Treatment of Drop Attacks with Nifedipine: A Case Report

James W. Mold, MD
Oklahoma City, Oklahoma

Drop attacks are sudden, unexpected, nonsyncopal falls, which are not preceded or accompanied by loss of consciousness, dizziness, lightheadedness, or loss of balance. They can be a manifestation of epilepsy, brain stem tumors, and a variety of other conditions. In the elderly, they have been associated with vertebrobasilar insufficiency, cervical spondylosis, or both. However, the specificity and etiology of drop attacks have come under some scrutiny in recent years. The patient described in this case report experienced frequent drop attacks that were effectively prevented with nifedipine. Possible pathophysiologic mechanisms are discussed and the relevant literature is reviewed.

Key words. Drop attacks; falls; vertebrobasilar insufficiency; geriatrics; aged; drug therapy. (*J Fam Pract 1995; 41:91–94*).

Drop attacks have been defined by Sheldon[1] as "sudden unexpected falls to the ground, usually while standing or walking, and often following neck movement, in an otherwise healthy elderly individual who vigorously denies loss of consciousness." Such attacks have previously been blamed for as many as 15% of geriatric falls.[2] Several pathophysiologic mechanisms have been implicated, most involving acute intermittent disturbances of brain stem function.[3,4] Drop attacks have traditionally been considered one of the specific fall syndromes observed in elderly patients. More recently some geriatricians argue that drop attacks should be considered symptomatic of a spectrum of diseases rather than a distinct clinical entity, and that the 15% figure is an overestimate of the true prevalence of drop attacks.[5] Some have suggested abandoning the term altogether.[6]

In children and younger adults, drop attacks have been reported to be a manifestation of seizure disorders.[7–10] A specific syndrome, which has been described in middle-aged women, seems to be hereditary to some degree and of unknown etiology.[11–14] A variety of other causative factors have been reported, including medications (eg, clozapine),[15] muscular dystrophy,[16] colloid cysts,[17] aneurysms and other masses in the third ventricle or posterior fossa,[18] congenital cardiac lesions,[19] Parkinson's disease,[20] Meniere's disease,[21,22] and hypothyroidism.[23] Some elderly patients who experience drop attacks may simply have weak quadriceps muscles that "give out" suddenly after extended use.

Because of the prevalence of cerebrovascular disease and the reported association of drop attacks with neck hyperextension and with abnormalities of the brain stem, vertebrobasilar insufficiency is generally considered to be a common cause of true drop attacks in the elderly.[24] Drop attacks have been reported to occur in up to 25% of patients with symptomatic vertebrobasilar insufficiency.[25] In such cases, the pathophysiologic mechanisms may include mechanical obstruction (kinking) of the vertebrobasilar arteries,[26] sudden systemic hypotension (caused by arrhythmia, for example), embolization, or spasm. Other than the avoidance of extreme neck movement and the empiric use of aspirin or warfarin, no effective treatment has been identified for drop attacks that result from vertebrobasilar insufficiency. Because of the sudden, bilateral, reversible nature of drop attacks, vascular spasm involving an atherosclerotic vertebrobasilar system seems a plausible mechanism, particularly when there has been no

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From the Research Division, Department of Family Medicine, University of Oklahoma Health Sciences Center, Oklahoma City. Requests for reprints should be addressed to James W. Mold, MD, Associate Professor, Director, Research Division, Department of Family Medicine, University of Oklahoma Health Sciences Center, Family Medicine Center, Suite 2207, 900 NE 10th St, Oklahoma City, OK 73104.

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history of neck extension or turning and no evidence for hypotension. It was on this basis that nifedipine was chosen as a pharmacologic agent to treat the patient in this case.

Case Report

An 86-year-old widowed woman was referred by her family physician to a geriatric evaluation clinic in January of 1986 because of recurrent falls. Over the previous 5 years, she had experienced 12 falls, at least 8 of which had occurred during the most recent 2 years. In the past year, she had injured her right side during one fall and her right shoulder during two others, and fractured her left hip as a result of a fourth fall. Each fall had occurred suddenly and without warning, as if her legs had simply collapsed. By her recollection, the episodes were not associated with looking up or to the side and had not occurred while she was wearing tight neckwear. She reported having felt posterior neck pain, which lasted for an hour or so following about half of the episodes. The pain was accompanied by a feeling that blood was rushing up and down the back of her neck. Although she acknowledged that she had been unable to extend her neck very far without experiencing a "funny feeling" in her head, she carefully avoided doing so and, thus, did not relate this to her falls.

The patient's medical history was significant for a stroke in 1974, resulting in left-sided hemiparesis that eventually resolved with almost no residual. She also reported a vague history of coronary artery disease with angina, and even some "heart attacks" in the past (no hospitalization) but no related problems within the most recent several years. She had a 10-year history of hypertension. She also had been treated for glaucoma for 2 years with timolol eye drops, and had used decongestants off and on between 1971 and 1983 for frontal sinusitis but was no longer bothered by this problem. Previous surgical procedures included a thyroidectomy for a goiter, a cholecystectomy, and a hysterectomy, all in the remote past, and she had a left hip fixation in October 1985.

At the time of initial evaluation her medications were: aspirin 325 mg daily; dipyridamole 50 mg three times daily; hydrochlorthiazide 25 mg once a day; timolol eye drops twice daily; calcium carbonate 500 mg three times daily; a multivitamin with iron daily; and occasional doses of acetaminophen with codeine for pain resulting from her healing hip fracture.

