Prolonged QT Interval, Syncope, and Delirium with Galantamine

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OBJECTIVE: To describe a case of QT interval prolongation, syncope, and delirium associated with galantamine use and to analyze similar cases related to acetylcholinesterase inhibitors (AChIs) reported to the Australian Adverse Drug Reaction Advisory Committee (ADRAC).

CASE SUMMARY: An 85-year-old man with dementia was treated with prolonged release galantamine 8 mg daily for 1.5 years. Three months prior to the current admission, he had a syncopal episode with low blood pressure and bradycardia. Two months later, galantamine was withdrawn, but within 2 weeks, the man developed marked cognitive, behavioral, and functional deterioration and galantamine was restarted. Three weeks later, he developed syncope, delirium, hypotension, and prolonged QT interval with serious cardiac arrhythmias, in addition to vomiting and diarrhea. A complete blood cell count and biochemistry panel performed on admission were normal. No infection was detected. Galantamine and irbesartan were ceased. The delirium fully resolved in 6 days, and the QT interval shortened from 503 to 443 msec (corrected by Bazett's formula) 4 days after discontinuation of galantamine and remained normal.

DISCUSSION: In the ADRAC reports, galantamine was associated with 18 cases of delirium/confusion, 8 of syncope, 13 of bradycardia, 6 of other arrhythmias or conduction abnormalities, and 6 of hypotension. Donepezil was associated with 56, 15, 26, 15, and 5, and rivastigmine with 21, 8, 6, 2, and 2, respectively, of these reactions. Five fatal outcomes were reported in association with galantamine, 11 with donepezil, and 3 with rivastigmine, including 3, 6, and 0 sudden deaths, respectively. This case, along with previously published reports and cases identified from the ADRAC database, illustrates that AChIs may lead to delirium, syncope, hypotension, and life-threatening arrhythmias. The Naranjo probability scale indicated that galantamine was the probable cause of QT interval prolongation, syncope, and delirium in this patient.

CONCLUSIONS: Administration of galantamine and other AChIs requires vigilance and assessment of risk factors that may precipitate QT interval prolongation, syncope, and delirium.

KEY WORDS: delirium, galantamine, prolonged QT interval, syncope.

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Acetylcholinesterase inhibitors (AChIs) are used in treatment of mild-to-moderate Alzheimer's disease.[1,2] These drugs also improve cognition in patients with other types of dementia, as well as those with schizophrenia, and may have neuroprotective properties.[3] A Cochrane review concluded that there is no difference among AChIs with respect to efficacy.[1]

AChIs are well tolerated.[1,4] Among cholinergically mediated adverse effects, gastrointestinal symptoms (nausea, vomiting, diarrhea, anorexia) predominate. Patients receiving AChIs rarely develop potentially serious cardiovascular adverse effects such as syncope, bradycardia, atrial arrhythmias, and sinoatrial and atrioventricular block.[5] Seventeen cases of cardiac dysrhythmias associated with donepezil have been reported.[6] Two patients, one on galantamine[7] and another on rivastigmine,[8] developed prolonged cardiac repolarization (prolonged QT interval), which may lead to life-threatening polymorphic ventricular arrhythmias (torsade de pointes).

We report a case in which galantamine was associated with QT interval prolongation, syncope, delirium, vomiting, and diarrhea. We also searched the Australian Adverse Drug Reaction Advisory Committee (ADRAC) database for similar effects related to AChIs.

Case Report

An 85-year-old white man, who was a widower and a resident of an assisted living facility was brought to the emergency department by ambulance because of agitation,

Author information provided at the end of the text.

facial trauma, and lacerations to his forehead, nose, and right elbow following a fall. Witnesses reported that the man had loss of consciousness. He was nauseated and had vomited the previous day and developed weakness, diarrhea, and dizziness. He fell getting out of the bed on the admission day. Three weeks prior to admission he was restarted on galantamine prolonged release 8 mg in the morning after being off the drug for 2 weeks.

