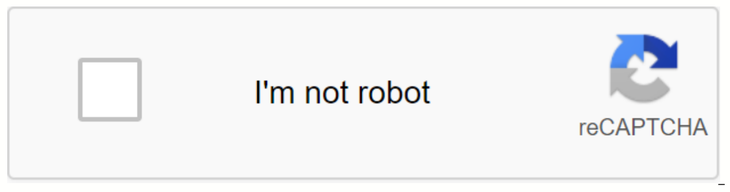

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Management of aneurysmal subarachnoid hemorrhage guidelines

To prevent vasosophism, maintaining normovolemia, normothermia and normal oxygen is paramount. The state of volume should be carefully monitored, avoiding the contraction of volume, which can lead to vasospazus. Oral uimodipine is the most studied calcium channel blocker for the prevention of vasospam after SAH. The American Heart Association/American Stroke Association guidelines recommend using it for this purpose (Class I, evidence level). [32] Calcium channel blockers have been shown to reduce the incidence of ischemic neurological deficits and that plasticine has improved over 3 months after aneurysm SAH. Calcium channel blockers and other antihypertensive medicinal products should be used with caution to avoid the harmful effects of hypotension. [33, 34] The mechanisms of protective action of uimodipine in vasospaz are not proven. However, it seems that plasticine can prevent ischemic complications of vasospazmas neuroprotective effect by blocking the influx of calcium into damaged neurons. In May 2013, the U.S. Food and Drug Administration (FDA) approved a new oral solution for nimomipine (Nymalize) for the treatment of patients with SAH. Nimoipine was previously only available as a liquid-filled gel capsule. Intravenous administration of uyoyodipin for oral administration has been reported to cause death, cardiac arrest, severe low blood pressure and other cardiac complications. The oral form may reduce or remove the drug inadvertently intravenously. [35] Some data suggest that subarachnoid removal of clots achieved by recombinant tissue plasminogen activator (rTPA) intracisternal injections may significantly reduce the risk of vasospose. This is done after the clipping of the aneurysm. Thrombolytic therapy is associated with a theoretical risk of intracranial bleeding, and although preliminary studies are favorable, rigorous clinical trials are needed to determine the safety and efficacy of this approach. Intracisteral antioxidants and anti-inflammatory drugs are unclear. Aspiration and irrigation of the subarachnoid clot during aneurysm cutting usually leads to the removal of a suboptimal clot and is associated with a high risk of iatrogenic trauma on the pial surfaces and small vessels. Intraoperative washing of sodium chloride for the purification of blood products from the subarachnoid space can be beneficial, but its effectiveness remains unproven. Some authors suggest that early CSF drainage through ventricular drainage may reduce the frequency of vasospazm. This intervention is carried out after the aneurysm is secured. Be careful to avoid fast or excessively aggressive CSF drainage, which can precipitate an aneurysm reconnection. One author suggests draining CSF if the intracranial pressure exceeds 20 mm Drainage should be set to a height of 20 mmHg to avoid excessive decrease in ICP. Statin therapy has been proposed as a means to prevent vasospazmas and delayed cerebral ischemia. Statins can improve the brain's vasomotor reactivity through cholesterol-dependent and cholesterol-dependent mechanisms. [36, 37] The use of statins in SAH is controversial. Several small studies have shown promise. Two meta-analyses showed contradictory results. Sillberg et al concluded that statin treatment reduces vasospazus and cerebral ischemia [38], while Vergouwen and others found no benefit from statin therapy. [39] As long as no further data is available, the use of statins is generally not recommended. [40] Simvastatin, a multicentre, randomised controlled clinical trial of subarachnoid bleeding (STASH), will investigate the effects of simvastatin 40 mg in SAH patients. The pilot is currently recruiting participants. The planned sample size is 1,600 patients who should be powerful enough to respond to the controversial SAH therapy of surrounding statins. Treatment of symptomatic vasospasm has traditionally been associated with induction of hypertension, hypervolemia and hemodilution or triple treatment of H. This treatment should be prescribed to patients with aneurysms, guaranteed surgically cut or endovascularly, to reduce the risk of repeated bleeding. The efficacy of triple H treatment is still under consideration. A review of controlled studies did not show a positive effect of triple H treatment or its components on increased cerebral circulation. [41] If necessary, aggressive hypertensive therapy with inotropes and vasopressors (e.g. dobutamine) may be initiated. Hypervolemia can be achieved by the use of packaged erythrocytes, isotonic crystalloids, colloidal and albumin infusions in combination with vasopressin