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Recurrent Syncopeal Attacks after Treated Dysuria

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ABSTRACT

A 65-year-old man is presented who was started on finasteride for postoperative dysuria. The dysuria resolved, but he had a series of syncope attacks, which were due to polymorphic ventricular tachycardia. The authors discuss the possible causes and draw attention to the significance of hypogonadism and post pause U wave augmentation, which indicates a reduced repolarization reserve.

Keywords: Syncope; Hypogonadism; Finasteride; Post pause U wave augmentation; Repolarization reserve

CASE REPORT

A 65-year-old obese hypertensive man with atrial fibrillation was treated with a combination of ACE inhibitor, diuretics and NOAC therapy. Medical history includes androgen deprivation therapy (degarelix 80 mg subcutaneous for one year) two years earlier for locally invasive and metastatic prostate carcinoma. Patient gained 30 kg during treatment (170 kg, BMI 50). Prior to admission, patient underwent total knee replacement where a detailed preoperative examination, did not reveal abnormalities which would contraindicate orthopedic surgery.

An ECG showed atrial fibrillation with bradyarrhythmia, right bundle branch block and left anterior fascicular block, which were already known from previous evaluations. Finasteride treatment was initiated due to prolonged dysuria and urinary retention post-surgery. The dysuria resolved, but had experienced several syncopal attacks at home.

The pre-surgery ECGs and after the syncopal attacks are shown in **Figures 1** and **Figure 2**.

