Enteral Nimodipine in Aneurysmal Subarachnoid Hemorrhage: Real-World Application and Challenges

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ABSTRACT

Background: Guidelines recommend nimodipine as the standard of care for patients with aneurysmal subarachnoid hemorrhage (aSAH). Compared with placebo, this agent has been shown to reduce death and dependency on others for activities of daily living. However, retrospective data suggest that patients may not receive full treatment with nimodipine.

Objectives: The primary objective was to determine the proportion of patients with aSAH admitted to an intensive care unit (ICU) or high-acuity unit (HAU) at a tertiary referral hospital who received the guideline-recommended dose and duration of nimodipine. A secondary objective was to describe barriers to receiving full treatment.

Methods: This retrospective chart review involved a convenience sample of 100 patients with aSAH who were admitted to the ICU or HAU of a tertiary referral hospital between January 1, 2012, and August 31, 2022. The analysis was based on descriptive statistics.

Results: Of the 100 patients with aSAH admitted to the ICU or HAU, 1 (1%) received the guideline-recommended dose and duration of nimodipine. Ninety-five (95%) of the patients experienced a delay to initiation, mainly due to transfer from another hospital (n = 45, 47%) and/or lack of a safe enteral route (n = 62, 65%). Sixty-six (66%) of the patients received alternative dosing, most because their blood pressure was below target (n = 16, 24%) or because of vasospasm requiring a higher blood pressure target (n = 22, 33%). A total of 99 patients (99%) had early discontinuation and/or treatment interruption of nimodipine; reasons included vasospasm requiring a higher blood pressure target (n = 12, 12%) and nimodipine not being continued on transfer or discharge (n = 14, 14%).

Conclusions: Most of the patients in this study did not receive the full course of nimodipine therapy due to multiple barriers. Pharmacists can play a role in optimizing treatment by educating staff at transferring sites about timely initiation of therapy, reconciling medications on transfer or discharge, and mitigating interactions with concomitant medications.

Keywords: aneurysmal subarachnoid hemorrhage, cerebral vasospasm, nimodipine, calcium channel blocker

RÉSUMÉ

Contexte : Les lignes directrices recommandent la nimodipine comme la norme de soins pour les patients atteints d'une hémorragie sous-arachnoïdienne anévrismale (HSA). Il a été démontré que ce médicament réduisait la mortalité et la dépendance à l'égard d'autrui pour les activités de la vie quotidienne, par rapport au placebo. Cependant, des données rétrospectives indiquent que les patients pourraient ne pas recevoir le traitement complet à base de nimodipine, tel que prescrit.

Objectifs : L'objectif principal consistait à déterminer la part de patients atteints d'une HSA admis en unité de soins intensifs (USI) ou en unité de soins de haute acuité (USHA) dans un hôpital de référence tertiaire ayant reçu la dose de nimodipine recommandée par les lignes directrices pendant la durée du traitement également recommandée. L'objectif secondaire consistait quant à lui à décrire les obstacles empêchant le patient de recevoir le traitement complet, tel que prescrit.

Méthodologie : Cette étude rétrospective des dossiers portait sur un échantillon de commodité de 100 patients atteints d'une HSA, admis en USI ou en USHA d'un hôpital de référence tertiaire entre le 1er janvier 2012 et le 31 août 2022. L'analyse se basait sur des statistiques descriptives.

Résultats : Sur les 100 patients atteints d'une HSA admis en USI ou en USHA, un seul (1 %) a reçu la dose de nimodipine recommandée par les lignes directrices pendant la durée de traitement recommandée. L'initiation du traitement était retardée chez 95 patients (95 %), les raisons principales étant le transfert depuis un autre hôpital (n = 45, 47 %) et l'absence d'une voie entérale sécuritaire (n = 62, 65 %). Soixante-six patients (66 %) ont reçu un autre dosage, soit parce que la valeur de leur tension artérielle était inférieure à la cible (n = 16, 24 %), soit en raison d'un vasospasme nécessitant une valeur cible de tension artérielle plus élevée (n = 22, 33 %). Au total, 99 patients (99 %) ont vu l'arrêt précoce ou l'interruption de leur traitement par nimodipine; les raisons comprenaient un vasospasme nécessitant une valeur cible de tension plus élevée (n = 12, 12 %) et la non-continuité de la nimodipine lors du transfert ou du congé de l'hôpital (n = 14, 14 %).

