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Chronic Kidney Disease and Nosocomial Anemia

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Abstract

Anemia, defined as a low haemoglobin (Hgb) level in the blood, is frequent in patients with acute myocardial infarction (AMI) and is an independent and specific predictor of in-hospital or long-term mortality in AMI patients. There is considerable evidence available addressing the significant consequences of anaemia in AMI patients. Nosocomial anaemia is caused mostly by a lack of immunity; patients grow weaker and more susceptible to nosocomial anaemia as a result of chronic renal disease.

Keywords: -*Acute myocardial infarction (AMI), Chronic kidney disease, Nosocomial anemia, Effects, Hemoglobin (Hgb) level*

INTRODUCTION

Few research have been conducted to investigate the impact of hospital-acquired anaemia (HAA), i.e. Bleeding is one of the most prevalent non-cardiac problems in AMI patients. However, in the absence of obvious bleeding, anaemia can develop or worsen during hospitalisation. Furthermore, anaemia is common in people with chronic kidney disease (CKD), as well as in patients who have acute renal damage (AKI). This discovery appears to be explained by the failure of erythropoietin production to respond to decreasing Hgb levels. Yoo et al. (1992) found a strong temporal link between decreased renal function, decreased erythropoietin production, and the development of anaemia.

There is little research on the involvement of renal illness in anaemia in MI patients, particularly in HAA cases. The prognostic impact of HAA in conjunction with renal illness has not before been described (Wu et al., 2001). Because anaemia and renal

disease are independent risk factors for death in patients with AMI, it is critical to understand the crucial role and prognostic implications of renal disease in HAA (Nikolsky et al., 2004).

Anemia is most common in patients hospitalized with acute myocardial infarction (AMI) and is associated with aggrandized mortality rates, higher hospitalization rates, and worse health-related quality of life. However, most prior investigations of anemia in the setting of AMI have evaluated chronic anemia (present at admission) or short-term changes in the hemoglobin during hospitalization. Little research has mainly focused on hospital-acquired anemia (HAA), which develops acutely during AMI admission in those with the normal baseline hemoglobin (Hgb). Given that the etiology of HAA, an acute process, is likely to be different from that of chronic anemia, it is key to understand the risk factors for its development and its prognostic implications (Salisbury et al., 2011).

Chronic anemia is often due to the nutritional deficiencies, chronic inflammation, and renal or bone marrow disorders and can be difficult to manage. In contrast, the HAA may be more likely to result from the in-hospital treatments and processes of care. If associated with adverse outcomes, HAA may be mainly preventable and could represent an actionable target for hospital-based quality improvement efforts (Smoller et al., 1986). Potential benefits of the preventing HAA may include reducing patients' exposure to the risks from the acute anemia treatments such as blood transfusion, improving clinical outcomes, and reducing costs. But before resources are directed to HAA prevention, it is necessary to better understand the incidence and predictors of HAA and its collaborations with clinically relevant outcomes.

To address these gaps in the knowledge, we sought to describe the incidence and predictors of HAA, the variation in the HAA prevalence between hospitals, and its relationship with subsequent mortality and the health status outcomes in the Translational Research Investigating Underlying disparities in acute Myocardial infarction Patients' Health Status study (TRIUMPH), a prospective 24-center observational registry of the AMI treatment and outcomes (van der Bom et al., 2015). TRIUMPH garnered an ideal opportunity to address these important questions, as it collected detailed patient information on in-hospital hemoglobin,

hospital-based treatments, complications and processes of care, as well as the serial assessments of patient outcomes after discharge (Thavendiranathan et al., 2005).

SPECIFIC ETIOLOGIES OF NOSOCOMIAL-ACQUIRED ANEMIA

Surgical Loss Etiology

Surgery-linked blood loss varies greatly according to the type of surgery, patient comorbidities, and technical factors. Surgical advances of the technology and technique, encompassing vessel-sealing energy devices and minimally invasive surgical techniques, have stemmed losses in some frequently performed operations. Many of the surgeries, however, still confer high amounts of blood loss. Highly invasive operations, such as those involving mainly the liver, pancreas, or major bony structures, frequently require intraoperative or postoperative transfusions to maintain adequate hemoglobinlevels (Sabatine et al., 2005).

