(*Refer to page 48*)

Answer: Brugada syndrome

The patient did not have any cardiac symptoms or any cardiac enzyme changes suggestive of an acute myocardial infarction (AMI). Although not always present, the finding of reciprocal ST segment depression in opposing leads favours myocardial infarction. This was also not present in the patient's ECG. Echocardiogram did not show any regional wall motion changes that could be seen in patients with myocardial infarction. Thus the probability of this patient having AMI based on the ECG alone is very unlikely.

In hyperkalemia there are tall T waves and conduction abnormalities which were also not present in the ECG.

The ECG shows typical findings of Brugada syndrome. [1-3] There is right bundle branch block (RBBB) with ST segment elevation in V1-2 without reciprocal ST depression. The descending arm of ST elevation in V1 and V2 is very abrupt rather than coved as in AMI. Brugada syndrome is a sodium channel disease that is associated with sudden cardiac death due to ventricular tachycardia (VT).

Brugada syndrome has a genetic link. Illnesses like any febrile illness and diabetic ketoacidosis can occasionally unmask the ECG findings of Brugada syndrome.

Fleccanide test can be done in patients with atypical ECG changes but suspected to have Brugada syndrome. High risk patients are those who have history of palpitation, syncope or presyncope and family history of sudden cardiac death. Electrophysiology study is recommended in some patients.

- Average estimated to be between 5 to 12 per 10,000 population.
- Estimated to be responsible for at least four percents of all sudden deaths and at least 20% of sudden deaths in patients with structurally normal hearts.
- Endemic in Southeast Asia and Japan
- Male preponderance (8 to 1).
- Also known as Lai tai (death during sleep"), bangungut ("to rise and moan in sleep"), *pokkuri* ("sudden and unex pectedly ceased phenomena").

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