Conclusions : La plupart des patients de cette étude n'ont pas reçu le traitement complet à base de nimodipine en raison de multiples obstacles. Les pharmaciens peuvent jouer un rôle pour optimiser le traitement en sensibilisant le personnel des établissements de transfert à l'initiation rapide de la thérapie, en conciliant les médicaments lors du transfert ou du congé et en atténuant les interactions avec les médicaments concomitants.

Mots-clés : hémorragie sous-arachnoïdienne anévrismale, vasospasme cérébral, nimodipine, bloqueur des canaux calciques

INTRODUCTION

Aneurysmal subarachnoid hemorrhage (aSAH) is a subset of stroke that results in bleeding into the subarachnoid space secondary to a ruptured aneurysm.[1] In North America, the mortality rate for patients with aSAH has been estimated at 32%,[2] and approximately 20% of survivors will experience global cognitive impairment.[3] Several possible complications may ensue from aSAH, including cerebral vasospasm, defined as transient narrowing of angiographically visible arteries, for which peak incidence occurs 7 to 10 days after aneurysm rupture.[1] Delayed cerebral ischemia may also arise, whereby patients exhibit neurological deficit secondary to a reduction in cerebral blood flow.[1] Neurological deficits occur in fewer than half of patients with cerebral vasospasm, in cerebral areas not supplied by blood vessels visibly undergoing spasm, which supports the view that the pathogenesis of delayed cerebral ischemia is likely multifactorial.[1]

Current guidelines recommend enteral administration of nimodipine, a dihydropyridine calcium channel blocker, for 21 days after aSAH.[4,5] Nimodipine has multiple proposed mechanisms of benefit, including reducing cerebral artery vasoconstriction, preventing neuronal apoptosis, and opposing the formation of micro-emboli.[6-8] In a 2007 Cochrane review, Mees and others[9] determined that oral nimodipine reduced poor outcomes (defined as death or dependence on help for activities of daily living) and secondary ischemia relative to placebo.

Retrospective data have suggested that many patients may not receive nimodipine for the recommended 21 days because of hypotension.[10,11] More specifically, Barfejani and others[10] reported that only 33% of patients with aSAH received the recommended dose and duration of nimodipine, with hypotension cited as one of the most common reasons for dose reduction and early discontinuation. Similarly, Sandow and others[11] found that 44% of patients with aSAH received the full daily dose of nimodipine, with 29% of patients requiring dose reduction and 28% requiring discontinuation due to hypotension. The current prescribing practices and drug utilization for nimodipine at our institution have not been reviewed.

Our primary objective was to determine the proportion of patients with aSAH who received the guideline-recommended dose and duration of nimodipine therapy, specifically 60 mg enterally every 4 hours for a period of 21 days. We also aimed to describe factors limiting nimodipine use and to explore how pharmacists may play a role in promoting adherence.

METHODS

A retrospective chart review was conducted at a large tertiary care referral centre in British Columbia, Canada. The study was approved by the University of British Columbia Clinical Research Ethics Board and Fraser Health Authority Research Ethics Board.

