abnormalities. No causative abnormalities were found in patients with CSF xanthochromia. **Conclusion:** Repeat catheter angiography performed 7 days after presentation is a valuable adjunct in the evaluation of patients with SAH who have negative initial catheter and CT angiograms, demonstrating a causative vascular abnormality in 5.6% of patients.

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194

A New Generation of Flow-Disruption Device for Endovascular Treatment of Intracranial Aneurysms - Preliminary Clinical and Angiographic Results of a Multicenter Study

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Based on previous in vitro and in vivo experimental studies¹ a new generation of flow disruption device (Surpass FD, Tel Aviv, Israel) was developed and evaluated in a multicenter study for the treatment of intracranial aneurysms. A total of 20 patients were treated (mean age 60 years, range 30-75) for single or multiple aneurysms (n=19) and for a tumor encased internal carotid artery (ICA, n=1). Aneurysms were of blister or fusiform type, large or giant and originated from the cavernous ICA, the superior hypophyseal or the ophthalmic artery, paraclinoid ICA, PcomA or basilar trunk and measured in largest diameter 1.5 - 30mm. To achieve the calculated flow disruption between the parent artery and aneurysm and required for an occlusion, single devices were tailored to local boundary conditions and placed endovascularly in parent arteries and covering the base of aneurysms. Implanted devices measured 3.5-5.3mm in diameter with a length of 20-80mm. Immediate control angiography demonstrated various degree of flow reduction within aneurysm up to a complete flow stagnation. Six-month follow-up angiography available in 4 patients showed a complete/near-complete occlusion in all aneurysms. In 2 patients tortuosity of ICA prevented a successful device deployment with the first generation of delivery system. All perforating arteries covered by the implant remained patent during the follow-up period of up to 15 months and included anterior choroidal artery, ophthalmic artery, PcomA, anterior cerebral artery and lenticulostriates. No stenosis of treated arterial segment was observed. Procedure related thromboembolic complications were seen in 2 patients and resolved in one patient while the other patient continues to have a hemianopia. A third patient experienced a wire perforation during device deployment that led to an ICH necessitating a