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Midazolam vs. Diphenhydramine for the Treatment of Metoclopramide-induced Akathisia: A Randomized Controlled Trial

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Abstract

Objectives: To compare the effects of midazolam, which is a fast and short-acting benzodiazepine, and diphenhydramine, which is a widely used anticholinergic agent, in clinical practice for the treatment of metoclopramide-induced akathisia.

Methods: All adults older than 17 years given metoclopramide for nausea and vomiting or for headache and who had akathisia were eligible for this clinical, randomized, double-blind trial. Patients were randomized to receive diphenhydramine or midazolam. Subjective, objective, and total akathisia scores and modified Ramsay Sedation Scale scores were recorded. Repeated-measures analysis of variance was used to compare the efficacy and side effects of the medications.

Results: Forty-one (73.3%) of the 56 enrolled patients were women. The mean (±SD) age was 39.9 (±15.7) years in the diphenhydramine group and 40.9 (±16.2) years in the midazolam group. Mean subjective, objective, and total akathisia scores in the first 5 minutes declined considerably in the midazolam group compared with the diphenhydramine group ($p < 0.001$). However, the mean Ramsay Sedation Scale score in the first 15 minutes increased significantly in the midazolam group compared with the diphenhydramine group ($p < 0.001$).

Conclusions: Midazolam can correct the symptoms of metoclopramide-induced akathisia faster than diphenhydramine, but it causes more sedation.

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Keywords: akathisia, metoclopramide, midazolam, diphenhydramine, benzodiazepines, anticholinergics

Metoclopramide is a frequently used agent in emergency departments (EDs) for the management of nausea, vomiting, and vascular-type headache.[1,2] Even though it is generally accepted as safe, it has been frequently reported to cause extrapyramidal symptoms such as rigidity, tremor, and akathisia.[3,4] Akathisia can be defined as a state of mental and motor restlessness. It includes a subjective component that consists of intense feelings of internal discomfort and tension with a compulsive urge to move, as well as an objective component characterized by motor agitation particularly affecting the legs and resulting in the observable inability to remain still, either while sitting or standing.[5,6]

Akathisia can easily be missed by physicians in crowded and busy EDs. If the initial symptoms are underdiagnosed, massive anxiety and agitation can lead to refusal of treatment and patients pulling out their intravenous lines, leaving the ED, and even attempting suicide.[7–11] After diagnosing an episode of drug-induced akathisia, prompt treatment, including discontinuation of the drug and initiation of treatment to reduce unpleasant symptoms, is advisable. Anticholinergics, benzodiazepines, beta-blockers, α_2-agonists, and opiates have all been used to treat akathisia.[6–8,12–15] Anticholinergic drugs seem to be successful in relieving all symptoms of akathisia, whereas benzodiazepines tend to best relieve the subjective components.[11]

Anticholinergics currently are the first-line therapy for akathisia in most clinics in the United Kingdom, although these have become a less popular option in the United States.[16] Centrally acting anticholinergics presumably

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restore the relative balance between dopaminergic and cholinergic activity. Diphenhydramine has been used effectively to relieve the symptoms of akathisia.[12–14,17–20] An alternative approach is the use of benzodiazepines. Although benzodiazepines may represent a safe and familiar treatment modality, the evidence to support their efficacy (specifically from studies using diazepam, lorazepam, and clonazepam) in acute akathisia is largely anecdotal or derived from open studies. More research is needed to support the use of benzodiazepines for akathisia.[11,16–21]

The aim of this study was to compare the effects of midazolam, which is a fast and short-acting benzodiazepine, and diphenhydramine, which is a widely used anticholinergic agent, for the treatment of metoclopramide-induced akathisia. To the best of our knowledge, this is the first randomized controlled trial to compare the efficacy and side effects of these two medications in this clinical setting.

METHODS

Study Design

This was a clinical, randomized, double-blind trial. Permission from the institutional review board of Dokuz Eylul University was obtained. All participants provided written informed consent for the study.