About 1.5 years earlier, the patient was diagnosed with mild dementia of mixed etiology (Alzheimer's and vascular) and was treated with galantamine 8 mg daily with satisfactory stabilization for the first 3 months and slow worsening in the next year. He scored 26 of 30 on the Folstein Mini-Mental State Examination (MMSE) before and 3 months after therapy started and 23 nine months later. Three months prior to the current admission he had a syncopal episode with low blood pressure (100/80 mm Hg) but without orthostatic hypotension. An electrocardiogram (ECG) showed sinus bradycardia of 59 beats/min, with normal PR and QTc intervals (Table 1[9,10]). He was hospitalized, but the cause of syncope was not identified. Two months later, at a routine check-up, galantamine was discontinued by his physician because of the syncopal episode. Within 2 weeks of galantamine discontinuation, family members noted worsening in cognitive, behavioral, and functional status. Galantamine was restarted at the same dose (8 mg daily).

The patient had a history of coronary artery disease, hypertension, hypercholesterolemia, osteoarthritis, hiatus hernia, and surgical treatment for benign prostatomegaly. At the time of presentation, he was also receiving, for more than 3 years, irbesartan 75 mg daily, clopidogrel 75 mg daily, simvastatin 20 mg daily, pantoprazole 40 mg daily, ergocalciferol 1000 IU daily, calcium carbonate 600 mg twice a day, and acetaminophen 1 g 2 times a day.

On admission, the patient was confused, restless, agitated, and incontinent of urine. His blood pressure was 84/46 mm Hg, pulse 79 beats/min, respiratory rate 18 breaths/ min, temperature 36.5 °C, and room-air oxygen saturation 95%. He had deep lacerations of his forehead, nose, and right elbow and loose front upper teeth, but no focal neurologic deficit. Complete blood cell count biochemistry, thyroid function, cardiac troponin I, and C-reactive protein were normal. No blood, urinary, or fecal infection was detected.

The QTc interval was significantly prolonged regardless of the formula applied (Table 1). Electrocardiographic 24 hour Holter recording on the third admission day showed sinus rhythm with average heart rate 60 beats/min (minimum 40, maximum 90), frequent multifocal ventricular ectopics, runs of ventricular beats, ventricular bigeminy and trigeminy, frequent supraventricular ectopics, aberrantly conducted supraventricular run of 10 beats, supraventricular bigeminy and trigeminy, supraventricular tachycardia, but no pauses. Echocardiogram revealed normal left ventricular size, mild segmental left ventricular dysfunction, and estimated left ventricular ejection fraction of 51%.

Computed tomography (CT) of the head did not show any evidence of acute intracranial bleed or infarct, but there was prominence of cerebral sulci and the ventricles in keeping with cerebral atrophy, and there were old lacunar infarcts. CT of the cervical spine revealed fractures of the spinous process of C4, right superior articular process of C3, and left superior articular process of C5. Findings from chest and abdominal X-rays were unremarkable.

Galantamine and irbesartan were ceased. No other changes in the patient's long-term medications were made. The patient was treated with a cervical collar, infusion of NaCl 0.9% (1.5 L/day), prophylactic antibiotics, and thromboembolic prophylaxis (enoxaparin sodium 40 mg daily). Lacerations were sutured and loose teeth were removed. His delirium resolved over 6 days, but he required haloperidol for agitation (0.25 mg intravenously 6 times in 6 days). The QTc interval shortened to 443 msec (by

Table 1. Electrocardiographic Characteristics and Corrected QT Intervals Calculated by Different Correction Formulas

Date	Heart Rate, beats/min	PR, msec	RR, msec	QT, msec	QTc, msec, by formula[a]			
					Bazett	Fridericia	Framingham	Hodges
Feb 6, 2007	59	146	1017	425	421	423	422	423
May 14, 2007[b]	83	168	723	428	503	477	471	468
May 18, 2007	54	144	1111	468	443	452	451	457
May 19, 2007	63	128	952	420	430	427	427	425
May 22, 2007	55	136	1091	456	436	443	442	447
May 24, 2007	59	136	1017	444	440	441	441	442
Upper normal limit[9]					483	460	457	457
Upper normal limit[10]					457	445		

ECG = electrocardiogram.
[a]Bazett: QTc = QT/($RR^{1/2}$); Fridericia: QTc = QT/($RR^{1/3}$); Framingham: QTc = QT + 0.154 (1 – RR); Hodges: QTc = QT + 105 (^{1}/RR—1). Upper normal limits of QTc interval were determined by Luo et al.,[9] using 10,303 normal ECGs, and by Mason et al.,[10] using 46,129 normal ECGs.
[b]Day of hospital admission.