She was 5'0" tall and weighed 104 pounds. Her blood pressure supine was 148/78 mm Hg, sitting 150/92 mm Hg, and standing 150/78 mm Hg, with pulse rates of 78, 80, and 86 beats per minute, respectively. Her skin was generally dry, and the results of her eye examination were normal. She had mildly impaired hearing bilaterally. She was edentulous with well-fitting dentures. She had decreased range of neck movement without pain, and hyperextension caused a "funny feeling in my head" but no loss of postural control. There was a thyroidectomy scar, and the residual thyroid tissue felt slightly larger than a normal gland.

Examinations of her chest, heart, and abdomen revealed no abnormalities. Carotid, radial, and dorsalis pedis pulses were intact with no bruits, but her femoral pulses were somewhat diminished with a bruit on the right, and her posterior tibial pulses were absent. She had bilateral 1+ pitting ankle edema.

On neurologic examination, her cranial nerves, strength, tone, deep tendon reflexes, and sensation to pin and touch were normal. Vibratory sensation was diminished at the ankles. There was no tremor. Her Romberg test was positive and her gait asymmetric, as she favored her left leg. Her Folstein Mini-Mental State score was 29/30.

Laboratory testing included a complete blood count, chemistry panel, thyroid-stimulating hormone, vitamin B_{12}, folate, rapid plasma reagin, and urinalysis. The results were all within normal limits with the exception of a nonfasting total cholesterol of 300 mg/dL (7.76 mmol/L) and triglycerides of 240 mg/dL (2.71 mmol/L).

She was returned to the care of her primary physician with the following recommendations regarding the drop attacks: (1) switch from the thiazide diuretic to nifedipine and avoid decongestants; (2) avoid neck hyperextension (which she was already doing); (3) conduct a home-safety evaluation; (4) obtain a telephone connection to Lifeline, an emergency assistance service; (5) obtain cervical spine radiographs; (6) discontinue dipyridamole; and (7) obtain an informal vascular surgery consultation regarding the potential value of cerebral arteriography.

The cervical spine radiographs revealed marked degenerative joint disease but no other abnormalities. This type of imaging study is actually of limited value in these cases.[27] The vascular surgeon did not think arteriograms would be helpful since no satisfactory surgical procedure was available to treat the vertebral stenosis. This advice was not entirely correct, as there are a number of studies to indicate otherwise.[28–33] Her son helped her safety-proof her home, and a Lifeline hookup was obtained. She tolerated nifedipine 10 mg every 8 hours with no substantial change in blood pressure but with some increase in her dependent edema.

Since the relocation of her original primary care physician to another town, she has been followed by the author. During 9 years of follow-up, she has experienced only two falls. The first, which occurred in 1987 while she was visiting her sister in a nursing home in Denver, was a

classic drop attack that resulted in fractures of her right hip and right elbow. During that hospitalization, she had an electrocardiogram, which showed normal sinus rhythm, frequent premature atrial contractions, and diffuse nonspecific ST-T wave changes. The second was associated with tripping over a door jam in her home in 1989. She was switched to sustained-release nifedipine 30 mg once daily when it became available.

At the time of this report (1995), the patient lives independently in her own home with regular support and assistance from her son.

Discussion

While it is certainly possible that the patient's drop attacks ceased spontaneously, both she and I are convinced that nifedipine played a major role in the resolution of this problem. Other possible factors include the discontinuation of hydrochlorothiazide, dipyridamole, or both, and the instructions regarding avoidance of neck hyperextension. These factors, however, seem less likely than the nifedipine to have made the difference.

The patient's symptoms fit the classic description of drop attacks. It is possible that they could have been the result of a seizure disorder or an intermittent arrhythmia. However, the absence of lightheadedness, loss of consciousness, spontaneous motor activity or postictal drowsiness mitigates against these possibilities. Neither an electroencephalogram nor a Holter monitor was felt to be indicated in her evaluation. Another potential contributing factor to the patient's drop attacks is highlighted in a study by Glynn et al[34] of patients with glaucoma. This study found an association between falls and the use of nonmiotic eye drops, such as timolol. However, her attacks all but ceased despite continuation of this medication.

Although coronary artery spasm has been well documented and can be prevented or reversed with calcium channel blocking agents, spasm in the cerebral circulation has been less well studied. In several different circumstances, cerebral vasospasm has been postulated or observed. Following subarachnoid hemorrhages, cerebral artery spasm has been documented angiographically beginning as early as 3 to 4 days following the event.[35] Signs of progressive cerebral ischemia are not uncommon during this period. In dogs, at least, this spasm can be blocked or reversed with nimodipine, a calcium channel blocker.[36] Nimodipine may limit ischemic brain damage in humans,[37] but the mechanism may or may not involve a direct effect on vascular spasm.[38,39]

Migraine headaches are associated with vascular constriction that may cause neurologic symptoms followed by vascular relaxation accompanied by pain. At least some calcium channel blocking agents appear to be effective in blocking migraine attacks.[40,41] Again, it is not clear that their effect is related to vascular relaxation.[42]

In experimentally induced vertebrobasilar insufficiency in both rabbits and dogs, Hirschberg and Hofferberth[43] have demonstrated a beneficial effect of calcium channel blockers on ischemia-induced vestibular nystagmus. They were, however, unable to determine whether this was related to an increase in blood flow or to a direct effect on the vestibular system. Winterkorn et al[44] have recently reported the successful treatment of nine patients with recurrent vasospastic amaurosis fugax with calcium channel blockers.

Based on a single case, it would be imprudent to suggest that elderly patients experiencing drop attacks should be treated with nifedipine. However, since few alternative treatment options are available, this case suggests that further investigational trials should be undertaken in selected patients.

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