Study Setting and Population

The study was conducted in the ED of Dokuz Eylul University between November 2001 and November 2002. Subjects were recruited from patients who developed acute akathisia following treatment with metoclopramide requiring immediate medical intervention for relief of symptoms. Subjects were between 17 and 65 years of age and weighed 50–90 kg. Their akathisia scores were ≥ 7 (Table 1), which represents moderate and severe forms of akathisia[22]; their peripheral oxygen saturation level was $\geq 90\%$ while breathing room air, and all were free of any respiratory problems. Patients who had liver and renal insufficiency, electrolyte imbalance, acute respiratory symptoms, chronic obstructive pulmonary disease, or blood pressure <90/60 mm Hg; who were uncooperative or pregnant or lactating; or who had a preexisting motor disorder, restless legs syndrome, Parkinson's disease, organic brain disorder (e.g., dementia), or epilepsy were excluded. Also excluded were patients who were admitted to the ED for acute psychiatric symptoms; who had decreased mental status, advanced hearing loss, malnutrition, an acute asthma attack, a serious physical illness (especially glaucoma, prostatic hypertrophy, or cardiac disease), a contraindication to anticholinergic medications, or who had taken an antiemetic within three days of study entry; who were taking antihistamine, antipsychotic, antispasmodic, alpha-blocker, or Ca^{2+} channel

Table 1
Akathisia Rating Scale and Modified Ramsay Sedation Scale

The Prince Henry Hospital Rating Scale of Akathisia*	
Objective ratings (ratings by observer)	
I. Sitting	
1. Semipurposeful/purposeless leg/feet movement	0 1 2 3
2. Semipurposeful hand/arm movements	0 1 2 3
3. Shifting body position in chair	0 1 2 3
4. Inability to remain seated	0 1 2 3
II. Standing	
1. Purposeless/semipurposeless leg/feet movements	0 1 2 3
2. Shifting weight from foot to foot and/or walking on spot	0 1 2 3
3. Inability to remain standing on one spot (walking or pacing)	0 1 2 3
Sum score	
Subjective ratings (three questions were asked)	
1. Do you feel restless, or urge to move, especially in the legs?	0 1 2 3
2. Are you unable to keep your legs still?	0 1 2 3
3. Are you unable to remain still, standing or sitting?	0 1 2 3
Key: 0–3	
0 = absent	
1 = mild and present some of time	
2 = mild and present most of the time or severe and present some of the time	
3 = severe and present all the time	
Sum score	
Total score	
Modified Ramsay Sedation Scale: This scale has 6 levels	
Three for awake levels	
1 = patient is anxious, agitated, or restless,	
2 = patient is cooperative, oriented, and tranquil	
3 = patient is responsive to verbal stimulus	
Three for asleep levels	
4 = patient responds to pain	
5 = patient has a sluggish response to pain	
6 = patient has no response	

Global rating (by rater): 0 = absent; 1 = mild; 2 = moderate; 3 = severe.
* Reprinted with permission from Sachdev P. A rating scale for acute drug-induced akathisia: development, reliability, and validity. Biol Psychiatry. 1991; 35:270–1. Copyright Society of Biological Psychiatry (1991).

blocker medications; or who had taken an antidepressant, barbiturate, benzodiazepine, other sedative/hypnotic, opioid, lithium, or illicit sympathomimetic agent within two weeks of study entry.

Study Protocol

The medications used to treat akathisia in this trial were midazolam 2 mg (Dormicum; Roche Ltd., Basel, Switzerland) or diphenhydramine 20 mg (Benison; Biosel, Istanbul, Turkey), both administered intravenously. Midazolam is an imidazobenzodiazepine derivative that is two to three times as potent as diazepam. It is water-soluble, has a rapid onset and short duration of action, and produces a profound amnesic effect.[23] Patients received medication for akathisia according to their random allocations. Randomization was achieved by using computer software to generate random numbers.

During the intervention, participants' oxygen saturation, blood pressure, heart rate, and cardiac rhythms were monitored. One researcher blinded to patient allocation observed the patients during the study and recorded the akathisia and sedation scores. The Prince Henry Hospital Rating Scale of Akathisia[22,24] was used to measure the level of objective, subjective, and total akathisia scores.[13,14,25–27] The modified Ramsay Sedation Scale (RSS)[28] was used to measure the level of sedation (Table 1).

