中文題目：一個治療毛地黃中毒心室頻脈易犯的錯誤

英文題目：A pitfall in treating digoxin toxicity related ventricular arrhythmia

作　　者：鄭孟瑜[1]，張坤正[1,2]，林圀宏[1,2]，陳建佑[1,2]，林晏年[1,2,3]

服務單位：中國醫藥大學附設醫院內科部[1] 中國醫藥大學附設醫院心臟血管系[2]
中國醫藥大學臨醫所[3]

Case presentation

The 78-year-old housewife with heart failure, chronic atrial fibrillation, hypertension presented to emergency department with one-week worsening nausea, vomiting, and weakness. She also reported chest tightness and developed frequent syncope. Her medication included furosemide 40mg, spironolactone 25mg, amiodarone 100mg, digoxin 0.25mg, aspirin 100mg, and olmesartan 40mg. On visiting, her consciousness was clear. Blood pressure measured 150/74mmHg, while heart rate was 52beats per minute. Physical examination was remarkable for irregular slow heart sound with frequent extrasystole. ECG showed junctional bradycardia with non-sustained bidirectional ventricular tachycardia (Figure1) and polymorphic ventricular tachycardia. For worsening hypotension, she received dopamine 24mcg/min/Kg and developed frequent pulseless sustained bidirectional ventricular tachycardia. Frequent defibrillation and CPCR were applied. Laboratory test were remarkable for creatinine 0.96 mg/dl, potassium 2.9 mmol/L and digoxin level 3.03ng/mL. Digibind (Digoxin-specific antibody (Fab) fragments 38mg/vial) 3 vial were therefore administered. The bidirectional ventricular tachycardia subsided gradually, while Torsades de pointes developed. Frequent defibrillation with CPCR were re-initiated. Follow up laboratory test showed Potassium 2.3mmol/L, Digoxin level <0.2 ng/mL. She then received aggressive correction of potassium with gradual resolution of residual ventricular arrhythmia. Unfortunately, despite intensive management and care, she developed multiple organ failure and was expired at the same day for prolonged CPCR and frequent defibrillation.

Discussion

Digoxin is commonly used for heart failure and supraventricular tachyarrhythmia. It is its narrowing therapeutic index that causes the nature of easy overdose and induction of life-threatening arrhythmia. Bidirectional ventricular tachycardia is one of typical digoxin induced life-threatening arrhythmias, while other arrhythmias, such as atrioventricular block, ventricular fibrillation, junctional bradycardia are not infrequently encountered.

The mechanism of digoxin toxicity is increasing intracellular calcium in myocardial cells with overloading and inhibiting the sodium pump (Na/K-ATPase) in

the cell membrane that delayed afterdepolarization. Besides, parasympathetic activation predisposes patients to develop sinus bradycardia and AV block.
The digoxin toxicity can be exacerbated with electrolyte imbalance and drug-drug interaction. Amiodarone is well known for inhibition of P-glycoprotein and resulting in slow down metabolism of digoxin. Hypokalemia and hypercalcemia will also worsen the cellular environment and change membrane potentiall, that provoke uncontrolled ventricular arrhythmia.

Digibind (Digoxin-specific antibody (Fab) fragments), antidote of digoxin, showed pivotal role in treating digoxin toxicity and its associated fatal arrhythmia. But, Digibind may also cause hypokalemia which may induce Tosades de pointes via early afterdepolarization mechanism. Aggressively correction potassium should be applied at the beginning of management of this patient.

In conclusion, digoxin toxicity with life-threatening arrhythmia is emergency and critical condition. Give Digibind is mandatory under the life-threatening arrhythmia. Aggressive correction and closely monitor potassium level should never be overemphasized in treating patients with digoxin overdose.

Figure1