Bazett's formula) 4 days after discontinuation of galantamine and remained normal thereafter (Table 1). A repeat 24-hour electrocardiographic monitoring 3 weeks later showed sinus rhythm with an average heart rate of 76 beats/min (minimum 49, maximum 126), ventricular (n = 626) and supraventricular (n = 260) and ectopy. He was discharged, with an MMSE score of 21/30 on his usual medications except for galantamine and irbesartan. He remained asymptomatic for 6 months.

Australian Reports

A summary of cases of cardiac, neurologic, and gastrointestinal disorders associated with 3 AChIs reported to ADRAC (to May 31, 2007) is shown in Table 2. No cases of torsade de pointes were reported. There have been 5 fatal outcomes associated with galantamine, 11 with donepezil, and 3 with rivastigmine, including 3, 6, and 0 sudden deaths, respectively. When the number of reported cases was analyzed per 100,000 prescriptions written, an estimated 8.47 cases of delirium, confusion, or agitation; 3.76 of syncope; 2.82 of any type of arrhythmia or block; 3.29 of vomiting; 3.76 of diarrhea; and 1.41 of sudden death were related to galantamine. For rivastigmine, the corresponding figures were 14.10, 5.37, 1.30, 7.38, 3.36, and 0, respectively, and for donepezil 4.77, 1.28, 1.27, 2.13, 1.28, and 0.51, respectively. These data suggest that use of galantamine may be associated with the highest rate of arrhythmia and sudden death, with rivastigmine having the highest rate of delirium, syncope, and vomiting; no sudden death was reported. The rate of reported adverse effects with donepezil was lower than that with other AChIs (except that sudden death was not reported with rivastigmine). The total number of reports per 100,000 prescriptions was similar for galantamine and rivastigmine (52.3 and 57.1, respectively) but was about 2 times lower for donepezil (25.9).

Discussion

Although galantamine is widely used in the treatment of mild-to-moderate Alzheimer's disease and known to be well tolerated,[11] its cholinergically dependent gastrointestinal adverse effects are well recognized and cardiac disturbances have been reported.[5] To our knowledge, this is the first case report of a patient who developed withdrawal syndrome and multiple adverse effects when galantamine was reintroduced.

Galantamine is a reversible, competitive AChI and an allosteric modulator of nicotinic acetylcholine receptors. In our case, at least 2 mechanisms of clinical deterioration warrant discussion. First, galantamine discontinuation could lead to a withdrawal syndrome similar to that observed with donepezil.[12] Within 2 weeks of galantamine discontinuation, our patient's condition deteriorated, with obvious cognitive, functional, and behavioral decline, and therefore, the medication was resumed. Since galantamine's half-life is about 6 hours,[13] complete galantamine elimination (5 half-lives) will require approximately 30 hours, when the effects of sudden withdrawal may appear. Abrupt discontinuation of AChIs should be avoided.

Second, when galantamine was restarted in our patient, symptoms and signs of cholinergic excess ("toxicity") developed. These manifested as nausea, vomiting, diarrhea,

Table 2. Main Adverse Effects Reported to Australian Adverse Drug Reactions Advisory Committee

	Galantamine[a]		Donepezil[a]		Rivastigmine[a]	
Adverse Effect	A	B	A	B	A	B
Bradycardia	13	6.12	26	2.21	6	4.02
Syncope	8	3.76	15	1.27	8	5.37
Dizziness	10	4.71	7	0.59	7	4.69
Confusion/delirium/agitation	18	8.47	56	4.77	21	14.09
Arrhythmia	5	2.35	8	0.68	2	1.34
Bundle branch block	1	0.47	1	0.08		
Atrioventricular block			6	0.51		
Hypotension/circulatory collapse	6	2.82	5	0.42	2	1.34
Myocardial infarct/cardiac arrest	1	0.47	7	0.59	1	0.67
Nausea	12	5.65	25	2.13	17	11.41
Vomiting	7	3.29	25	2.13	11	7.38
Diarrhea	8	3.76	15	1.27	5	3.35
Fatal outcome	5	2.35	11	0.93	3	2.01
Sudden death	3	1.41	6	0.51	0	
Total number of reports	111	52.28	304	25.9	85	57.06
PBS prescription (to May 2007)	212,309		1,173,553		148,958	

PBS = Pharmaceutical Benefits Scheme.
[a]A = absolute number of reported cases; B = number of reported cases per 100,000 prescriptions.

weakness, delirium, syncope, hypotension, tachypnea, QTc interval prolongation, and arrhythmias, indicating overactivity of muscarinic and nicotinic sites of autonomic and somatic nerves, as well as of the central nervous system. This may be due to a significant upregulation of acetylcholine receptors caused by decreased synaptic concentration of acetylcholine due to abrupt discontinuation of galantamine after prolonged exposure to the drug.