A list of potential participants who received nimodipine between January 1, 2012, and August 31, 2022, was created in Excel spreadsheet software (Microsoft). Patients were identified using the *International Statistical Classification of Diseases and Related Health Problems, 10th Revision* (ICD-10) code for nontraumatic subarachnoid hemorrhage, as documented in patient charts accessed through the electronic medical records system (Meditech Client/Server, release 5.67, Medical Information Technology Inc). Potential participants were screened for study inclusion and were included in the analysis if they were 18 years of age or older, had been admitted to the intensive care unit (ICU) or high-acuity unit (HAU), and had a diagnosis of aSAH confirmed with either noncontrast computed tomography (CT), lumbar puncture, CT angiography, or magnetic resonance imaging (MRI). Exclusion criteria were subarachnoid hemorrhage due to causes other than aneurysm, admission to the ICU or HAU more than 21 days after onset of aSAH, and death within 72 hours of symptom onset. We targeted a convenience sample of 100 patients.

Patients who received nimodipine for a period of 21 days and received a total cumulative dose of 7560 mg without any dose or interval changes were considered to have received the guideline-recommended dose and duration of therapy (primary outcome); this value is presented as a proportion.

We determined the median time (in hours, with the associated range) from time of diagnosis to initiation of nimodipine. The time of diagnosis was defined as the earliest confirmation of aSAH by noncontrast CT, lumbar puncture, CT angiography, or MRI, and the initiation of nimodipine was defined as the first dose given, as recorded in the nursing medication administration record (MAR). We also determined the median time from diagnosis to surgical intervention, as well as the median durations of hospitalization and ICU or HAU stay.

We determined the median times from nimodipine initiation to the first dose or interval change, the first treatment interruption, and discontinuation. The first treatment interruption was defined as a hold order or a discontinuation followed by reinitiation, as recorded in the nursing MAR. Discontinuation was defined as the last nimodipine dose on the nursing MAR without a subsequent restart.

We determined the proportions of patients who experienced a delay in initiation, did not receive prescribed nimodipine, required a change in dose or interval, required treatment interruption, had treatment discontinuation earlier than 21 days, or died. A delay in initiation was defined as a delay of more than 6 hours from the time of diagnosis. For patients who were discharged sooner than 21 days after the onset of aSAH, discharge prescriptions were used to determine whether they received a full course of nimodipine. Furthermore, we determined the proportions of patients

who experienced cerebral ischemia confirmed on imaging, cerebral vasospasm confirmed on imaging, and symptomatic vasospasm. Symptomatic vasospasm was defined as clinically determined neurological deficits or decreased level of consciousness presumed to be due to vasospasm. We described factors potentially contributing to patients not receiving a prescription for nimodipine, not receiving prescribed nimodipine, requiring treatment interruption or a change in dose or interval, and having early discontinuation.

For patients who experienced challenges with blood pressure that necessitated dose or interval change, treatment interruption, or early discontinuation, we also determined the proportion who were using vasopressors, the median number of vasopressor agents used, and the dose of vasopressors at the time of nimodipine change. We also determined the median number of blood pressure–lowering medications being used within 24 hours preceding a change in or discontinuation of nimodipine, as recorded in the nursing MAR.

We collected baseline characteristics, including World Federation of Neurosurgical Societies (WFNS) grade on admission. The WFNS grade is recommended by the guideline of the American Heart Association/American Stroke Association (AHA/ASA)[4] to determine initial clinical severity and to predict outcomes.

RESULTS

A total of 636 patients with the ICD-10 code for nontraumatic subarachnoid hemorrhage were admitted to our site from January 1, 2012, to August 31, 2022. Of these, 100 patients were selected, by means of a random number system, for inclusion in our study. The median age was 59 (range 30–86) years, and 62 (62%) were female. Thirty-eight patients (38%) had an initial WFNS grade of I, and 35 (35%) presented with an initial WFNS grade of V, the most severe presentation. Common symptoms at presentation included loss or lowered level of consciousness (54%) and headache (48%). Seventy patients (70%) underwent coiling as the means of surgical intervention. Table 1 describes baseline characteristics in more detail.

