Aortic Dissection in a Patient on Axitinib: Fatal Presentation of a Potential Complication

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Introduction
Axitinib is a selective second-generation tyrosine kinase inhibitor which blocks angiogenesis and tumor growth by inhibiting vascular endothelial growth factor (VEGF) receptors.

It is used for the treatment of renal cell carcinoma.

Hypertension is a well known side effect of axitinib. There are few reported cases of aortic dissection in patients on axitinib with the mechanism behind this being unclear.

Case Presentation
An 81-year-old male with medical history of metastatic renal cell carcinoma, resistant hypertension, and chronic known aneurysms of mid-ascending and mid-descending aorta.

Chief Complaint: Acute four minute episode of bilateral vision loss that self resolved.

History of Present Illness: Patient was at home not doing any activity when he had sudden onset loss of vision in both eyes lasting for about four minutes. Vision returned spontaneously prior to arrival to the emergency department. It was not associated with any focal weakness, sensory disturbance, or facial droop.

Vitals: Blood pressure 123/90 mm Hg, otherwise unremarkable.

Workup: Patient was a stroke alert in the emergency department. CT head without contrast and CTA head and neck were done. This did not show acute ischemic or hemorrhagic infarct but showed an acute dissection of aortic arch. CTA of chest showed dissection of the ascending aortic aneurysm compressing the true lumen.

Hospital Course: Upon evaluation by Cardiothoracic surgery, he was immediately taken to the operating room and a Bentall Procedure was planned. Intra-operatively, his dissection was noted to extend into the left and right coronary ostia. Immediate post-operative course was complicated by hemorrhagic shock, leading to the patient’s death.

Discussion
Aortic dissection is a life-threatening condition reported in about 0.3% of the patients treated with VEGF inhibitors for cancer.

Most common risk factors of aortic dissection are hypertension, atherosclerosis, and connective tissue disorders.

VEGF inhibitors cause capillary rarefaction and nitric oxide downregulation, which is postulated to contribute to the development of hypertension.

The exact mechanism behind aortic dissection and VEGF inhibitors remains elusive. VEGF is expressed in smooth muscle cells in large vessel walls and is highly expressed in atherogenic lesions. Inhibiting this is postulated to possibly destabilize the vessel wall.

In our patient, we suspect that the addition of axitinib created a perfect storm due to his pre-existing aneurysms and hypertension that led to his dissection.

This case adds to the growing literature reporting the potential link between aortic dissection and VEGF inhibitors and aims to increase awareness of this association among the cardio-oncology community.

References

Disclosures
None of the above named authors have any disclosures.