

PEER-REVIEW REPORT

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Title: Acute inferior myocardial infarction in a young man with testicular seminoma: A case report

Reviewer's code: 00978063

Position: Peer Reviewer

Academic degree: MD

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Scientific quality	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Very good <input type="checkbox"/> Grade C: Good <input type="checkbox"/> Grade D: Fair <input type="checkbox"/> Grade E: Do not publish
Language quality	<input type="checkbox"/> Grade A: Priority publishing <input checked="" type="checkbox"/> Grade B: Minor language polishing <input type="checkbox"/> Grade C: A great deal of language polishing <input type="checkbox"/> Grade D: Rejection
Conclusion	<input type="checkbox"/> Accept (High priority) <input type="checkbox"/> Accept (General priority) <input checked="" type="checkbox"/> Minor revision <input type="checkbox"/> Major revision <input type="checkbox"/> Rejection
Re-review	<input checked="" type="checkbox"/> Yes <input type="checkbox"/> No
Peer-reviewer statements	Peer-Review: <input checked="" type="checkbox"/> Anonymous <input type="checkbox"/> Onymous Conflicts-of-Interest: <input type="checkbox"/> Yes <input checked="" type="checkbox"/> No

SPECIFIC COMMENTS TO AUTHORS

This is quite an interesting case report about a young man who suffered an apparent acute myocardial infarction (AMI) following chemotherapy for testicular carcinoma: treatment had included cisplatin. Coronary angiography was normal except for the presence of an apparent coronary stenosis and associated thrombosis (not directly visualised) near the crux. The study is an important reminder of the risk of ischemic events, even after chemotherapy, in such situations. While the report is interesting, the authors have missed several opportunities:- (1) What caused the apparent AMI? The lesion looks like an area of coronary vasoconstriction rather than atheroma, as best as I can guess. Was the RCA injected with NTG to see whether the "lesion" disappeared? If not, why not? Cisplatin is known to induce vascular endothelial dysfunction, perhaps mainly via impairment of nitric oxide synthase activity, but also perhaps via impairment of hydrogen sulphide availability. The authors need to incorporate some of this information into their report. (2) Was Takotsubo excluded.? I think that this is a very minor point, because the chemo agents concerned do not usually cause TTS, but for innocent readers this is a big issue. The authors should provide, at the very least, echo results. (3) How was treatment selected? There is actually some justification for the atorvastatin, but the beta-blocker is quite bizarre in the context of possible coronary spasm. The authors can hardly claim that the infarct was large enough to leave the patient with heart failure and/or risk of VF. (4) Was this the only episode of chest pain? This is an important point either way. Most patients with coronary spasm have recurrent episodes. Normal treatment would include calcium antagonists, and it is worth knowing that thrombus formation has now been implicated in the pathogenesis of coronary spasm.