Other causes for clinical deterioration were excluded. There was no metabolic abnormality, electrolyte imbalance, or signs of infection. Galantamine is the most likely cause, given that it was the only medication recently restarted. The patient's condition resolved with discontinuation of galantamine and irbesartan. The latter may contribute to hypotension but is unlikely to cause delirium, syncope, QTc interval prolongation, or arrhythmia (MEDLINE searched). Drug interaction is a theoretical possibility, as galantamine, irbesartan, and simvastatin undergo metabolism via the CYP3A4 isoenzyme. However, the role of this enzyme in metabolism of irbesartan is minimal,[14] and simvastatin was not ceased. Therefore, this mechanism is not likely to be significant.

Nausea, vomiting, and diarrhea, which were seen in our patient, are the most common adverse effects of AChIs linked to muscarinic enhancement. Urinary incontinence, hypotension, and bradycardia were other muscarinic effects in our case. Weakness, tachypnea, initial "normal" heart rate (followed by bradycardia) may be considered as mainly peripheral nicotinic effects, while CNS effects included delirium, restlessness, and agitation. There are few case reports of delirium,[15] syncope,[16] and QTc interval prolongation[7] associated with galantamine, and the mechanisms of these adverse effects remain unclear.

Although exposure to anticholinergic medications is associated with delirium,[17] and galantamine has been shown to produce effective reversal of this syndrome,[18] in our patient, galantamine precipitated agitated delirium. Irreversible inhibition of acetylcholinesterase by organophosphorous insecticides causes confusion and coma.[19] A case report described a 77-year-old man with early Alzheimer's disease who developed paranoid delusion after 4 days of galantamine use.[15]

All 3 AChIs used in Alzheimer's disease, according to a recent Cochrane review of pooled data from randomized controlled trials, have demonstrated a significantly higher incidence of syncope compared with placebo (3.4% vs 1.87%).[1] An AChI edrophonium has been used as a provocative agent for inducing syncope during head-up tilt-table testing.[20] In the majority of published studies, syncope was associated with donepezil and was usually due to new-onset bradycardia. However, in two-thirds of 16 patients treated with donepezil and hospitalized for evaluation of syncope, its cause was considered unrelated to donepezil.[21] There is one case report of syncope due to complete atrioventricular block that occurred after the first administration of galantamine.[16]

In our patient, the first syncopal episode was observed 14 months after he started galantamine and was associated with hypotension and sinus bradycardia. The next syncopal episode occurred 3 weeks after galantamine was restarted and was associated with severe hypotension, QTc interval prolongation, and arrhythmias. Although our patient had coronary artery disease, he developed syncope only when he received galantamine.

The most extraordinary feature of our case is the marked QTc interval prolongation with potentially serious cardiac arrhythmias. Our patient had a QTc interval of 503 msec (by Bazett's formula), an increase of more than 60 msec above the drug-free period. According to the Food and Drug Administration and the Committee for Proprietary Medicinal Products, any QTc interval over 500 msec or any increase of more than 60 msec is a cause for concern for the development of torsade de pointes. In 92% of 116 torsade de pointes cases related to treatment with noncardiac drugs, QTc intervals were more than 500 msec.[22] Acquired or congenital QTc interval prolongation can result in ventricular arrhythmias leading to syncope, seizures, and sudden death. The Cochrane review of pooled data from unpublished studies found that use of galantamine was associated with a significantly higher rate of unexplained death.[11] There has been one report of galantamine-associated QTc interval prolongation (518 msec) in a 47-year-old schizophrenia patient.[7] Another report described a 78-year-old man with Alzheimer's dementia who developed a QTc interval of 477 msec a week after commencement of rivastigmine.[8] Our patient did not have the confounding factors present in these 2 cases: use of psychotropic drugs in the first and hypokalemia in the second. He did not receive any other drugs that may precipitate QTc interval prolongation and did not have electrolyte abnormalities or hypothyroidism. In our patient, QTc interval prolongation was associated with nonsustained ventricular tachycardia revealed by Holter monitoring 3 days after galantamine was discontinued. Three weeks later, Holter recording was unremarkable. In our case, polymorphic ventricular tachycardia (torsade de pointes) could not be ruled out, given that there was no ECG monitoring during the first days after admission.