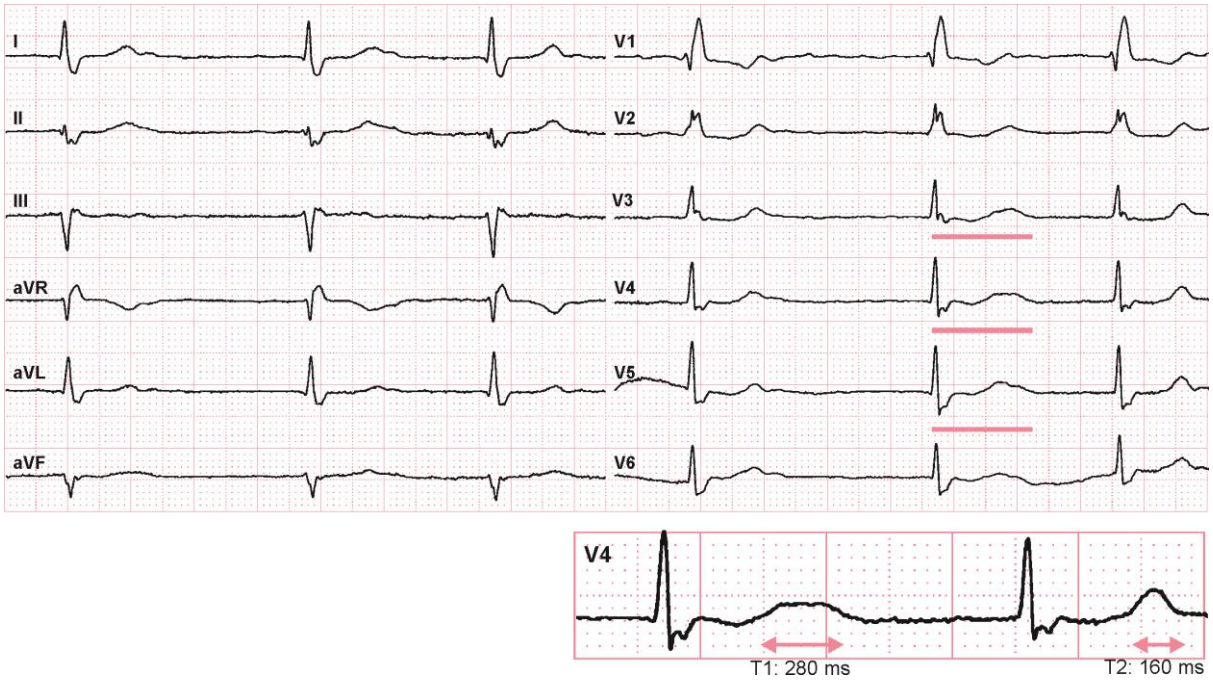

Figure 1

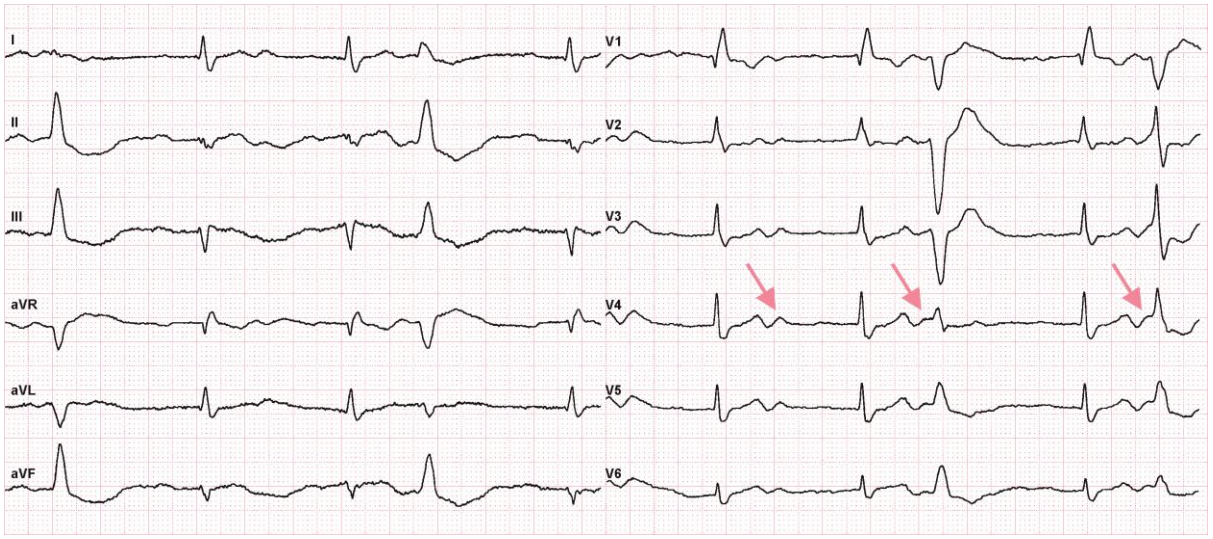

Figure 2

Based on the ECG, it is most likely that the syncopal episides were caused by polymorphic ventricular tachycardia, as there is an observed postpause excessive augmentation of the T-wave amplitude leading to a bizarre T-wave (Figure 1) and the pronounced U-waves after the syncopal episodes indicate this (Figure 2). Three of the most important ECG characteristics of hypokalemia were present: T:U ratio equal to 1 in V3-4 leads, U-wave greater than 1 mm, and frequent ventricular ectopy. The first and most important step was to closely monitor the patient and find the cause of the QTU prolongation. During monitoring, polymorphic ventricular tachycardia occurred (Figure 3), after which a DC shock was followed by a return of sinus rhythm with Grade II AV block, T-wave inversion, and prolonged QT time. Laboratory tests confirmed mild

hypokalemia (3.3 mmol/L, normal: 3.5-5.2 mmol/L), normal magnesium levels (0.71 mmol/L, normal: 0.70 to 0.91 mmol/L) and low testosterone levels (5.1 nmol/L, normal: 6.7-27 nmol/L) and low free testosterone index (18%). In addition to the rapid administration of potassium and magnesium, testosterone-lowering finasteride and diuretic therapy were discontinued, and then a DDD ICD was implanted for the initially high 100 bpm stimulation. No further malignancies were detected during the one-week follow-up. With normalization of potassium (4.3 mmol/L) and testosterone levels (10.43nmol/L, 36%), the QT prolongation was resolved, but post-pause T-wave prolongation was maintained (Figure 4).

