Acute aortic dissection with sporadic aortic calcifications during chemotherapy with sunitinib

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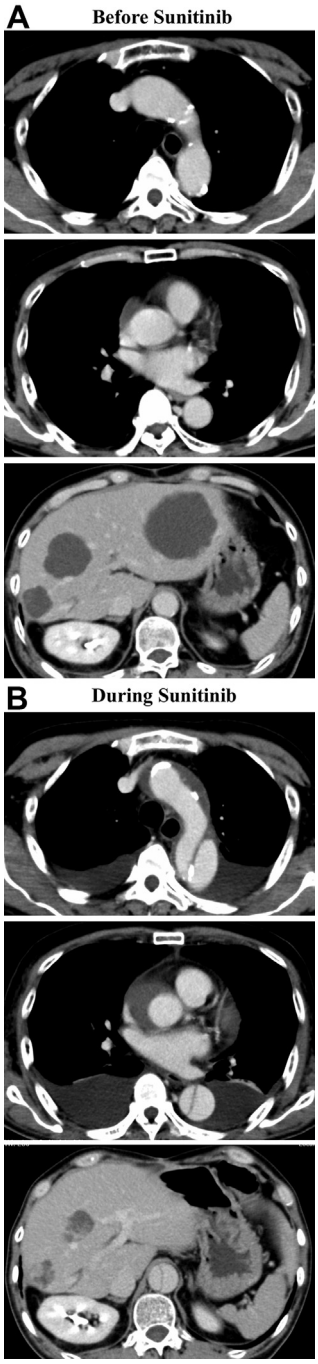
A 66-year-old man with imatinib-resistant metastatic liver tumors of gastrointestinal stromal tumor started chemotherapy with sunitinib. Baseline computed tomography showed sporadic aortic calcifications and liver tumors (*A*). His systolic blood pressure increased to 160 mm Hg during chemotherapy[1] and decreased to 130 mm Hg with administration of antihypertensive medication. During his sixth cycle of chemotherapy, he developed an acute aortic dissection (AAD, Stanford A) with thrombosed false lumen of the ascending aorta despite good control of blood pressure and reduction of the liver tumors (*B*). The entry site of the AAD was already calcified before chemotherapy as in *A*.

The presumable mechanism of the onset of AAD during chemotherapy with sunitinib is interruption of vascular endothelial growth factor function, which could be related to vascular stability.[2] In this case, the AAD was considered to have developed on the basis of simultaneous hypertension due to sunitinib and vascular fragility due to both calcification and sunitinib presence. In daily practice, careful attention should be paid to the onset of AAD in patients with sporadic aortic calcifications during chemotherapy with sunitinib.



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