One patient (1%) received the guideline-recommended dose and duration of nimodipine. Six patients (6%) received a cumulative nimodipine dose of more than and 93 (93%) a cumulative dose of less than 7560 mg, the prespecified threshold. The median cumulative dose of nimodipine was 3810 (range 0–8010) mg. The median time to nimodipine initiation was 22.9 (range 0–313) hours, and the median duration of nimodipine treatment was 11.6 (range 0–25.2) days.

The median time from diagnosis to surgical intervention was 35.4 (range 0–353.0) hours. The median times to first dose or interval change and treatment interruption were 3.1 (range 0–13.4) days and 5.0 (range 0.2–15.1) days, respectively. The median durations of the critical care and hospital stays were 10.7 (range 0.4–52.1) days and 19.7 (range 1.1–138.8) days, respectively. The median time to nimodipine discontinuation was 11.5 (range 0–25) days.

Among the 99 patients who did not receive the recommended course of nimodipine treatment, barriers were categorized as follows: delayed initiation (n = 95, 96%), receipt of alternative dosing regimens (n = 66, 67%), treatment interruption (n = 13, 13%), and early discontinuation (n = 86, 87%) (Table 2).

Among the 95 patients for whom nimodipine initiation was delayed by at least 6 hours, the reasons were transfer from another hospital (47%) and lack of a safe enteral route (65%). Among the 66 patients for whom alternative dosing strategies were implemented, the main rationales were to mitigate vasospasm requiring a higher blood pressure target (33%) and below-target blood pressure (24%). Among the 99 patients with early discontinuation or treatment interruption, justifications included nimodipine not being continued on transfer or discharge (14%) and vasospasm requiring a higher blood pressure target (12%). Table 2

TABLE 1. Baseline Characteristics of Patients

Characteristic	No. (%) of Patients (n = 100)
WFNS grade	
I	38 (38)
II	6 (6)
III	6 (6)
IV	11 (11)
V	35 (35)
Insufficient information to be calculated	4 (4)
Symptoms during onset[a]	
Loss of consciousness, lowered level of consciousness	54 (54)
Headache	48 (48)
Nausea/vomiting	35 (35)
Sentinel headache	7 (7)
Impaired motor, verbal skills	7 (7)
Modality to confirm diagnosis	
CT angiography	89 (89)
CT	10 (10)
LP	1 (1)
MRI	0 (0)
Type of surgical intervention	
Coiling	70 (70)
Clipping	8 (8)
Coiling with stenting	10 (10)
Coiling and clipping	1 (1)
Incomplete charts	2 (2)
No surgical intervention	9 (9)

CT = computed tomography, LP = lumbar puncture, MRI = magnetic resonance imaging, WFNS = World Federation of Neurosurgical Societies.
[a]Sum of proportions is greater than 100 because some patients presented with multiple symptoms.

TABLE 2. Barriers to Nimodipine Treatment (Identified Reasons)

Reason	No. (%) of Patients
For delay in initiation[a]	*n* = 95
Transfer from another hospital	45 (47)
Enteral route unavailable	62 (65)
Prescriber omission/error	8 (8)
Blood pressure below target	2 (2)
Allergy to nimodipine	0 (0)
Reason not documented	5 (5)
For alternative dosing	*n* = 66
Vasospasm requiring higher blood pressure target	22 (33)
Blood pressure below target	16 (24)
Liver cirrhosis	0 (0)
Adverse effects	0 (0)
Reason not documented	28 (42)
For early discontinuation or treatment interruption	*n* = 99
Vasospasm requiring higher blood pressure target	12 (12)
Blood pressure below target	4 (4)
Deteriorating condition	7 (7)
Death	24 (24)
Medication not continued during transfer or discharge	14 (14)
Adverse effects	0 (0)
Reason not documented	38 (38)

[a]Proportions do not sum to 100 because some patients had multiple reasons for delays in initiation.

describes in more detail the reasons for delay in initiation, alternative dosing, and early discontinuation and treatment interruption.

