Cesium chloride-induced torsades de pointes

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The chloride salt of cesium, a group 1A element, is gaining popularity as an alternative treatment of advanced cancers. Cesium chloride has primarily been used in cardiovascular research for arrhythmogenesis in animals because of its potassium-blocking effects. The present report describes a 45-year-old woman with metastatic breast cancer who experienced repeated episodes of torsades de pointes polymorphic ventricular tachycardia after several months of oral cesium therapy. There was a clear temporal relationship between cesium ingestion and the arrhythmia, which later resolved following discontinuation of cesium therapy. Serial cesium plasma and whole blood levels were measured over the ensuing six months and pharmacokinetic analysis was performed.

Key Words: *Alternative medicine; Arrhythmia; Cancer; Cesium chloride*

CASE PRESENTATION

A 45-year-old woman was brought to the emergency department by ambulance due to a syncopal episode during the night. Her medical history was significant for node-positive breast cancer (in 2005), for which she received a lumpectomy. She declined further surgery, radiation and chemotherapy. Instead, she chose alternative therapy in Mexico. Her therapy in Mexico was a modified Gerson regimen, which consisted primarily of a diet of natural juices, herbal vitamins, coffee enemas and electrolyte supplementation. After several months, her cancer progressed, prompting her to seek further care in California (USA), where she began a cesium chloride protocol. This regimen consisted of 3 g oral cesium chloride daily. She also received small doses of molybdenum, indium, rubidium, selenium, germanium, vanadium and daily potassium chloride. She was not taking any other prescription or nonprescription medications.

In the emergency department, her vital signs were stable. She was afebrile and her clinical examination was otherwise unremarkable. Her initial 12-lead electrocardiogram showed normal sinus rhythm with a prolonged corrected QT interval (QTc) of 516 ms (Figure 1). Her initial electrolyte panel was normal, with a potassium level of 3.9 mmol/L. Five hours later (09:55), she had a narrow complex tachycardia at a rate of 240 beats/min that responded to adenosine 6 mg intravenous push. At 13:13, her cardiac monitor revealed a pulseless torsades de pointes polymorphic ventricular tachycardia in the context of QT prolongation, which responded to 200 J of direct current cardioversion (Figure 2). At this time, amiodarone was initiated with 150 mg bolus, followed by a 1 mg/min infusion. Her postarrest 12-lead electrocardiogram showed normal sinus rhythm at 78 beats/min, with a QTc of 487 ms. At 16:26, she had another episode of torsades de

Des torsades de pointe induites par du chlorure de césium

Le sel chloré de césium, un élément du groupe 1A, gagne en popularité dans le traitement de deuxième ligne des cancers avancés. Le chlorure de césium est surtout utilisé chez les animaux dans la recherche cardiovasculaire de l'arythmogénèse parce qu'il bloque le potassium. Le présent rapport décrit le cas d'une femme de 45 ans ayant un cancer du sein métastatique qui avait subi des épisodes répétés de tachycardies ventriculaires polymorphiques du type torsades de pointe après avoir subi un traitement au césium par voie orale pendant plusieurs mois. Il y avait un lien temporel évident entre l'ingestion de césium et l'arythmie, qui s'est résorbée après l'arrêt du traitement au césium. On a mesuré les taux sériels de césium et de sang total dans le plasma au cours des six mois suivants et procédé à une analyse pharmacocinétique.

pointes polymorphic ventricular tachycardia, which again responded to 200 J of direct current cardioversion. At 18:32, following another episode of successfully cardioverted pulseless polymorphic ventricular tachycardia, the amiodarone was discontinued due to potential further prolongation of QTc (Figure 3). A lidocaine 100 mg intravenous bolus was administered, followed by a continuous infusion of 2 mg/min, in addition to magnesium sulfate. The patient remained on lidocaine at a rate of 2 mg/min until 07:17 the following morning, when the dose was decreased to 1 mg/min. Within 2 h of this dose reduction, she began experiencing runs of premature ventricular contractions. The lidocaine was increased to 2 mg/min until the following day, when she was started on oral mexilitine therapy. She remained stable over the following six days and was discharged home. An echocardiogram during this admission revealed normal left atrial and left ventricular size, with normal biventricular systolic function. Over the course of the following six days, her potassium remained below 4.0 mmol/L (the lowest measurement was 3.0 mmol/L) despite aggressive potassium supplementation. During the course of her admission, she did not receive any drugs known to cause QT prolongation (other than an initial dose of amiodarone), nor did she receive any known QT prolonging drugs before admission other than cesium chloride.

