Case report

ST-segment elevation and ventricular tachycardia after ingestion of a common ornamental plant—a case report

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KEYWORDS

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Procainamide challenge test

ABSTRACT

Japanese yew is a widely used ornamental plant. However, most people are unaware that it is also a poisonous plant. It has potent cardiac toxicities that can lead to sudden cardiac death.

A 37-year-old female patient presented to the emergency room with altered mental status and sustained ventricular tachycardia (VT). Electrocardiogram (ECG) after cardioversion showed profound QRS prolongation and ST-segment elevation suggestive of either hyperkalaemia, acute myocardial ischaemia, or Brugada syndrome. Her electrolytes and coronary angiography were normal.

After improvement of the patient's mental status, she admitted that she has been consuming Japanese yew from her yard for several months. Few hours later, QRS duration normalised, but mild ST-segment elevation persisted in the right pre-cordial leads, making it more suspicious for Brugada syndrome. However, a procainamide challenge test and electrophysiology study failed to induce typical Brugada pattern ECG and VT. The absence of coronary artery disease and electrolyte disturbances points toward the fact that her arrhythmia and ECG changes are secondary to yew intoxication. The patient was monitored for a few days. She was haemodynamically stable and has not had any arrhythmia.

This case highlights the importance of public awareness of severe toxicity from Japanese yew or other yew plants. Yews contain taxines that are responsible for the ECG abnormalities due to its inhibitory effect on the cardiac sodium and calcium channels. They cause conduction abnormalities, VT, and ST-segment elevation that can resemble acute myocardial infarction, hyperkalaemia, and Brugada syndrome.

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Case presentation

A 37-year-old Caucasian woman presented to the emergency room because of decreased level of consciousness and slurred speech. On physical examination, blood pressure was 76/40; heart rate was 170; oxygen saturation was 98% on room air. She was diaphoretic and had slurred speech. Laboratory investigations showed a normal cell count and chemistries, troponin was 0.03; creatine phosphokinase (CPK), CPK-MB and liver function test were normal. Urine toxicology and alcohol level both were negative.

Cardiac monitoring revealed ventricular tachycardia (VT) at a rate of 175/min. The patient promptly received electric

cardioversion. The baseline electrocardiogram (ECG), 10 minutes after cardioversion, showed junctional rhythm with a rate of 62 beats/min and severe prolongation of the QRS complex (162 ms) with some Brugada-like pattern (Figure 1).

Further questioning of the patient revealed that she had been consuming Japanese yew leaves in the last 3–4 months, thinking that this plant might help with her flu.

A coronary angiogram was performed and showed normal coronary arteries with normal left ventricular function.

Subsequent ECG showed almost normalisation of the QRS complex but V1 showed incomplete right bundle-branch block with coved type ST-segment elevation of about 1 mm, and PR prolongation of 274 ms. Serial 12-lead ECGs showed regression of the ST-segment elevation and normalisation of the QRS and PR interval.

Her ECG was suspicious for Brugada syndrome, although QRS prolongation was more profound than what is expected

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Figure 1 Electrocardiogram 10 minutes after cardioversion showing profound QRS prolongation, ST-elevation (arrow) and T wave inversion in the right pre-cordial leads.

in typical Brugada pattern. It was unclear if these changes were all due to the toxic effect of yew or, the yew merely unmasked a true Brugada syndrome. To determine this possibility, an electrophysiology study with procainamide challenge test was performed. Programmed ventricular extra stimulus testing, with up to triple extra stimuli failed to induce any tachycardia. Procainamide at 10 mg/kg was infused >10 minutes and there was no inducible ST-segment elevation in the right pre-cordial leads.

Discussion

Japanese yew (*Taxus cuspidata*) is an ornamental plant that is used as a garden tree and widely grown in eastern Asia and eastern North America. It is a member of the genus *Taxus*, which includes *Taxus Baccata* (European yew), *Taxus canadensis* (Canada yew) and many others.

Historically, there is evidence of its previous use as a crude drug to treat diabetes, to promote diuresis, and as an emmenagogue. It is the source of paclitaxel, a chemotherapeutic agent used in the treatment of several cancers.¹ However, it has also been associated with cases of animals and humans intoxications, including death, mainly due to its arrhythmogenic effect on the heart.²

Chemical analysis of yew species leads to the isolation of several toxic alkaloids including taxine A and B. All parts of *Taxus* plants are toxic except the pulp, or aril, which contains very little taxine.³ Taxine B accounts for 30% of the total taxine fraction and taxine A represents only 1.8% of the taxine fraction.⁴ The taxines inhibit both the cardiac calcium channel (I_{Ca}) and the fast cardiac sodium channel (I_{Na}) in a dose-dependent manner. This can lead to suppression of cardiac contractility, sinoatrial node automaticity, atrioventricular (AV) node conduction and also depolarisation and repolarisation abnormalities.⁵

The profound QRS prolongation in our case (Figure 1) is mainly due to the severe inhibition of the fast cardiac sodium

Figure 2 Electrocardiogram 8 hours after cardioversion. Persistent mild ST-elevation in lead V1 with T-wave inversion.

channel by the taxines, during the phase 1 of the action potential.⁵ The degree of QRS prolongation depends on the severity of sodium channel inhibition.

The ST-segment elevation in the right pre-cordial leads few minutes after cardioversion (Figure 1) and in the lead V1, 8 hours after the event (Figure 2), resembles those seen in Brugada syndrome. It is secondary to the preferential sodium channel blockade of right ventricular epicardial cells leading to the loss of action potential dome in the right epicardium but not in the endocardium. This can also result in spatial dispersion of repolarisation that can lead to VT due to phase 2 re-entry.⁴

Because an underlying congenital Brugada syndrome or other sodium channel susceptibility disorder could not be excluded, a procainamide challenge test was performed and it was negative, indicating that the ECG findings including VT and ST-elevation were purely caused by the toxic effects of yew.

Conclusion

Our case was suspicious of induced Brugada syndrome; however, it does not meet the criteria for Brugada phenotype. A case of Brugada ECG secondary to yew intoxication was previously reported in the literature.⁶ These two cases suggest that a combination of I_{Na} and I_{Ca} blockade could be more effective in reproducing the ECG pattern of Brugada syndrome. This was demonstrated in an in vitro study by Fish and Antzelevitch.⁴ However, the clinical value of this combined test has not been fully established. This case emphasizes again the importance of public awareness to the cardiac toxicity of yew plants.

References

- Shigemori H, Kobayashi J. Biological activity and chemistry of taxoids from the Japanese yew, *Taxus cuspidata*. *J Nat Prod* 2004; 67:245–56.
- Yersin B, Frey JG, Schaller MD, Nicod P, Perret C. Fatal cardiac arrhythmias and shock following yew leaves ingestion. *Ann Emerg Med* 1987;16:1396–7.

3. Krenzelok E, Jacobsen T, Aronis J. Is the yew really poisonous to you? J Toxicol Clin Toxicol 1998;36:219.
4. Fish JM, Antzelevitch C. Role of sodium and calcium channel block in unmasking the Brugada syndrome. Heart Rhythm 2004; 1:210–7.
5. Wilson CR, Sauer J, Hooser SB. Taxines: a review of the mechanism and toxicity of yew (*Taxus* spp.) alkaloids. Toxicon 2001; 39:175–85.
6. Veltmann C, Borggreffe M, Schimpf R, Wolpert C. Yew causes Brugada ECG. Circulation 2009;119:1836–7.