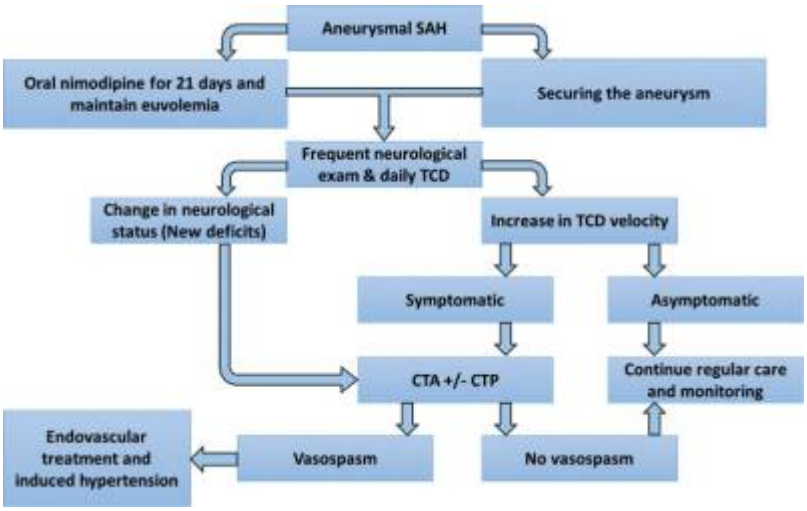
Nimodipine for aneurysmal subarachnoid hemorrhage



see also Aneurysmal subarachnoid hemorrhage treatment

Prophylaxis with the calcium channel blocker nimodipine does not affect the cerebral vasculature but lessens poor outcomes by one-third [1].

Nimodipine 60 mg every four hours is administered to all patients with aneurysmal subarachnoid hemorrhage, ideally within four days of SAH. The typical dose is 60 mg every four hours by mouth or nasogastric tube. Nimodipine must be given orally or by nasogastric tube because inadvertent intravenous administration has been associated with serious adverse events, including death. Treatment is continued for 21 days.

The calcium channel blocker nimodipine was initially studied in patients with SAH as a means to prevent vasospasm. However, despite the vasodilatory effects of nimodipine on cerebral vessels, there is no convincing evidence that nimodipine affects the incidence of either angiographic or symptomatic vasospasm [2] [3] [4] [5] [6] [7].

Nevertheless, nimodipine has been demonstrated to improve subarachnoid hemorrhage outcomes and is the standard of care in these patients [8] [9] [10] [11] [12] [13] [14].

Nimodipine can cause arterial hypotension requiring either a dosage reduction or its discontinuation. Aim of a study of Kieninger et al., from the University Hospital Regensburg, was to examine the effect of different nimodipine formulations on arterial blood pressure in aneurysmal or perimesencephalic SAH patients and to measure the plasma levels after both, peroral administration as tablet or solution and IA infusion.

In a prospective setting, over a 1-year observation period, data on the course of arterial blood pressure and nimodipine dosage were collected for 38 patients undergoing treatment for aneurysmal or perimesencephalic SAH in an intensive care unit. In addition, plasma concentrations of nimodipine

were measured by liquid chromatography-tandem mass spectrometry.

The intended full dose of 60 mg of nimodipine given orally every 4 h could only be administered on 57.2% of the examined days. Ninety-seven episodes of relevant arterial hypotension probably caused by the administration of nimodipine were observed within the first 14 days of treatment. Drops in blood pressure occurred about three times as often after administration of nimodipine as oral solution than as tablet. However, there were no differences in nimodipine plasma levels between the two formulations. In patients suffering from higher-grade SAH, arterial hypotension and consequent dosage reduction or discontinuation of nimodipine were more frequent than in patients with lower-grade SAH. Plasma concentrations of nimodipine during CIAN did not exceed those achieved by oral administration.

Dosage reduction or discontinuation of oral nimodipine is often necessary in patients with higher-grade SAH. Oral nimodipine solutions cause drops in blood pressure more frequently than tablets. Intra-arterial infusion rates of less than 1 mg/h result in venous plasma concentrations of nimodipine similar to those observed after oral application of 60 mg every 4 h [15].

References

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