In terms of complications, imaging showed that 50 patients (50%) experienced cerebral ischemia and 44 patients (44%) experienced vasospasm; in addition, 36 (36%) experienced symptomatic vasospasm, and 44 (44%) ultimately died. Patients received a median of 2 (range 0–8) concomitant medications with blood pressure–lowering effects. Overall, 28 of the 100 patients received antihypertensives, 49 received analgesics, 39 received sedatives, and 9 received neuroleptics. The median number of vasopressors used was 1 (range 1–3); for this group of medications, 47 patients received norepinephrine, 8 received vasopressin, 5 received milrinone, and 1 received dobutamine.

DISCUSSION

Of the 100 patients with aSAH admitted to the critical care areas at our institution, 93 received less than the guideline-recommended dose and duration of nimodipine. The recommendations for nimodipine therapy are largely based on a review conducted by Mees and others,[9] who showed that nimodipine reduced the composite "poor outcome" of death or dependence on help for activities of daily living and secondary ischemia relative to placebo. The 4 trials that were analyzed for this outcome studied nimodipine for a duration of 21 days, but multiple limitations, including significant heterogeneity among the included studies,[9] could hinder generalizability to our study population. In particular, 3 of the 4 trials excluded patients with renal, cardiac, or hepatic insufficiency.[12-15] Unlike the majority of patients in our study, none of the studies in the Cochrane review included patients who underwent endovascular coiling.[12-15]

In the current study, the median treatment duration of 11.6 days may arguably still provide some protection against cerebral vasospasm, given that the incidence of vasospasm peaks at 7 to 10 days, should that be a contributing mechanism.[1] In contrast, in a retrospective analysis of 170 patients admitted to the Department of Neurosurgery at a university hospital, Hernández-Durán and others[16] found that nimodipine interruption was correlated with greater incidence of delayed cerebral ischemia ($\rho = 0.431$, $p < 0.001$). Only 44% of patients in the current study experienced vasospasm demonstrated on imaging compared with 70% of patients in the study by Lawton and Vates.[1] However, our approximation of imaging-confirmed vasospasm may be an underestimate, as our institution does not employ CT as part of routine vasospasm screening, and the timing of imaging depends on the availability of scanners and staffing. The mortality rate in our study was 44%, higher than the North American incidence of 32% reported previously,[2] possibly because our study took place in a critical care setting.

The most commonly reported reasons for a delay in initiation were lack of a safe enteral route and transfer from another hospital. Prioritizing early confirmation of enteral access and improving communication with and education for referring hospital sites about the importance of timely nimodipine therapy may help to expedite nimodipine initiation. Although it is possible to administer 21 days of nimodipine treatment despite delay of initiation, prior studies have suggested that early and unaltered nimodipine therapy is critical for patients with aSAH.[4,5,10] Of the patients with early discontinuation of nimodipine, a substantial group (14%) did not have nimodipine continued on discharge or transfer. Pharmacist-led medication reconciliation might improve continuity of nimodipine therapy and has been shown to reduce medication errors during transfers and discharge.[17] About a quarter of the patients received an alternative nimodipine dosing regimen because their blood pressure was below target. However, patients in this study also received a median of 2 other medications with the potential to lower blood pressure. Clinical pharmacists can play a pivotal role in mitigating these interactions by optimizing medication management without compromising nimodipine therapy.[18]