Over the course of the next 126 days, five whole blood and six serum cesium levels were collected (Figure 4). The patient refused further sampling due to the deterioration of her condition. An analysis was performed in the trace elements laboratory at the Children's and Women's Health Centre of British Columbia in Vancouver. The technique used was previously described in detail (1). The analysis revealed that cesium appears to follow the laws of first-order elimination, two compartments being within the blood, one within erythrocytes and one within serum or

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Figure 1) Presenting 12-lead electrocardiogram

Figure 2) First episode of torsades de pointes polymorphic ventricular tachycardia at 13:13 (pre- and postarrest)

Figure 3) Torsades de pointes polymorphic ventricular tachycardia at 18:32

plasma. The half-life in whole blood and serum was calculated to be 43 and 55 days, respectively. The physiological basis of the single outlier on the serum cesium concentration versus time graph is unknown.

DISCUSSION

The use of alternative therapies for treating cancer is a practice that is quickly gaining momentum. It has been estimated that more than 60% of patients will use vitamins and herbal therapies during their cancer therapy (2). Many alternative care providers claim superior cure rates and fewer side effects than conventional therapies, often using advertising on the Internet. The Internet is how the patient initially discovered the availability of cesium as an alternative treatment.

Cesium is a group 1A element and has chemical properties similar to lithium, potassium and sodium. The cardiovascular effects of cesium chloride have been known for decades and although the

precise mechanism of actions remains to be fully elucidated, it is believed that blockade of the potassium channels with resultant prolongation of repolarization is its primary mode of arrhythmogenesis (3). This property has made cesium chloride an agent of choice for induction of ventricular arrhythmias in the canine heart failure research model.

Over the past 20 years, cesium chloride has played an increasing role in the alternative treatment of various late-stage cancers. A rationale for the use of cesium in the treatment of cancer was described in 1984 by AK Brewer, who hypothesized that cesium established alkaline conditions inside neoplastic cells, which ultimately resulted in apoptosis. Evidence of the benefit of cesium in human cancer is limited to brief case reports and one case series published in 1984 by Sartori (4). This case series was subject to several major flaws including its uncontrolled nature, retrospective design and highly probable case selection bias,

making it unsuitable evidence to rationally support its use in humans. Despite this, cesium therapy appears to be a growing industry and is advertised, primarily through Internet-based cancer treatment groups, as a highly effective modality for the treatment of end-stage cancers. To date, of the six published case reports describing potentially fatal adverse cardiovascular events secondary to cesium therapy, ours is one of only three with a laboratory analysis of cesium pharmacokinetics and the only report with a comprehensive pharmacokinetic analysis and long-term follow-up (5).

Due to the prolonged half-life of cesium, it is not surprising that the arrhythmias did not occur earlier because it takes approximately 200 days to reach a steady state. Our patient had been taking cesium for approximately 150 days, and thus was only approaching steady-state serum concentrations at the time of her arrhythmias. Other case reports describe arrhythmias occurring after several weeks to months of therapy with cesium, consistent with our finding (5).

CONCLUSION

Cesium chloride is an unproven yet potentially toxic therapy in the treatment of cancer. Clinicians treating patients with cancer must obtain an accurate medication history that includes both prescription and nonprescription products. Furthermore, clinicians must familiarize themselves with current trends in natural therapies and be prepared to discuss the dangers of these options with their patients.

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Figure 4) A Serum cesium concentrations over 126 days. **B** Whole blood concentrations over 79 days