Nicardipine

- Chemical Stability Testing of Solutions for Intraventricular Irrigations via IRRAflow Ventricular Drain System
- A posterior reversible encephalopathy syndrome in a young patient with goodpasture's disease
- Validation of a reverse phase high-performance liquid chromatography method for the detection of major components and related substances in nicardipine hydrochloride injection
- Intrathecal nicardipine for symptomatic, refractory vasospasm treatment in pediatric patients: a case series
- Effects of antihypertensive drugs on the metabolism of mobocertinib in rats both in vitro and in vivo
- A PBPK modeling approach for personalized dose optimization of nicardipine in renal and hepatic dysfunction
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- A case of abnormal hypertension and Takotsubo syndrome caused by adrenal hemostasis using an electric scalpel: a case report

Nicardipine was approved by the FDA in December 1988. The patent for both Cardene and Cardene SR expired in October 1995.

Nicardipine hydrochloride (Cardene) belongs to the class of calcium channel blockers.

It is available in oral and intravenous formulations.

see Intraventricular nicardipine.

Ampules contain 25 mg and must be diluted before.

Does not require arterial line. Does not produce intracranial hypertension. Does not reduce heart rate [1)].

May be used in conjunction with e.g. labetalol or esmolol if that is desired.

Its mechanism of action and clinical effects closely resemble those of nifedipine and the other dihydropyridines (amlodipine, felodipine), except that nicardipine is more selective for cerebral and coronary blood vessels.

Furthermore, nicardipine does not intrinsically decrease myocardial contractility and may be useful in the management of congestive heart failure. Nicardipine also has a longer half-life than nifedipine. It has been used in percutaneous coronary intervention.

Indications

Intravenous nicardipine is commonly used for blood pressure reduction in patients with acute stroke.

Endovascular approaches with balloon angioplasty and intra-arterial nimodipine, nicardipine, and milrinone have shown consistent benefits [2)].

Nicardipine for aneurysmal subarachnoid hemorrhage

see Nicardipine for aneurysmal subarachnoid hemorrhage.

Side effects

Headache

Nausea

Hypotension

Reflex tachycardia.

Phlebitis is significantly associated with administration of a maximum concentration of nicardipine greater than 130 µg/mL [3].

Intravenous nicardipine-related phlebitis was retrospectively analyzed. From July 2015, a simple proposition was made to dilute maximum intravenous nicardipine concentration to lower than 130 µg/mL. The maximum intravenous nicardipine concentration and the incidence of phlebitis were compared between patients treated from July 2014 to June 2015 (preproposition group) and patients treated from July 2015 to June 2016 (postproposition group).

A total of 300 patients (preproposition group, 138; postproposition group, 162) were included. The postproposition group demonstrated significantly lower maximum intravenous nicardipine concentration (in µg/mL, 76.9, 47.6-104.5 versus 130.4, 69.8-230.8; $P < .001$) and incidence of phlebitis (9.9%, 16/162 vs. 30%, 42/138; $P < .001$) than the preproposition group. Multivariable logistic regression analysis revealed that the maximum intravenous nicardipine concentration lower than 130 µg/mL (odds ratio [OR] .15; 95% confidence interval [CI] .06-.35; $P < .001$) and National Institutes of Health Stroke Scale on admission (OR .95; 95% CI .91-.99; $P = .007$) were the statistically significant independent factors for phlebitis, which indicated the usefulness of the proposition to dilute maximum intravenous nicardipine concentration to lower than 130 µg/mL.

The simple and appropriate proposition about nicardipine administration lowered maximum nicardipine concentration and reduced the incidence of nicardipine-related phlebitis in patients with acute stroke [4].

Dosing

IV: 5 mg/hr by slow infusion (50 mL/hr) initially; may be increased by 2.5 mg/hr every 15 minutes; not to exceed 15 mg/hr.

Off label 10 mg/hr may be used in situations where urgent reduction of arterial hypertension is needed.

Decrease to 3 mg/hr once control is achieved.

Effects on cerebrovascular hemodynamics

Few studies have described its effects on cerebrovascular hemodynamics as measured by transcranial Doppler (TCD) waveform analysis and pulsatility index (PI).

Lahiri et al., report examples of a consistent but paradoxical finding associated with nicardipine that suggests intracranial vasoconstriction, contrary to what is expected from a vasodilator.

The data presented are from a convenience sample of patients who underwent TCD monitoring before, after, or during nicardipine administration. In each case, TCD waveform morphologies and PIs were compared.

The TCD waveforms during nicardipine infusion are characterized by a prominent systolic peak and dicrotic notch. Systolic deceleration was more pronounced and PIs were significantly elevated in patients who were on nicardipine ($p < 0.001$). This finding was not evident when patients were not on nicardipine.

This study provides the first evidence of paradoxical intracranial vasoconstriction associated with intravenous nicardipine. In the authors' experience, this finding is consistently encountered in the vast majority of patients who are treated with intravenous nicardipine, and is contradictory to what is expected from a vasodilator. Future studies are needed to confirm this finding in larger populations and diverse clinical settings and to examine mechanisms that explain this phenomenon [5].

1)
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