All medications used during the study were recorded. Additionally, pulse, systolic blood pressure, diastolic blood pressure, respiration rate, and oxygen saturation were recorded at baseline (0 minutes) and at 15, 30, and 60 minutes after receiving the study drugs. Objective, subjective, and total akathisia scores were also recorded at baseline (0 minutes) and at 5, 15, 30, and 60 minutes. RSS score was also recorded at baseline (0 minutes) and at 5, 15, 30, 60, and 120 minutes. Adverse reactions (hemodynamic or respiratory compromise, dizziness, fever, perspiration, palpitation, and so on) during the study were also recorded.

Data Analysis

Data are summarized as means (±SD) and percentages. Chi-square and t-test were used for comparisons. Repeated-measures analysis of variance was used to compare the efficacy and side effects of the medications. Ordinal data were also examined using nonparametric tests, which confirmed the statistical significance of findings. A p-value less than 0.05 was considered statistically significant. SPSS 10.0 for Windows (SPPS Inc., Chicago, IL) was used for statistical analysis.

RESULTS

Sixty participants were randomized into two groups; two from each group left the ED before completing the trial. Forty-one (73.3%) of the 56 patients were female, and the mean (±SD) age was 39.9 (±15.7) years in the diphenhydramine group and 40.9 (±16.2) years in the midazolam group. There were no significant baseline differences among groups in terms of patients' demographic and hemodynamic characteristics. However, there were significant differences between the groups in terms of baseline objective, subjective, and total akathisia scores ($p = 0.01$, $p = 0.04$, and $p = 0.007$, respectively) (Table 2). Mean subjective, objective, and total akathisia scores in the first 5 minutes declined considerably in the midazolam group compared with the diphenhydramine group ($p < 0.001$) (Figure 1A–C). However, the mean RSS score in the first 15 minutes increased significantly in the midazolam group compared with the diphenhydramine group ($p < 0.001$). There were no significant differences among groups in terms of changes in mean vital signs during the study (Figure 2A–D). One patient in the diphenhydramine group and four patients in the midazolam group reported dizziness. In addition, in the diphenhydramine group, there were two patients with high blood pressure, one patient with nausea, one patient with rising fever, and one patient with palpitations. In the midazolam group, one patient experienced diaphoresis and one patient had palpitations. None of the patients showed hemodynamic or respiratory compromise or returned to the ED with continued symptoms of akathisia.

DISCUSSION

The current study found that both medications were effective in the treatment of akathisia induced by a single 10-mg dose of intravenous metoclopramide. Parenteral midazolam was associated with a rapid improvement of the signs and symptoms of akathisia, especially within the first 5 minutes. However, midazolam also generated more sedation in the first 15 minutes.

The pathophysiology of drug-induced akathisia has not been clearly explained, and its pharmacologic treatment has not been standardized.[6,29] Because acute neuroleptic-induced akathisia is most frequently associated with

Table 2
Distribution of the Baseline Characteristics between the Study Groups

Variables	Medication		p-value*
	Diphenhydramine ($n = 28$)	Midazolam ($n = 28$)	
Gender (%)			0.76
Male, $n = 15$ (27)	8 (28.6)	7 (25.0)	
Female, $n = 41$ (73)	20 (71.4)	21 (75.0)	
Age (yr)	39.9 (15.7)	40.9 (16.2)	0.64
Weight (kg)	65.0 (10.9)	67.4 (10.0)	0.41
Pulse rate (beats/min)	78.9 (11.4)	82.4 (8.9)	0.21
Systolic blood pressure (mm Hg)	124.9 (27.3)	122.7 (17.1)	0.72
Diastolic blood pressure (mm Hg)	81.2 (14.1)	80.3 (10.1)	0.78
Respiration rate (breaths/min)	17.5 (2.7)	17.4 (3.1)	0.89
Objective akathisia scores	5.4 (1.9)	6.6 (1.5)	0.01
Subjective akathisia scores	7.2 (2.0)	8.1 (1.2)	0.04
Total akathisia scores	12.6 (3.3)	14.7 (2.3)	0.007
Ramsay Sedation Scale score	1.4 (0.5)	1.4 (0.5)	0.79

All values are mean (SD) unless otherwise noted.
* p-values from chi-square and t-tests.