Incidence and magnitude

Surgery is quiet frequent cause of a medical blood loss of 20% or more of a patient's total blood volume. Such extreme losses can certainly pave not only to anemia but also to other morbidities and even death. In fact, more than half of the 21 million units of blood and blood products transfused annually are the perioperative in nature. Patients undergoing the high-risk procedures-such as cardiac, hepatic, and certain orthopedic surgeries-are especially prone to hemorrhage. Trauma patients are also at the extreme risk for bleeding from injuries; hemorrhage is the leading cause of death in trauma patients. Surgical hemostasis is therefore paramount during all the types of surgery.

Prevention

Some surgical blood losses cannot be mainly predicted or prevented (Al Falluji et al., 2002). The diligent surgical technique concentrating on meticulous hemostasis and the use of quiet advanced modalities, such as laparoscopy, can aid in decreasing the intraoperative blood losses. Conscientious anesthesia care with optimal fluid and volume management also plays a much strong role (Salisbury et aal.,2010). Careful patient selection is equally important, recognizing the patient factors that may lead to coagulopathy from comorbidities or technical challenges due to the body habitus. Screening for abnormal hemostasis and coagulopathies preoperatively may spare the vulnerable patient from hemorrhage and the need for sporadic correction of missing factors intraoperatively. Prudent discontinuation

of the anticoagulants preoperatively should be performed whenever possible. In patients undergoing the coronary artery bypass grafts, cessation of aspirin has been linked with the lesser requirement for transfusion of blood products after surgery, and continued utilization of tirofiban (Aggrastat [Medicure Pharma]) and clopidogrel preoperatively has been connected with higher transfusion requirements(Salisbury et al.,2011).

Correction of the surgically significant coagulopathy also can be promoted pharmaceutically. Aprotinin (Bayer), the aminocaproic (Amicar [Xanodyne]), and tranexamic acid (Lystenda [Ferring]), and the antifibrinolytics, have been studied for their perceived ability to achieve decreased surgical blood loss and, therefore, decreased transfusion requirements. Recombinant activated factor VIIa and the desmopress in also have been studied for this purpose with the variable results(Tahnk-Johnson et al.,1983).

A protinin, a plasmin inhibitor, has been studied and evaluated especially to this end in randomized controlled trials of the cardiac surgery patients. Results consistently have indicated the lower operative blood loss, ranging from 50 to 1350 mL, and a reduction in the need for the blood product transfusion, ranging from 1.5 times to 3 times reduction, even in patient populations predisposed to the bleeding(McGonigle et al.,1984).

Antifibrinolytics are less well studied but may hold some promise. In the study of 210 cardiac surgery patients, a nearly 70% reduction in the RBC transfusion was noted when tranexamic acid was used, and less than half as many as patients required any transfusion compared with the placebo. Researchers found that the use of the tranexamic acid or aprotinin saved approximately 1 unit of the blood for each surgery performed.

Treatment

While patient selection and the surgical technique are critical components of preventing surgical losses, the number of techniques can be employed to combat hemorrhage. Mechanical hemorrhage control remains the gold standard, but, as in the prevention, pharmaceutical intervention has the role in ceasing hemorrhage. Recombinant activated factor VII has been studied and assessed with success in severe blunt trauma. In a controlled trial of 144 patients, transfusion requirements were reduced by an average of 2.6 times with a more substantial reduction in the need for the massive

transfusion of more than 20 units (15% versus 33% of patients)(Cotes,1982).

There are a host of the topical hemostatic agents also available commercially for use during operative procedures (Lipkin et al, 1989). In general, these agents specifically promote clot formation in the region of injury. Application techniques differ greatly, and a comprehensive understanding of application techniques is quite imperative to being an attentive surgeon or proceduralist (Nielsen et al., 1989).

Special populations in surgery

Surgical losses can be further mitigated with the specific interventions. Much can be learned from centers that offer "bloodless" the surgical techniques, aimed at upholding the religious beliefs of groups like Jehovah's Witnesses (Nielsen et al., 1990). There are various agents causing nosocomial infection (Sreeremya, 2019).A recent research focused on the community of Jehovah's Witnesses and reviewed several strategies to avoid the transfusion needs in operations with the historically significant blood loss (Erslev et al., 1997).

CONCLUSION

There are several nosocomial agents that cause illness, the majority of which are bacterial infections. When a person's immune system is weak and they have chronic renal disease, they are more likely to be afflicted by nosocomial anaemia.

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