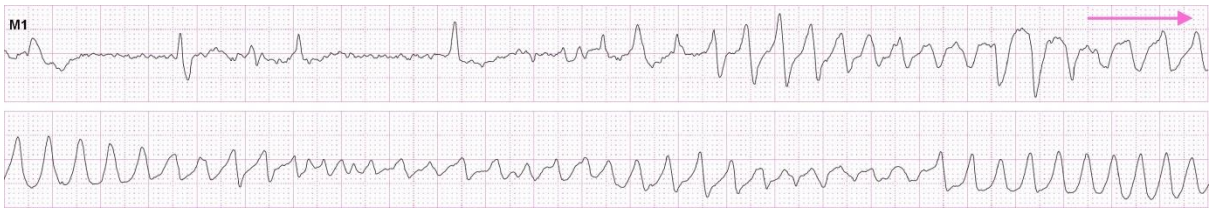

Figure 3

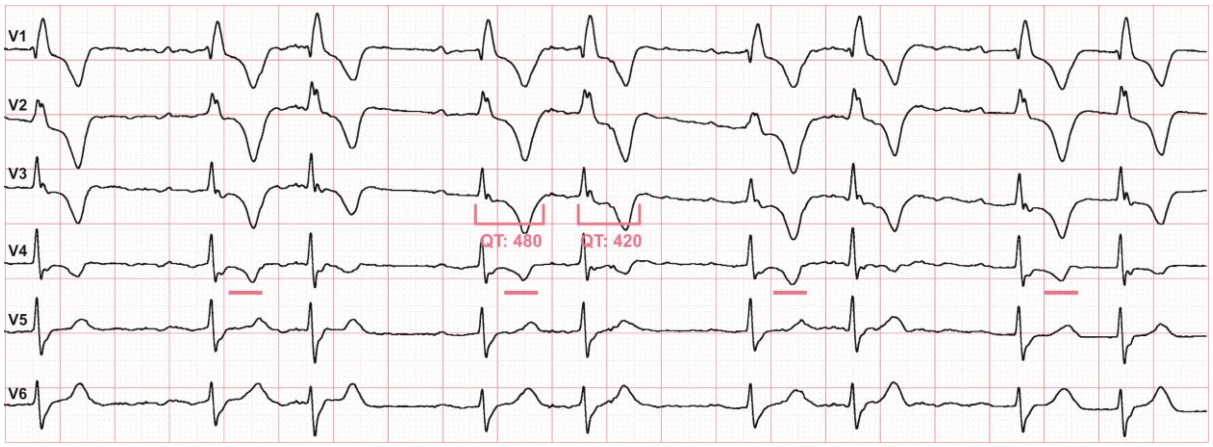

Figure 4 with QT values

DISCUSSION

The three most important factors leading to torsade de pointes polymorphic ventricular tachycardia causing syncope are:

A. Bradyarrhythmia-induced QT prolongation, which is seen in both Figure 1 and Figure 5 and is observed independently of other factors that reduce repolarization reserve.[1]

B. Hypokalemia, which in this case was mild, but is a common factor that can decrease repolarization reserve. Low extracellular potassium increases I Ks and I k1 according to the Nernst equation but paradoxically reduces I Kr and prolongs QT interval and increases drug-induced I Kr block. In addition, hypokalemia may have played a role in the appearance of ventricular ectopies, which is a trigger factor for torsade de pointes polymorphic ventricular tachycardia.

C. Low testosterone levels, which can also cause QT prolongation and TdP.[2]

Interestingly, previous testosterone-lowering therapy did not result in malignant ventricular arrhythmias, suggesting that potassium-wasting diuretic-induced hypokalemia was required to further reduce repolarization reserve and trigger ventricular extrasystoles.

The case demonstrates that it should be possible to recognize diminished repolarization reserve. However, such situations are usually complex, and attenuated repolarization reserve can be recognized only retrospectively, sometimes only after a fatal outcome.[3]

CONCLUSION

Our case highlights the dangers of drug therapy in high-risk patients that may result in hypogonadism.

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