

Part
one

1. (c). This history is typical of Gilbert's syndrome. It affects between 5 and 10% of Europeans, a third of whom do not know they have it. The fact that he feels so well tends to rule out the other diagnoses. It is caused by an inherited autosomic recessive single genetic change that leads to the liver being less efficient than normal in clearing bilirubin.

Part
two

2. (c), (d), and (e). The blood tests confirm almost completely that this is Gilbert's syndrome, but it is advisable to make absolutely sure that there is no other cause for his jaundice, and (c) and (d) will do so. There should be no bilirubin on the dipstick as unconjugated bilirubin (excess of which is the consequence of Henry's genetics) is water insoluble. Bilirubinuria should make you look for another diagnosis.

Part
three

3. (a), (b), (c), (d), and (e). All of these results together form the definitive characteristics of Gilbert's disease.

Part
four

4. (e). There is no need to investigate further. It is important not to make the patient anxious, and to explain that Gilbert's syndrome is not an illness, and has no dire consequences. Going into detailed testing may tend to suggest that it is more serious than you are saying, and may even lead to mistrust and raise doubts in the patient's mind.

Part
five

5. (a), (b), (c), (d), (e), and (f). All these statements are part of the management of the syndrome. As for drug avoidance, among them are drugs that may precipitate jaundice in people with Gilbert's, such as gemfibrozil, atazanavir, and indinavir. Although having the syndrome may alter paracetamol metabolism, it does not seem to increase the risk of toxicity. In theory people with Gilbert's syndrome may be at higher risk than usual of muscle pains when taking statins with gemfibrozil, but this has also not been confirmed by case reports.



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