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Electrocardiogram Quiz – Case 6

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A 76-year-old male was admitted to our hospital in order to undergo a scheduled coronary angiography due to a history consistent with exertional angina pectoris. The patient's personal history included a poorly controlled arterial hypertension, dyslipidemia and a mild chronic renal failure. The laboratory tests, including a complete set of cardiac biomarkers and electrolytes were within normal limits. The patient underwent percutaneous coronary angioplasty and received a drug eluting stent to the left anterior descending branch. The patient was scheduled to be discharged on the next day; however, the ECG depicted below had been the reason for further investigations.

Questions

a. What could be the reason for the further investigations ordered for the patient?
b. How would you treat the patient having set the diagnosis?

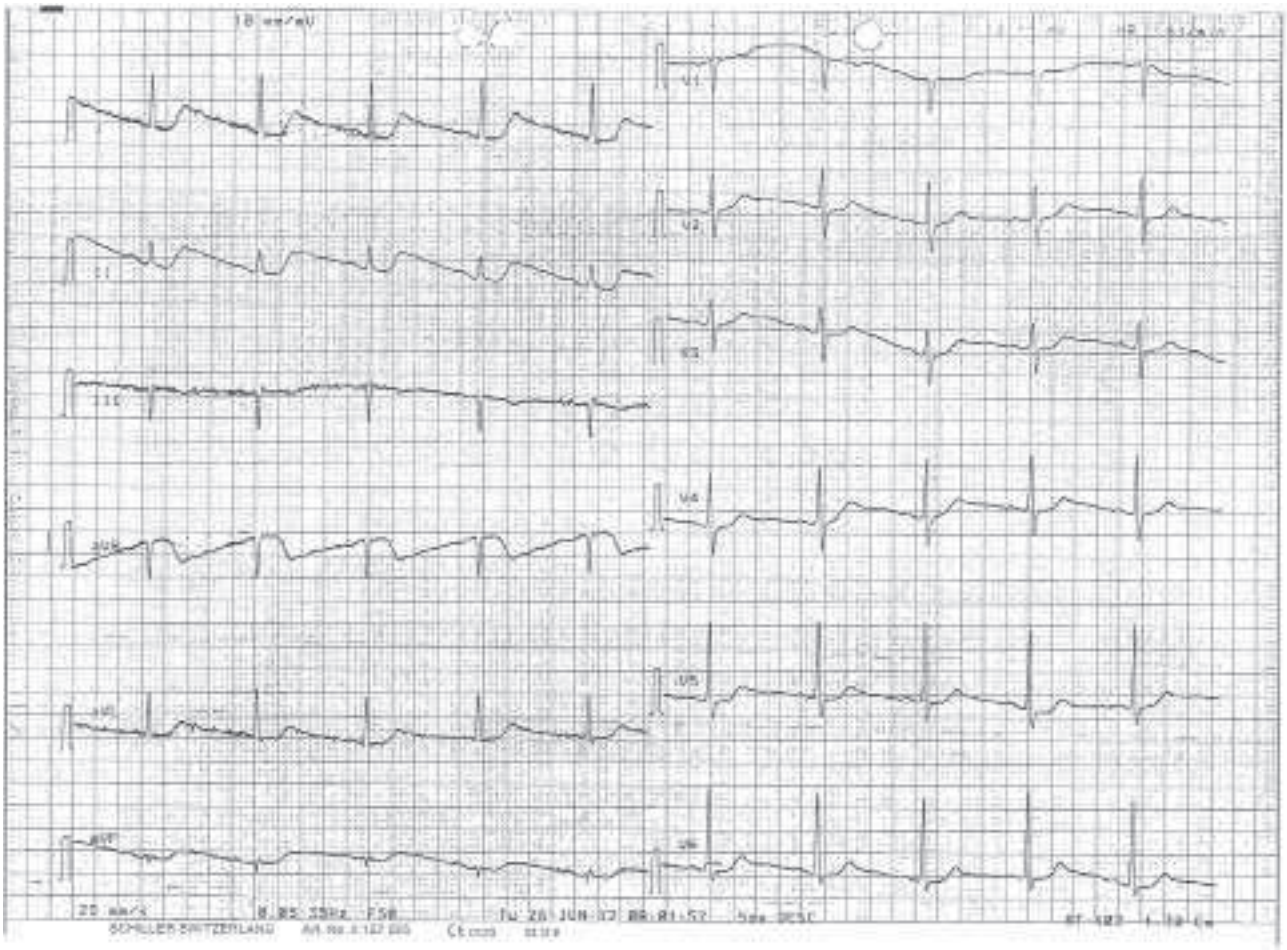

Figure 1

Comment

The reason for further investigation of the patient has been the biphasic and depressed ST segment pronounced in I, II, aVR and aVL leads on the 12-lead ECG. The differential diagnosis of the afore-mentioned ST segment morphology includes ischemia, hypokalemia and pericarditis. Bedside echocardiography demonstrated no pericardial effusion, whether a new complete set of cardiac biomarkers excluded acute ischemia. However, serum potassium levels of the patient on the day of scheduled discharge were 3.4 mmoL/L (reference levels 3.5–4.6 mmoL/L), suggesting a mild transient hypokalemia.

Hypokalemia is the most common electrolyte abnormality encountered in clinical practice. Potassium values of <3.6 mmoL/L are seen in over 20% of hospitalized patients. As many as 10% to 40% of patients on thiazide diuretics and almost 50% of patients resuscitated from out-of-hospital ventricular fibrillation have low potassium levels. Hypokalemia results from decreased potassium intake, transcellular shift, and, most commonly, increased renal or extrarenal losses.

The electrophysiological effects of hypokalemia include the following: Decrease in conduction velocity, shortening of the effective refractory period, prolongation of the relative refractory period, increased automaticity and early afterdepolarizations.

Hypokalemia may produce several electrocardiographic changes, especially when there is total body depletion of both potassium and magnesium. The most common repolarization alterations include decreased amplitude and broadening of the T waves, ST segment depression, prominent U waves, and T and U waves fusion in severe hypokalemia ("T-U-P" syndrome or pseudoatrial flutter). Conduction abnormalities, particularly in the presence of coexistent hypomagnesemia, include an increase in QRS duration, atrioventricular block, increase in P wave amplitude and duration, slight prolongation of the P-R interval, ventricular extrasystoles, and malignant ventricular arrhythmias such as ventricular tachycardia, torsades de pointes, and ventricular fibrillation. Electrocardiographic changes are not common with mild to moderate hypokalemia, unlikely in our case, and it is only when serum concentrations of potassium are below 2.7 mmoL/L that changes reliably appear. Finally, there are early reports describing profound hypokalemia mimicking acute myocardial infarction.

Our patient was treated with per os potassium substitution, until normal serum values were restored. He was discharged on the next day with a normal ECG and explicit instructions for potassium reassessment.

In conclusion, hypokalemia comprises a potentially life-threatening condition that requires immediate action in the everyday, routine clinical practice.

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