Noncompressible Torso Hemorrhage

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KEYWORDS

• Noncompressible torso hemorrhage • Damage control surgery
• Damage control resuscitation • Endovascular hemorrhage control • Trauma surgery
• Military surgery

KEY POINTS

- Noncompressible torso hemorrhage (NCTH) is a leading cause of potentially preventable trauma mortality.
- Open surgery remains the mainstay of management, within a damage control resuscitation paradigm.
- Endovascular hemorrhage control techniques are also evolving and may compliment open surgery for NCTH.

INTRODUCTION

Hemorrhage is responsible for 40% of civilian trauma-related deaths and more than 90% of military deaths from potentially survivable injuries.[1,2] In trauma patients, most early deaths are caused by hemorrhage, and occur at a median of 2.6 hours after admission.[3]

Hemorrhage is a pathologic process that can have profound implications for physiologic homeostasis.[4] The clinical consequence is dependent on both the severity and duration of the bleeding, as well as the body's physiologic reserve. The natural history of uncontrolled exsanguination is of cardiovascular collapse, failure of tissue oxygenation, ischemia, and organ dysfunction. When this process is rapid, cerebral and myocardial ischemia will result in coma, cardiac arrest, and death.

Traumatic injury is a major cause of hemorrhage, as the physical disruption to vasculature, either within organ parenchyma or discreet arterial or venous structures, provides for a hemorrhagic focus.[5] A first-line therapeutic maneuver is to compress the site of bleeding, which if this pressure exceeds the pressure driving the bleeding, hemostasis will generally result. Although a more definitive hemostatic maneuver is likely still

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required, the cycle of exsanguination and ischemic organ dysfunction will have been halted.

This is referred to as *compressible* hemorrhage and is found in accessible sites, such as the extremities, where either a pressure dressing or tourniquet can be applied. Following from the military lessons of the past decade, mortality from compressible hemorrhage is theoretically preventable with rapid hemostasis and resuscitation.[6] However, where a hemorrhagic focus is inaccessible and therefore *noncompressible*, it is more difficult to break the cycle of bleeding and organ dysfunction.[7]

Hemorrhage from within the torso exemplifies the problem of *noncompressible* hemorrhage, where in general, formal surgical intervention is required for hemostasis. As hemorrhage is a time-dependent pathology, this can be difficult to achieve before significant and/or irreversible ischemic organ damage has occurred.

Noncompressible torso hemorrhage (NCTH) can be highly lethal and has seen significant changes in management in the past decade. This review aims to present the state of current practice and future research efforts.

DEFINITION OF NONCOMPRESSIBLE TORSO HEMORRHAGE

Despite recognition of the mortality burden associated with NCTH, there has been little done to define and codify this injury pattern so as to guide identification and management. Such efforts have been the cornerstone of other similarly important, but unrelated pathologies, such as sepsis and septic shock, in which clear definitions have proved critical to recognition and treatment.[8] A standardized NCTH definition enables research efforts to be harnessed and therapeutic approaches specifically targeted.[5]

Several facets need to be considered when defining NCTH: anatomic injury pattern, physiology, and the clinical need for intervention. All of these factors play an important role in decision making and should be reflected in any formal description.[9]

Anatomic injury pattern is an important basic consideration. Trauma patients are frequently polytraumatized, making the prioritization of critical treatments challenging. For example, a patient presenting with signs of hypovolemic shock and a trivial torso injury should prompt a meticulous search for alternate source of hemorrhage. Similarly, anatomic disruption must be coupled with a marker of hemorrhagic shock, providing evidence of failing homeostasis and the sequelae of reduced perfusion. This is critical, as the findings of "hemodynamic instability" significantly influence the need for aggressive hemostatic intervention.

However, whereas severe hemodynamic instability is easily recognized, many patients occupy a middle ground, in which they have sufficient reserve to compensate for a significant insult. Should this physiologic reserve be exhausted, such patients can decompensate in a rapid and life-threatening manner.[4] Clinician judgment is therefore very important and on occasion, hemostatic intervention may be deemed necessary despite relatively normal physiology. Furthermore, depending on data collection mechanisms, physiologic data can be poorly collected. Consequently, the addition of a procedural marker for the need for immediate hemorrhage control serves as another marker of NCTH.[9]

Morrison and Rasmussen[5] sought to use this scheme to define NCTH in trauma as the presence of hemorrhage due to vascular disruption from 1 or more of 4 anatomic categories (**Box 1**, **Table 1**). They included 4 anatomic domains of high-grade injury, associated with bleeding, with preexisting abbreviated injury scale (AIS) codes: complex pelvic fracture, major vascular, solid organ, and pulmonary injury. This was coupled to physiologic indicators of hemorrhagic shock (systolic blood pressure