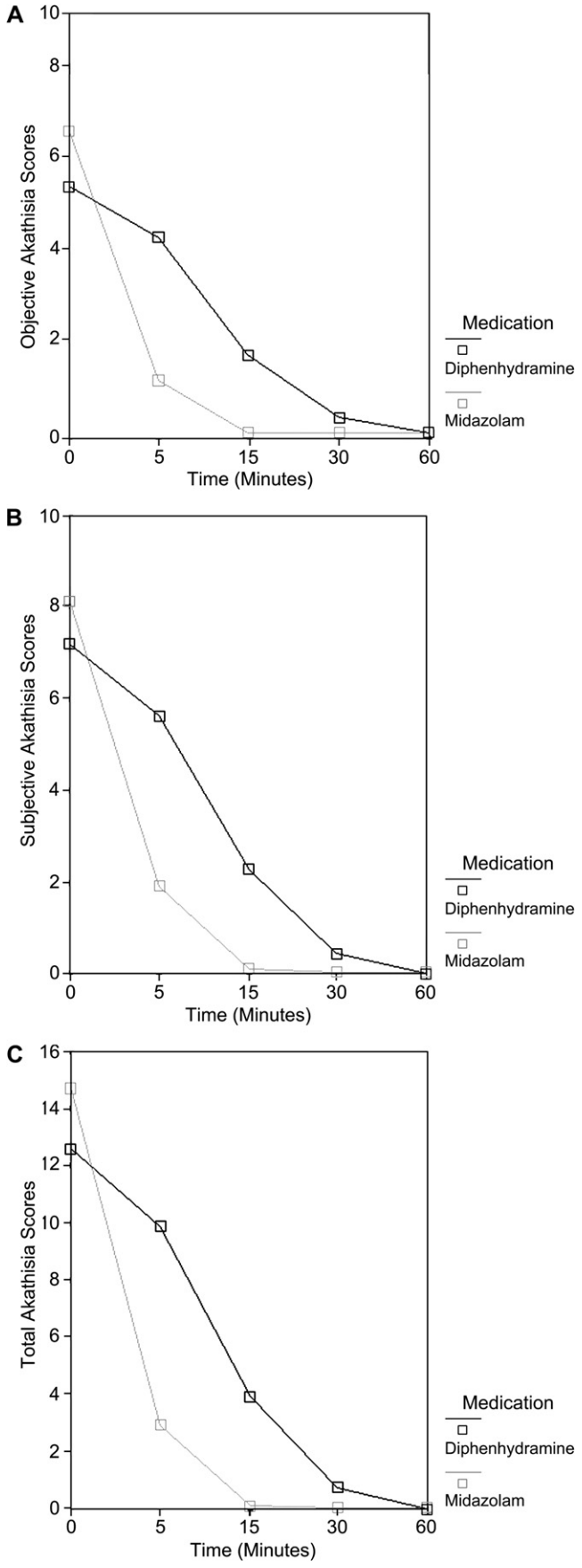

Figure 1. Mean (A) objective, (B) subjective, and (C) total akathisia scores for each study medication.

drugs that have high D2 antagonism and low antimuscarinic activity (i.e., prochlorperazine, metoclopramide), it is believed that drug-induced akathisia is at least partially attributable to the blockade of postsynaptic dopamine D2 receptors in the basal ganglia, particularly the mesocortical pathways.[5,6,24,30] Additionally, anticholinergic medications are believed to treat akathisia by restoring the necessary balance in central dopaminergic and cholinergic activity.[29] Centrally acting anticholinergics, such as diphenhydramine, presumably restore the relative balance between dopaminergic and cholinergic activity.[12–14,17–20] Diphenhydramine has been used for years on this basis.[14] The downside of diphenhydramine is the potential for anticholinergic symptoms, including cognitive decline and increased glaucoma or symptoms related to prostatic hypertrophy, especially among elders.[17,31]

An alternative approach to the management of drug-induced akathisia supported by some clinicians is the use of benzodiazepines. The rationale for the use of benzodiazepines in the treatment of akathisia stems from three observations: 1) the level of anxiety influences the manifestation of akathisia; 2) the subjective component of akathisia resembles anxiety; and 3) restless legs syndrome, which resembles akathisia phenomenologically, has been reported to respond to treatment with benzodiazepines.[21] However, the evidence to support the efficacy of benzodiazepines in acute akathisia has been largely anecdotal or derived from open studies.

