# CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

# **Electrocardiogram Quiz - Case 27**

A 64-year-old man was admitted to our hospital within the framework of follow up due to ischemic cardiomyopathy. The patient was hemodynamically stable and in an excellent clinical condition. The 12-lead surface electrocardiogram (ECG) is depicted in figure 1.

## Questions

- a. What abnormalities are depicted on the 12-lead ECG (fig. 1)?
- b. What is the clinical significance of the depicted abnormalities?

### Comment

Digoxin effect refers to the presence on the ECG of downsloping ST depression with a characteristic "sagging" appearance, flattened, inverted, or biphasic T waves, and shortened QT interval. Other electrocardiographic features of digoxin effect include mild PR interval prolongation of up to 240 ms (due to increased ARCHIVES OF HELLENIC MEDICINE 2016, 33(1):137–138 APXEIA ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2016, 33(1):137–138

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vagal tone), prominent U waves, peaking of the terminal portion of the T waves, and J point depression (usually in leads with tall R waves). It should be noted that the presence of digoxin effect on the ECG is not a marker of digoxin toxicity. It merely indicates that the patient is taking digoxin.

The incidence of digitalis toxicity has declined in recent years, due to decreased use of this drug along with improved technology for monitoring of drug levels and increased awareness of drug interactions. Nevertheless, cardiac glycoside toxicity continues to be a problem in everyday clinical practice due to the wide use of digoxin (a preparation of digitalis) and its narrow therapeutic window. Approximately 0.4% of all hospital admissions in the United States are related to digitalis toxicity, while about 1.1% of outpatients on digoxin and 10–18% of people in nursing homes develop this toxicity. In 2011, the American Association of Poison Control

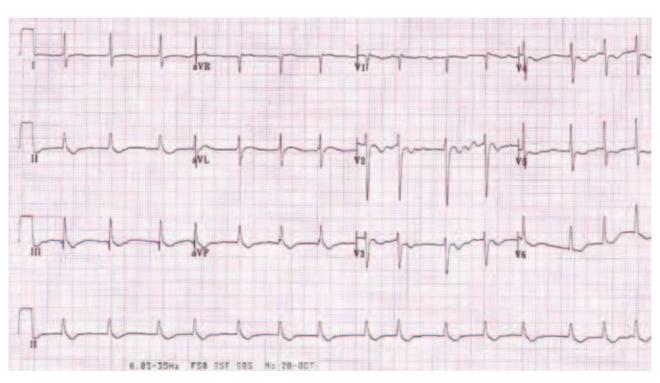


Figure 1

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Centers (AAPCC) reported 1,336 single exposures to plant cardiac glycosides and 1,601 single exposures to drug cardiac glycosides.

Digoxin can cause a multitude of dysrhythmias, due to increased automaticity and decreased atrioventricular (AV) conduction. The classic dysrhythmia associated with digoxin toxicity is the combination of a supraventricular tachycardia with a slow ventricular response, e.g. atrial tachycardia with block. Other arrhythmias associated with digoxin toxicity include frequent premature ventricular contractions, including ventricular bigeminy and trigeminy, sinus bradycardia or slow atrial fibrillation (AF), any type of AV block, regularized AF (AF with complete heart block and a junctional or ventricular escape rhythm), and ventricular tachycardia (both polymorphic and bidirectional).

### References

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