After organophosphate poisoning, QTc interval prolongation with ventricular arrhythmias, including torsade de pointes, often occurs between the third and fifteenth day. It is unrelated to serum electrolyte imbalance and is associated with high mortality, especially sudden and unexpected death.[19] The syncope experienced by our patient may well be related to QTc interval prolongation with dysrhythmia of torsade de pointes.

Although our patient had no family history of long QT interval syndrome or sudden cardiac death, a genetic car-

diac channelopathy could not be excluded. A silent mutation may predispose to QTc interval prolongation, which manifests only after exposure to a specific drug and/or other risk factors.[22] Increased vagal tone due to the increased amount of acetylcholine in the synapses is the most likely explanation for QTc interval prolongation and arrhythmias with AChIs. In a canine model, vagal nerve stimulation prolonged the QT interval by 1 msec for each 0.82 msec prolongation of effective refractory period.[23] Data from studies performed by pharmaceutical companies did not show statistically significant difference between galantamine- (or rivastigmine-) and placebo-treated patients with respect to QTc, PR, RR, and QRS intervals.[24] Our observation and other case reports are therefore important for postmarketing safety surveillance. The full spectrum of adverse effects of AChIs manifests only when a large number of patients with different comorbidities not present in the original trials are treated. The Naranjo probability scale indicates that in our patient, galantamine was the probable cause of QTc interval prolongation, syncope, and delirium because these reactions have been previously reported, the events appeared after galantamine was readministered and improved after removal of the drug, no alternative causes were identified, and the reactions were confirmed by objective evidence.[25]

Spontaneously reported adverse drug events are important means of postmarketing surveillance, especially when the events are rare. Analysis of the ADRAC database revealed that, in addition to well-known gastrointestinal effects, use of AChIs carries the risk of delirium, syncope, arrhythmias, other cardiovascular disturbances, and sudden death. Clearly, the reporting rates are not synonymous with incidence rates. Despite obvious limitations of spontaneous adverse event reporting (underreporting, missing clinical information, failure to identify comorbid conditions), this information should not be ignored. Galantamine had the highest rates of reported arrhythmias and sudden death in comparison with 2 other AChIs, while rivastigmine demonstrated the highest rates of delirium and syncope. As the total number of reports for each of these 2 newer drugs is about 2 times higher than the rates for donepezil, some reporting bias could not be excluded.

Conclusions

Our case, along with previously published reports and cases identified from the ADRAC database, illustrates that, in addition to gastrointestinal adverse symptoms, AChIs may lead to delirium, syncope, and prolongation of QTc interval, thereby increasing the risk of life-threatening arrhythmias. Such adverse effects may appear simultaneously after galantamine readministration, as in our patient. Administering, discontinuing, and restarting AChIs require vigilance and careful assessment of risk factors that may precipitate such events.

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Prolongación del Intervalo QT, Síncope, y Delirio con el uso de la Galantamina

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EXTRACTO

OBJETIVOS: Describir un caso de prolongación del intervalo QT, síncope, y delirio asociado al uso de la galantamina, y analizar casos similares reportados al Comité Consejero de Reacciones Adversas a Medicamentos Australiano, con el uso de los inhibidores de la acetilcolinesterasa.

RESUMEN DEL CASO: Un paciente de 85 años de edad con historia de demencia leve de etiología mixta (Alzheimer y vascular) había estado recibiendo galantamina de liberación prolongada en dosis de 8 mg diarios por un año y medio. Tres meses antes de su admisión a la sala de emergencia, el paciente había tenido un episodio de síncope con baja de presión arterial y bradicardia. Dos meses más tarde, la galantamina fue suspendida pero en el transcurso de 2 semanas, el paciente presentó marcado deterioro cognitivo, de comportamiento, y funcional por lo que la galantamina fue recetada de nuevo al paciente. Tres semanas más tarde, el paciente desarrolló síncope, delirio, hipotensión, prolongación del intervalo QT con arritmias cardíacas serias, además de vómito y diarrea. Su contaje sanguíneo y bioquímico era normal y no había indicio de infección. La galantamina y el irbesartan fueron suspendidos. En 6 días su delirio se resolvió completamente, y en 4 días el intervalo QT se acortó de 503–443 milisegundos (corregido con la fórmula de Bazett), y permaneció en un valor normal desde entonces.