Diazepam, lorazepam, and clonazepam have been reported to be effective in neuroleptic-induced dystonia and akathisia.[11,16,18–21,32–34] Recent knowledge of the interactions of dopamine, acetylcholine, and γ-aminobutyric acid in the basal ganglia suggests that a γ-aminobutyric acid agonist like diazepam might be equivalent to an anticholinergic in the alleviation of syndromes that result from dopamine receptor blockade.[17] Gagrat et al.[17] performed a double-blind study of a single 5-mg intravenous dose of diazepam versus 50 mg of diphenhydramine. The study did not find a statistical difference between the medications in reducing akathisia. The akathisia ratings after the study drug infusion were significantly lower than baseline in both the diazepam group and the anticholinergic group. The investigators suggested that intravenous diazepam was as effective as a standard anticholinergic drug in the treatment of acute neuroleptic-induced dystonia and akathisia. Schroeder et al.[35] found that patients with metoclopramide-induced akathisia responded favorably to midazolam, suggesting the possibility that premedication with midazolam may reduce or prevent its incidence. LaGorio et al.[11] and Miller and Fleischhacker[21] suggest that benzodiazepines tend to best relieve the subjective components of akathisia; addition of benzodiazepines would appear to be a sensible choice, especially if subjective distress persists. Kutcher et al. performed a double-blind trial of clonazepam ($n = 7$) versus placebo ($n = 7$).[36] All patients who received clonazepam showed an amelioration of their symptoms of akathisia, whereas symptoms in five of the patients in the placebo group were unchanged. The results of nonblind trials with lorazepam and clonazepam have shown that benzodiazepines can be effective in the treatment of acute and chronic akathisia.[36–38] Donlon[32] reported treatment with diazepam of 13 patients with akathisia in whom treatment with diphenhydramine (75

