Coagulopathy of Trauma

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KEYWORDS

• Trauma • Hemorrhage • Coagulation • Coagulopathy
• Trauma-induced coagulopathy • Targeted resuscitation • Bleeding

KEY POINTS

- Trauma-induced coagulopathy (TIC) is an endogenous hypocoagulable state distinct from iatrogenic causes.
- Activation of protein C pathway is a key mechanistic mediator of traumatic coagulopathy via downstream effects, including thrombin diversion, deactivation of coagulation factors, and de-repression of fibrinolysis.
- Standard coagulation tests and functional viscoelastic assays are commonly used in the diagnosis and management of TIC.
- Balanced resuscitation is the mainstay of coagulopathy treatment, but precise ratios for empiric resuscitation and optimal monitoring protocols for transfusion practice remain unknown.
- Patients with traumatic coagulopathy have worse outcomes, including increased rates of transfusion, infection, thromboembolism, acute lung injury, multiorgan failure, and death.

INTRODUCTION

Bleeding remains the leading cause of preventable death after injury.[1] Contributing to this problem, coagulopathy develops in approximately one-third of all injured patients,[2–4] resulting in worsened outcomes including higher transfusion requirements; increased multiorgan system failure, increased hospital, intensive care, and ventilator days; and increased mortality.[2,3,5,6]

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History of coagulopathy in trauma

Although coagulopathy was known to occur after injury, until recently coagulation was not viewed as a critical driver of postinjury physiology. Instead, injured patients were thought to be coagulopathic owing only to the iatrogenic secondary effects of hemodilution, hypothermia, and acidosis.[7,8] In 2003, 2 independent investigators described admission perturbations of prothrombin time (PT) and partial thromboplastin time (PTT) in newly injured patients before significant fluid administration.[2,3] This phenomenon, which correlated with increasing injury severity and mortality, became known as "acute traumatic coagulopathy" (now "trauma-induced coagulopathy" [TIC]) and effectively changed the paradigm of modern trauma care.[2,3,9] The study of coagulation and inflammation derangements after injury now constitutes one of the most active areas of ongoing trauma research.

This review addresses the current evidence regarding the diagnosis, mechanisms, and management of TIC, highlighting areas of ongoing debate and controversy. Although TIC is emphasized, it is equally important to recognize that coagulopathy after trauma is often caused or compounded by additional contributors of disordered coagulation including hypothermia, acidosis, dilution with large volume of intravenous fluid, or unbalanced blood product, all of which are termed iatrogenic coagulopathy. Management of the injured coagulopathic patient must therefore include a high suspicion for and treatment of multiple different potential etiologies of dysfunctional clotting.

PATIENT EVALUATION AND OVERVIEW

Mechanism and Pathophysiology

Multiple distinct but highly integrated pathways have been implicated as mediators of TIC (Fig. 1). Delineating the exact pathophysiology and interplay between disordered coagulation and inflammation mechanisms remains the subject of ongoing research. Herein, we describe the most important known contributors to TIC.

Activated protein C and fibrinolysis

TIC is an endogenous hypocoagulable state that occurs in the setting of tissue hypoperfusion (base deficit) and is primarily mediated by activation of protein C (Fig. 2).[10]

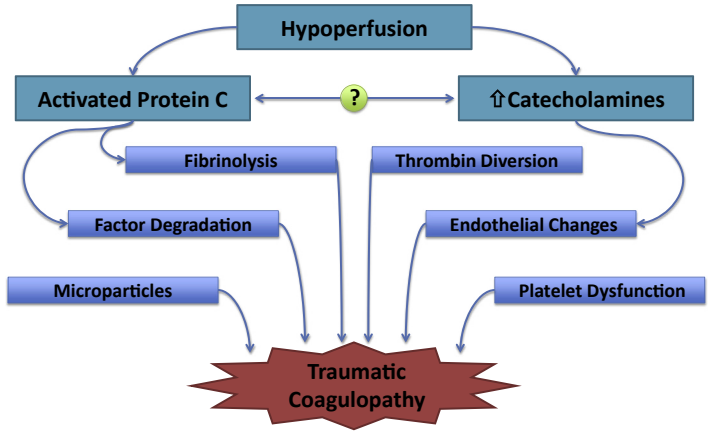

Fig. 1. Pathophysiology of traumatic coagulopathy. Multiple distinct but highly integrated pathways have been implicated as mediators of trauma-induced coagulopathy. Delineation and integration of these pathways remains an area of ongoing research.