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INFECTION AND TRAUMA (M SARTELLI, SECTION EDITOR)

Risk Factors for Infections in Trauma Patients

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Abstract
Purpose of Review This review aims to investigate the pattern of factors, which contribute to trigger and/or aggravate nosocomial infections in trauma patients in order to better prevent and treat such complications in those frail patients.
Recent Findings Trauma-related deaths have a tri-modal distribution, the third spike is commonly defined as the "late deaths" occurring after days or weeks and is due to sepsis and multiple organ failure. Hospitalized trauma patients with at least a single infection have a higher risk of mortality. Infections lead to a worse functional status and, in elderly patients, lower scores in social function, vitality, emotional, and general health. In those patients, the need for health care services and costs grows. Infection prevention in trauma patients represents a fundamental factor in improving outcomes. Every effort in preventing and treating them should be done.
Summary The reduction of unnecessary invasive devices use and the close monitoring of the patient's vital parameters are the cornerstones of prevention and treatment in order to promptly treat the infection before the progression toward systemic symptoms and sepsis. The knowledge of both the risk factors and the potential pathogens may help physicians in preventing both the nosocomial post-traumatic infections and the antibiotic overuse, which induces harmful drug resistances by selecting multidrug-resistant micro-organisms.

Keywords Trauma · Nosocomial infections · Surgical infections · Emergency medicine

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Introduction

It is well established that deaths secondary to trauma have a tri-modal distribution. The first spike occurs immediately after trauma because of non-salvageable injuries (i.e., heart or great vessel injuries). Subsequently, "early deaths" occur over the first 6 h after trauma and are due to evolving conditions (i.e., hemorrhagic injuries or expanding intracranial masses). Finally, "late deaths" occur after days or weeks and are due to sepsis and multiple organ failure (MOF) [1]. Because tissue integrity is disrupted and the immune defense system is low, trauma patients are more likely to develop infections, which may impact on morbidity and mortality. Czaja et al. carried out a multicenter study in the USA collecting data about a total of 4732 trauma patients. The study showed that hospitalized trauma patients with at least a single infection had a higher risk of mortality within 1 year after the traumatic event [2]. Firstly, infections led to a worse functional status and, in elderly patients, lower scores in social function, vitality, emotional, and general health. Secondly, in those patients, the need for health care services and costs grows. In fact, statistics state that a higher probability of repeat hospitalizations and home health services, besides it was estimated approximately three more hours of care from family per month. Finally, those patients were less likely to return to work [3].

Risk Factors

Risk factors for nosocomial post-traumatic infections (NPIs) are multiple and related both to the patient features (age, sex, and comorbidities) and the trauma (anatomical site of the trauma, required procedures, hospital services, and urgency of admission). The injury severity appears to be the predominant risk factor NPIs [4]. Therefore, a number of published studies pointed out significant relationships between the most popular scoring systems adopted worldwide (ISS, AIS, RTS, NISS, TRISS) and the incidence of NPIs. Jamulitrat et al. investigated about a potential relationship between a scoring system for trauma patients and the incidence of NPIs. After analysis and a number of scoring systems, such as ISS, NISS, RTS, and TRISS, they found that the only significant relationship was between the NISS and the incidence of NPIs [5]. It is well documented that a poor functional status at the baseline in a trauma patient may easily result in a longer hospital stay and increase the risk of developing NPIs. From this perspective, an advanced age and pre-existing comorbidities may exacerbate the clinical conditions [6]. Physiological change in elderly patients entirely affects the body and causes it to become more prone to opportunistic infections [7]. Bochicchio et al. demonstrated that elderly had a higher probability to develop a NPI over a significantly longer hospital stay compared to the younger counterparts [8]. Serrano et al. found that obesity generates an underlying inflammatory condition, which affects the immune defense system and causes it to become insufficient to adequately react to infections. In the study, pulmonary and wound infections were substantially more frequent among obese patients and obesity resulted in an independent risk factor for NPIs [9]. Some randomized prospective data suggest that early hyper-glycemia (glucose ≥200 mg/dL) is related to a higher risk of infection and mortality in trauma patients regardless of injury features [10].

Gannon et al. evaluated the risk factor in developing nosocomial pneumonia in trauma patients and highlighted that a higher incidence of pneumonia occurred in males and patients with history of cardiac disease, high ISS or RTS, and history of cancer. The risk of mortality was higher in patients with nosocomial pneumonia, however no gender-specific difference in mortality among pneumonia patients [11]. Edelman et al. added further independent risk factors for NPIs such as gastric, pancreatic, colonic injuries, and emergency transfusion [12]. A multicenter study, which looked at the anatomical site of the trauma in 24,711 patients, showed that severe liver injuries led to a poor prognosis when compared to severe abdominal trauma patients and control group [13]. As patients with a liver injury require a massive volume resuscitation, they are likely to develop severe hemorrhage, infections, sepsis, and MOF. Additionally, a report from an American group investigated the association of NPIs to a significant blood loss that leads to a generalized hypoperfusion secondary to the traumatic event. This study found that both the ISS and the hypoperfusion, if not corrected within 12 h, were independently predictive factors of NPIs [14]. Giamberardino et al. found that trauma patients were more likely to develop a nosocomial infection if they (1) presented injuries in multiple sites rather than in the same body area, (2) required mechanical ventilation for more than 3 days, and (3) required more than one surgical procedure and more than two invasive devices. Mortality within the first 5 days had high association with NPIs [15].