Vasospasm requiring a higher blood pressure target was a common reason for alternative dosing and early discontinuation or treatment interruption. Upon onset of vasospasm, the AHA/ASA guideline recommends induction of hypertension, unless patient hemodynamics preclude this approach.[4] The requirement for a higher blood pressure target may present a challenge to continuing nimodipine therapy due to the antihypertensive properties of this drug.[10,11] A recent study explored IV milrinone, a phosphodiesterase-3 inhibitor, as an investigational alternative for the treatment of vasospasm in the setting of aSAH.[19] Lakhal and others[19] conducted a controlled observational study in which patients who received milrinone therapy for cerebral vasospasm were compared with historical controls treated with induced hypertension alone. Both groups received nimodipine.[19] The investigators concluded that IV milrinone was independently associated with lower odds of 6-month functional disability (defined as a modified Rankin score between 2 and 6) and vasospasm-related brain infarction on imaging.[19] Patients who received milrinone also had reduced odds of requiring endovascular angioplasty.[19] Although Lakhal and others[19] conducted their study in an ICU, only 17% of the patients had a WFNS score of V, compared with 35% in our analysis. Initiation of milrinone was associated with hypotension and subsequent increases in norepinephrine infusions to maintain blood pressure targets.[19] Given that nimodipine was continued in both groups, milrinone may only be considered an adjunctive treatment on the basis of this evaluation. In our study, only 5% of patients received milrinone, but the information about concomitant medications was only collected within 24 hours of nimodipine dosage change, interruption, or discontinuation, which limited our ability to capture other relevant data over the course of the patient's entire hospital stay.

In the study conducted by Barfejani and others,[10] 36 of the 109 patients with aSAH received guideline-recommended dose and duration of nimodipine in a neurointensive care unit at a tertiary care centre. In contrast, only one of the patients in our study (i.e., 1%) received guideline-recommended dose and duration of nimodipine. This difference might be explained by the difference in mortality rates: 3% in the previous study[10] and 44% in the current study. Patients in our cohort generally presented with a higher severity of illness, as indicated by a total of 52% with a WFNS score of III to V, compared with 33% in the analysis by Barfejani and others.[10] Notably, those authors did not report the surgical interventions used. Sandow and others[11] reported that 96 of 220 patients with aSAH received the full daily dose of nimodipine in a neurointensive care unit in Germany. However, the investigators collected nimodipine data only for the first 14 days after diagnosis of aSAH.[11] Furthermore, 65% of their patients were treated with surgical clipping,[11] whereas 8% of our patients received that intervention. Although the choice of neurosurgical intervention is highly individualized, the guidelines recommend endovascular coiling for most patient populations.[4,5]

Our study was limited by its retrospective design, whereby the accuracy and completeness of our data sets was dependent on the quality of documentation available. For example, the reasons for changes in nimodipine therapy or early discontinuation often went undocumented in chart notes. Additionally, some of our data collection was further limited by the cross-sectional nature of the study. When collecting information about concomitant use of vasopressors and medications with blood pressure–lowering effects, data were collected only within 24 hours of nimodipine dosage change, interruption, or discontinuation. For patients discharged earlier than 21 days from the onset of aSAH, guideline adherence was assessed by determining whether nimodipine appeared on discharge prescriptions. This approach assumed that patients adhered to discharge instructions, even though the cost and availability of nimodipine may have hindered postdischarge adherence. Our use of an ICD-10 code for initial selection of potential participants relied on accurate coding in patients' records. Finally, we used a convenience sample due to limitations of time and resources.

CONCLUSION

Despite the inherent limitations of the evidence presented here, our best guidance in the treatment of aSAH still advocates unaltered nimodipine therapy for 21 days, with avoidance of interruptions. Our evaluation showed that the integrity of nimodipine treatment is most vulnerable during transfers and establishment of oral access at initial diagnosis, as well as during the management of patient-specific blood pressure targets. Clinical pharmacists are uniquely positioned to optimize nimodipine therapy by educating staff at transferring sites, advocating for better blood pressure management and protection of nimodipine therapy, and facilitating medication reconciliation on transfer and discharge.

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Competing interests: For activities unrelated to the study published here, Timothy Leung has served on the Fraser Health Research Ethics Board and the University of British Columbia Clinical Research Ethics Board; he was not involved in the ethics review for the current study. No other competing interests were declared.

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