**A Rare Case of Cefazolin Induced Coagulopathy and INR Derangement**

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**Introduction**

Cefazolin is a first-generation cephalosporin which is indicated in a wide variety of clinical settings. Particularly, this antibiotic has been used before, during, and after surgical procedures to prevent secondary infections. There are reports of patients having severe coagulopathic derangements while receiving intravenous Cefazolin. Such effects are more commonly seen in patients with diabetes, renal functional impairment, and malnourishment. The exact mechanism of this association remains unknown.

**Case Presentation**

A 53 y.o. female with a history of duodenal adenocarcinoma status-post Whipple Procedure, bicuspid aortic valve, and heart failure presented for evaluation of chest pain, decreased energy, and dyspnea on exertion. Echocardiography revealed severe aortic stenosis and an ejection fraction of 25-30%. She subsequently received mechanical aortic valve replacement (AVR) with no operative complications. She received Cefazolin 2g IV three times daily on the first and second day of admission. Cardiology recommended starting the patient on Warfarin, per protocol for mechanical AVR. However, the patient was not eligible for this medication due to an increase in international normalized ratio (INR) of unknown origin. The patient’s baseline INR was 1.0. On the third day of admission INR rose to 3.6, with a repeat INR of 4.7 and 4.9 that day. She had normal liver studies, mixing studies, and no evidence of active bleeding. She had not received any Warfarin or anticoagulation. Suspicions for DIC were low with stable hemoglobin, normal platelet count, and normal fibrinogen levels. She was discharged with Aspirin with plan to titrate warfarin outpatient. However, during outpatient testing 14 days post Cefazolin administration, INR rose to 8.4. At this time, she was given 5mg Vitamin K with normalization of INR to 1.0 two days later.

**Discussion**

Prior case studies have identified very similar coagulopathies secondary to Cefazolin use. Proposed mechanisms include alteration of gut flora inhibiting absorption of vitamin K; however, this has been shown to be inconclusive. Recent studies point to inhibition of epoxide reductase and/or gamma-glutamyl-carboxylase through Cefazolin’s thiol group, causing Vitamin K inhibition. Our patient’s normalization of INR after Vitamin K administration furthers this theory. Additionally, studies show malnourished patients are most susceptible to this side effect. Our patient’s BMI was 19 at the time of surgery, in addition to possible malnourishment secondary to Cefazolin’s thiol group, causing Vitamin K inhibition. Our patient’s normalization of INR after Vitamin K administration furthers this theory. Additionally, studies show malnourished patients are most susceptible to this side effect. Our patient’s BMI was 19 at the time of surgery, in addition to possible malnourishment secondary to Cefazolin administration. Due to the popularity of Cefazolin use for surgical infection prophylaxis, more emphasis ought to be placed on monitoring for potential adverse side effects of the drug. INR in high-risk patients, such as those with history of malnourishment, should be carefully monitored after Cefazolin administration.

**References**