REPORTES AUSTRALIANOS: La galantamina estuvo asociada a 18 casos de delirio/confusión, 8 de síncope, 13 de bradicardia, 6 de otro tipo de arritmias o anormalidades en la conducción, y 6 de hipotensión. El donezil estudo asociado a 56, 15, 26, 15, y 5 casos, y la rivastigmina a 21, 8, 6, 2, y 2 casos, respectivamente. Cinco de los casos reportados con la galantamina fueron fatales, 11 con el donepezil, y 3 con la rivastsigmina, incluyendo 3, 6, y 0 casos de muerte repentina, respectivamente.

DISCUSIÓN: Este caso junto con otros reportados previamente y los identificados mediante la base de datos australiana ilustran que los inhibidores de la acetilcolinesterasa pueden conllevar a delirio, síncope, hipotensión, y arritmias serias. La escala de probabilidad Naranjo indicó que la galantamina era probablemente responsable por la prolongación del intervalo QT, síncope, y delirio en este paciente.

CONCLUSIONES: La administración de la galantamina y otros inhibidores de la acetilcolinesterasa requieren de la vigilancia y evaluación de factores de riesgo que puedan precipitar una prolongación del intervalo QT, síncope, y delirio.

Traducido por Encarnación C Suárez

Prolongation de l'Intervalle QT, Syncope, et Delirium Suite à la Prise de Galantamine

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RÉSUMÉ

OBJECTIF: Décrire un cas de prolongation de l'intervalle QT, de syncope, et de delirium liés à la prise de galantamine et comparer à d'autres cas rapportés au Comité australien d'effets indésirables des médicaments concernant les inhibiteurs de l'acétylcholinestérase (AchE).

SOMMAIRE DU CAS: Un homme de 85 ans souffrant de démence a été traité par la galantamine à libération prolongée à raison de 8 mg par jour pendant 18 mois. Trois mois avant la présente hospitalisation, il a présenté un épisode de syncope avec hypotension et bradycardie. Deux mois plus tard, la galantamine a été cessée mais en moins de 2 semaines, le patient a présenté une détérioration marquée au niveau fonctionnel, comportemental, et cognitif ; la galantamine a donc été réintroduite. Trois semaines suite à la réintroduction de la galantamine, le patient a fait une syncope avec hypotension, un delirium, une prolongation de l'intervalle QT, et de graves arythmies cardiaques en plus de vomissements et de diarrhée. À l'admission, la formule sanguine et tous les tests de biochimie étaient normaux. Aucune infection n'a été détectée. La galantamine ainsi que l'irbésartan ont été cessés. Le delirium est complètement disparu après 6 jours et l'intervalle QT est passé de 503–443 millisecondes (corrigé selon la formule de Bazett) 4 jours après l'arrêt de la galantamine et est demeuré normal par la suite. La galantamine a été associée à 18 cas de delirium ou de confusion, 8 cas de syncope, 13 cas de bradycardie, 6 cas d'arythmies ou de troubles de conduction, et 6 cas d'hypotension. Le donépézil a, quant à lui, été associé respectivement à 56, 15, 26, 15, et 5 cas tandis que la rivastigmine l'a été dans 21, 8, 6, 2, et 2 cas respectivement. Cinq cas de décès ont été rapportés avec la galantamine, 11 avec le donépézil, et 3 avec la rivastigmine, incluant 3, 6, et 0 morts soudaines respectivement.

DISCUSSION: Ce cas, en plus des cas précédemment publiés et de ceux rapportés dans le cadre du programme australien de déclaration des effets indésirables des médicaments, montre que les inhibiteurs de l'AchE peuvent provoquer un delirium, une syncope, de l'hypotension, et des arythmies potentiellement mortelles. L'échelle de probabilité de Naranjo montre que la galantamine était la cause probable de la prolongation de l'intervalle QT, de la syncope, et du delirium observés chez ce patient.

CONCLUSIONS: La prise de galantamine et d'autres inhibiteurs de l'acétylcholinestérase doit être suivie rigoureusement et une évaluation des facteurs de risque pouvant précipiter une prolongation de l'intervalle QT, une syncope, ou un delirium doit être faite.

Traduit par Denyse Demers