Acute Hypotensive Transfusion Reaction: A Case Report

Waleed Shah, M.D., Elia S. Elia, M.D.

Department of Anesthesiology, Thomas Jefferson University Hospital, Philadelphia, Pa

Introduction

Acute hypotensive transfusion reactions are characterized by an abrupt and early onset of hypotension.[1] This is often severe and lacks other signs of symptoms associated with more common causes of hypotension or transfusion reactions. Once the transfusion is stopped, the hypotension rapidly resolves, usually without the need for therapy.[2]

Case Report

The patient was a 68 year old female undergoing a revision of T8-L4 lumbar fusion due to lower extremity back pain and history of spinal stenosis. Medical history is relevant for hypothyroidism, Rheumatoid arthritis, GERD, HTN treated with ACE inhibitor, and hypercholesterolemia.

The patient underwent general anesthesia with an arterial line, neural monitoring and was placed in the prone position. Removal of the pre-existing hardware went as planned and there was an estimated 1000 cc blood loss at this point. The decision to transfuse blood was made based on perioperative blood loss and pre-operative Hgb of 9.9. Within minutes of initiating the transfusion the patient began to have a severe and acute hypotensive episode refractory to IV fluids or phenylephrine boluses (sbp 120-->40). 100 mcg of epinephrine was required to increase pressure. The blood transfusion was immediately stopped and the surgery team was asked to inspect for sources of blood loss. No other change in vital sign was noted, HR, Spo2, temperature, PIP, ETCO2, and EKG were all unchanged from baseline. The patient stabilized and there was no continued need for pressors, the decision was made to continue surgery and transfuse an new unit of blood. Within minutes of transfusing the second unit of blood the patient had another acute episode of hypotension that was treated successfully with 20 mcg of epinephrine. The surgery was completed and the patient was transferred to the Surgical ICU.

A full cardiac and transfusion reaction workup was ordered and was negative. There were no post operative complications noted and the patient was discharged in a timely fashion.

AHTR Recognition

ACE-I use
Acute Hypotension with blood transfusion/apheresis procedure
Immediate resolution of Hypotension after transfusion disconuation
Negatively charged surface (IV tubing, filters)
Lack of other causes of hypotension

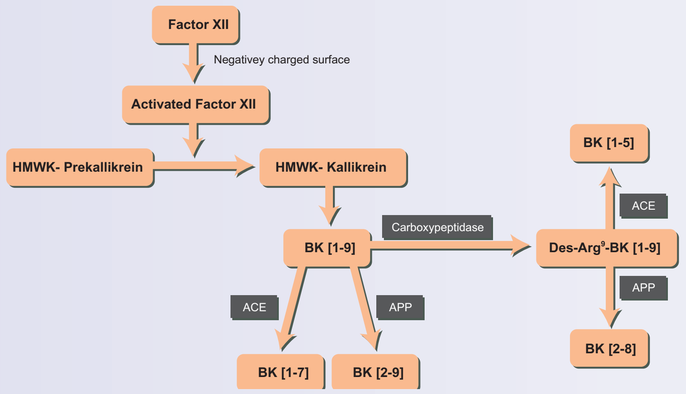
AHTR Management

1. Stop Transfusion
2. Evaluate for other types of hypotension/Transfusion reactions
3. IV fluid Bolus
4. Pressors (if necessary)
5. Discontinue ACE-I

Differential Diagnosis

1. Bacterial Contamination of Blood Products
2. Acute Hemolysis
3. TRALI
4. Anaphylaxis
5. Equipment Errors
6. Drug Errors
7. Bleeding

Acute Hypotensive Transfusion Reaction



Discussion

Life threatening transfusion reactions can result from multiple causes such as bacterial contamination, acute hemolysis, anaphylaxis, WBC or Plasma protein antibodies. A newly reported phenomenon called Acute Hypotensive Transfusion Reaction (AHTR) is defined by its predominant, and often only, sign of acute hypotension. A buildup of Bradykinin (BK) in combination with Angiotensin Converting Enzyme (ACE) inhibitors in patients with impaired BK metabolism lead to AHTR. BK is a powerful vasoactive peptide with a t1/2 of 30 seconds that activates the B2-kinin receptors on vascular endothelium, leading to massive nitric oxide release. It is formed in a cascade reaction with factor XII, prekallikrein, and high-molecular-weight kininogen. The reaction is catalyzed when factor XII contacts a negatively charged surface.[3] Leukoreduction filters and plastic IV tubing can trigger this activation process. Once activated, factor XII, transforms prekallikrein into kallikrein which generates BK from high-molecular-weight kininogen. In normal patients the activity of BK is limited due to rapid degradation by 3 enzymes: Kininase II (ACE), aminopeptidase P (APP), and Kininase-I. ACE is responsible 75% of BK inactivation, APP 20%, and Kininase-I 5%. Carboxypeptidase N transforms BK into the vasoactive metabolite Des-Arg9-BK which activates B1 receptors. This metabolite is in turn inactivated by ACE and APP. In patients with abnormal APP and ACE inhibitor use a large amount of BK and Des-Arg9-Bk can form and lead to profound hypotension. Based on the clinical presentation of isolated hypotension related to blood transfusion and ACE inhibitor use we suspect AHTR in our patient. It is critical to rule out more common sources of intraoperative hypotension such as bleeding, acute MI, other transfusion reactions, medication errors, and equipment errors. AHTR is a diagnosis of exclusion and is ased off clinical presentation and history of ACE inhibitor use.[4] It usually is self limited and resolves quickly once the transfusion is stopped.

Conclusion

AHTR is an uncommon cause of hypotension, caused by a inherent defect in APP and concomitant ACE -inihibitor use. As use of these drugs increase and awareness grows, so may incidence. Currently there are no commercially available tests for AHTR and diagnosis is that of exclusion, based on clinical history, key signs and symptoms and lack of other causes of hypotension. An alternative to ACE-inhibitors should be sought in these patients, especially if they have a need for blood transfusion or pheresis procedures. [1,4]

References

1 Kalra, A. et al. Acute hypotensive transfusion reaction with concomitant use of.angiotensin-converting enzyme inhibitors. American Journal of Theraputics.2012;19:90-94.

2 Papovsky, MA. Transfusion Reactions. Bethesda, MD: AABB Press; 2001.222.

3 Shiba, M et al. Activation of the contact system by filtrate of platelet concentrates. Transfusion. 1997;37:457-462.

4 Doria, C, Elia ES, et al. Acute hypotensive reaction during liver transplantation. Liver Transpl. 2008;14:648-687.

5 Owens HG, et al. Atypical reactions associated with use of ACE-I. Transfusion; 1994;34:891-894.