injection. Corticosteroids may have some benefits; however, such treatment remains controversial. Hemodilution or transfusion is used to maintain hematocrit by 30-35% in order to optimize the viscosity of the blood and the supply of oxygen. To start triple H therapy, a pulmonary arterial catheter should be performed to direct volume expansion and inotropic or vasopressor therapy. Central venous pressure (CVP) should be maintained at 10-12 mmHg. Pulmonary artery wedge pressure (PAWP) must be maintained at 14-20 mm Hg. Transluminal balloon angioplasty recommended to treat vasospazmas after unsuccessful routine treatment. In one study, neurological results were reported in 70% of patients with symptomatic vasospamas after transluminal angioplasty. A series of cases reports have shown that angioplasty is effective in treating vasospazmas of large proximal vessels. [42] Angioplasty is not effective in the treatment of direct vascular spasm, i.e. more distal vessels; however, distal blood flow may increase due to diameter of the vessel. Possible complications of angioplasty include rupture, dissection or occlusion of blood vessels, as well as intracerebral bleeding. The injection of papaverine into the artery has been reported to improve, but further data should be recommended prior to recommended. The positive effect of papaverine infusion seems short-lived compared to angioplasty. Magnesium is a neuroprotective agent that acts as an antagonist of the N-methyl-D-aspartate (NMDA) receptors and calcium channel blockers. It has been used to reduce cerebral ischemic events in SAH patients. Magnesium levels should be closely monitored. Studies of magnesium treatment SAH gave different results. A small, randomized, placebo-controlled study of Westermaier et al showed that maintaining serum magnesium levels of 2-2.5 mmol/l reduced the occurrence of cerebral ischaemic events after aneurysmal SAH. [43] A meta-analysis showed that magnesium reduced the risk of delayed ischemia and poor aneurysm SAH score. [44] However, a larger multicentre phase III Study of Wong et al did not significantly find a difference in the incidence of magnesium IV or placebo in 6 months. [45] Several new medicines for use in SAH have been studied, in particular to improve vasosopazus. In a randomised, double-blind, placebo-controlled trial study, methylprednisolone did not reduce vasospazma but improved functional results. [46] Tyrilazad, 21st negliukocorticoid, did not drink. [47] Intraarter colforzine is being investigated to improve vasospaz. [48] 1.Macdonald RL, Schweizer TA. Spontaneous subarachnoid bleeding. Lancet, I'm here. 2017;389:655–66.Article PubMed Google Scholar 2.Lawton MT, Vates GE. 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J Neurosurgeon Fiction. prospective cohort study in SAH patients with ventricular catheter for cerebrospinal fluid drainage (1) 33 °C with endovascular cooling device (2) Barbiturate coma N = 7 Unwritten information N = 8 There was no significant difference in neurological outcomes (GOS > 3) after 1 year (42, 9 and 50.0%). Anei R 2010 [40] Single centre pre-study Poorly grade SAH patients (WFNS scale > (3) Induction within 24 hours after bleeding (2) at 34 °C for 48 h with cooling blanket (3) Rewarming 1 °C/24 h N = 16 Described in detail N = 19 There was no significant difference in neurological results after 3 months. Badjatia N 2010 [41] Harmonised controlled analysis of single-centre, prospective cohort SAH patients with fever-resistant fever at 37 °C for 14 days N = 40 Oral acetaminophenon with or without water circulating blanket N = 80 TTM was associated with better neurological results after 12 months (79 vs. 54%). Kuramatsu JB 2015 [42] Harmonised controlled analysis of single-centre, prospective cohort SAH patients (3 > and WFNS grade > and WFNS scales > (3) Induction within 48 hours after bleeding (2) 35 °C for 7 days with endovascular cooling device (3) 0.5°C/24 h N = 12 Intravenous paracetamol N = 24 TTM patients had significantly lower VBP rates (50.0 vs. 84.5%) and a tendency to have a better functional result (mRS < 3) at 6 months (66.7 vs. 33.3%). hunt and Hess grade and modified Fisher scale > 2) (1) Induction as soon as possible after ruptured aneurysm treatment (2) 34.5 °C 48 h when the aneurysmic treatment device or surface cooling device has been discontinued for 48 h (3 1°C/24 h to 36.5°C N = 11 Undesigned Details N = 11 There were no significant differences in DCI cases (36.3 vs. 45.6%) neurological results (mRS < 3) in two groups (27.3% vs. 9.1%). SAH subarachnoid haemorrhage, TTM target temperature management, WFNS World Neurosurgery Society Federation, DCI Delayed Brain Ischemia, MRS Modified Rating Scale Score, GOS Glasgow Score Scale