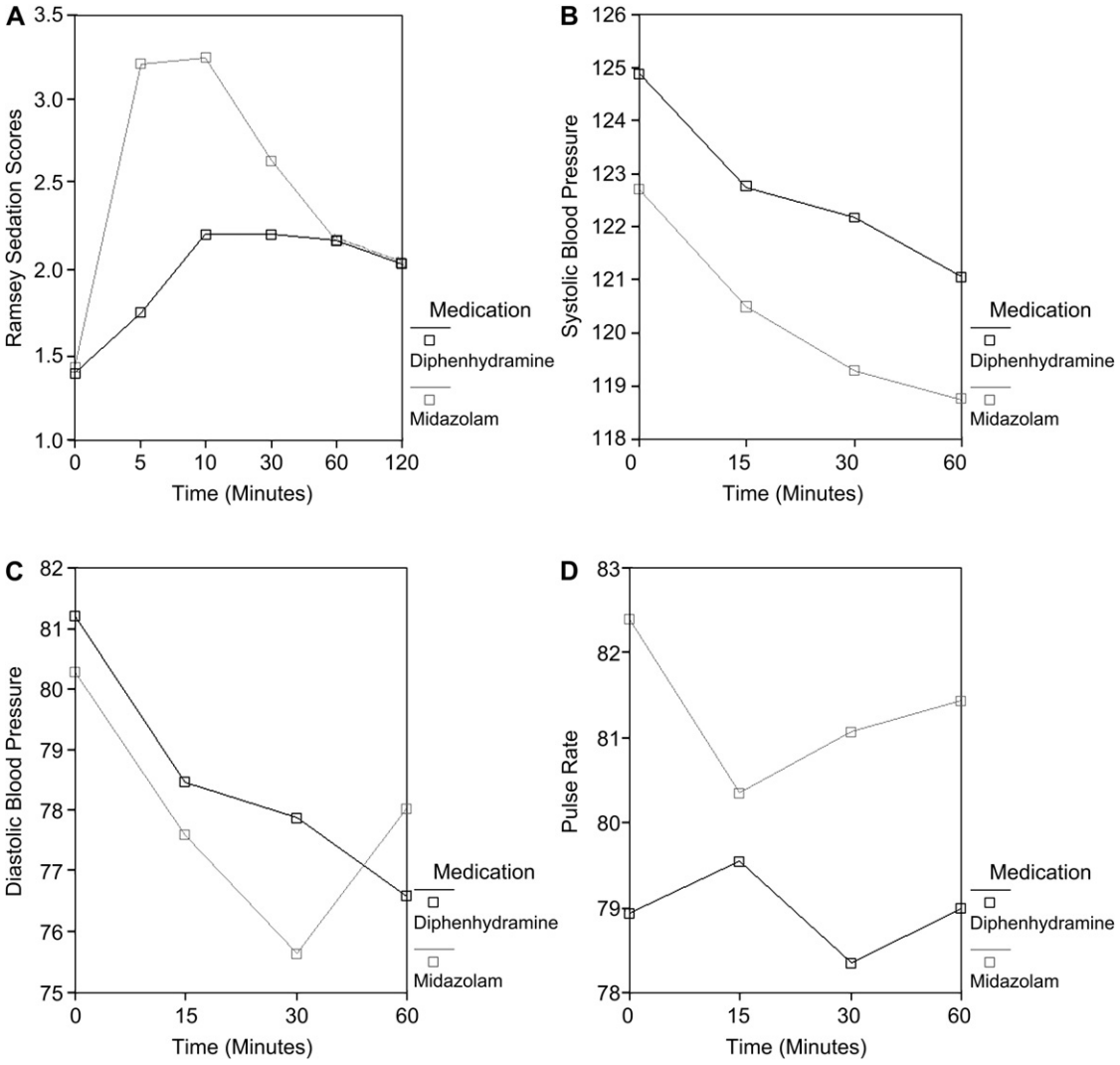

Figure 2. (A–D) Mean vital signs and modified Ramsay Sedation Scale scores for each medication.

mg/day) had been unsuccessful. He reported that diazepam effectively relieved akathisia in ten patients within three days. Hirose and Ashby[39] studied patients with neuroleptic-induced acute akathisia during treatment with antipsychotic medication and who required immediate relief from the distress of akathisia. Eighteen patients received intravenous diazepam (5 mg per 30 seconds); all 18 experienced immediate relief from akathisia after the injection of diazepam (mean [±SD] dose, 12.6 [±2.6] mg; range, 10–17 mg). The results suggest that intravenous diazepam can be used in the treatment of patients with severely distressing akathisia who require immediate relief.

In our study, we observed that akathisia scores in the midazolam group, especially in the first 5 minutes after treatment, significantly decreased, whereas the patients in the diphenhydramine group did not experience the same relief until after 15 minutes. Further, these results occurred even though the objective, subjective, and total akathisia scores at baseline were significantly higher in the midazolam group than in the diphenhydramine group. We also observed that many patients in both arms of the study became drowsy after treatment, and some fell asleep; none, however, were unable to be aroused. Midazolam seems to be a fast, effective treatment for akathisia and has the additional advantage of being safe for patients with glaucoma or prostatic hypertrophy.

LIMITATIONS

Our study had a larger sample size than many previous studies, but our limited sample size could be a shortcoming. Another possible limitation is that only patients who had an akathisia score ≥ 7 were eligible for this study. We believed that patients with lower scores could be spared from receiving another medication because there was a higher likelihood of a spontaneous fast recovery. Further, the usual dosage of diphenhydramine recommended is between 25 and 50 mg per dose.[13] However, the only available formulation for this medication in Turkey is 20 mg. Due to logistic concerns, we used one

flacon (2 mL = 20 mg) for patients in our study. This borderline dosage may also affect the results.

CONCLUSIONS

This randomized, controlled, double-blind study demonstrates that intravenous midazolam more rapidly reduces the signs and symptoms of metoclopramide-induced akathisia than diphenhydramine.

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REFLECTIONS

Tiny Hands

Sideways glances
Bitter looks
Arms folded across chests
Holding damaged hearts

Stones of hurt and anger
Resentment and blame
Two retaining walls
Prevent passage

Friends and family left to wonder
Will they ever remember?
His tiny hands grabbing
Their fingers the first time

The smell of baby hair
Freshly washed and combed
Pastel pajama feet hurry down the hall
Amidst giggling

Their eyes narrow harshly
As they look upon each other
Sending silent messages
Impossible forgiveness

The clock marks the hour
Cordial smiles and handshakes
The sheerest of fabric
They veil the memory

Wet baby hair
And tiny hands
At the cold gray bottom
Of the pool

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