Infections in Trauma Site by Site

Pneumonia

Hospital-acquired pneumonia (HAP) is one of the commonest NPIs and it is more likely in patients with injuries to the head, thorax, and abdomen due to changes in the respiratory mechanism [16]. Andermahr et al. found that independent factors, which may increase the incidence of pneumonia in polytrauma patients, were the advanced age, male gender, traumatic brain and thorax trauma, and ISS [17]. In elderly, the risk of developing a pneumonia is significantly higher than that in the younger counterparts because of pre-existing comorbidities such as the chronic obstructive pulmonary disease (COPD) which causes the lung tissues to become frail and less flexible [18].

Although several studies attempted to clarify the predisposing factors of HAP, the predominant risk factor appears to be a prolonged mechanical ventilation and the positive end-expiratory pressure [19]. Severely ill trauma patients are likely to be intubated and admitted in intensive care units (ICUs). Ventilator-associated pneumonia (VAP) is a type of pneumonia, which occurs after 48 h or longer of mechanical ventilation (i.e., endotracheal tube or tracheostomy). It may be categorized by the timing: the "early-onset VAP" develops within the first 4 days of hospitalization, whereas the "late-onset VAP" five or more days afterwards. Sadly, the late-onset VAPs are more commonly associated with multidrug-resistant (MDR) organisms [20].

Despite the well-established association between mortality and VAP, which ranges between 9 and 17%, there was no clear evidence that the risk of increased mortality because of VAP in patients with acute respiratory distress syndrome or after a traumatic event [21•]. In order to determine its impact on trauma patient outcomes, Magnotti et al. carried out a logic regression analysis and found that transfusions, advanced age, and VAP represent independent predictors of mortality [22].

Aspiration pneumonia is another frequent type of NPIs and physicians have to be able to recognize and treat. Severely ill trauma patients with head injury or with a low GCS represent a high-risk population for

macroaspiration. Miller et al. showed that 11.7% of the severely ill trauma patients enrolled developed a clinical event of macroaspiration. Despite the incidence of pneumonia was similar, those who had a macroaspiration event required a longer ICU stay with a prolonged mechanical ventilation [23].

The commonest pathogens, which cause approximately 80% of HAPs, were *Staphylococcus aureus* (28%), *Pseudomonas aeruginosa* (21.8%), *Klebsiella* species (9.8%), *Escherichia coli* (6.9%), *Acinetobacter* species (6.8%), and *Enterobacter* species (6.3%). There is a minor prevalence of *Serratia* species, *Stenotrophomonas maltophilia*, and community-acquired pathogens (pneumococci and *Haemophilus influenza*) [24]. Statistics state that whereas *S. aureus* is the commonest pathogen in HAPs, *P. aeruginosa* and *Acinetobacter baumannii* are significantly more frequent in VAPs. Lastly, the incidence of gram-negative species (especially *Haemophilus* species) is more prevalent in VAPs [25].

The current recommendations for empiric therapy for early-onset VAPs with no risk factors for MDR pathogens are one of the following antibiotics:

- Ceftriaxone
- Fluoroquinolones
- Ampicillin-sulbactam
- Ertapenem

Risk factors for HAP due to MDR pathogens are as follows: (1) antibiotics initiated within the preceding 90 days, (2) onset of pneumonia occurred after 4 days of hospitalization, (3) known MDR pathogens that are circulating in the community and/or hospital, and (4) active immunosuppressive disease or immunosuppressive therapy. Methicillin-resistant *S. aureus* (MRSA), MDR *P. aeruginosa* (carbapenems, fluoro quinolones, and antipseudomonal penicillins and cephalosporinases resistant), extended-spectrum beta-lactamase (ESBL)-producing *Enterobacter*, *E. coli*, and *Klebsiella pneumoniae*, *Acinetobacter* species, *Stenotrophomonas* (*Pseudomonas*) *maltophilia*, and *Burkholderia cepacia* are the commonest MDR pathogens entailed [26].

For early-onset VAPs with one or more of the aforementioned risk factors for MDR pathogens or for late-onset VAPs, the initial antibiotic treatment consist of one of the following:

- Antipseudomonal cephalosporins (e.g., cefepime, ceftazidime)
- Antipseudomonal carbapenems (imipenem or meropenem)
- Beta-lactam/beta-lactamase inhibitors (piperacillin-tazobactam) with an antipseudomonal fluoroquinolone (ciprofloxacin) or aminoglycoside plus linezolid or vancomycin (if risk factors for methicillin-resistant *S. aureus* are present)

If *Legionella pneumophyla* is suspected, the antibiotic treatment should include a macrolide or fluoroquinolone rather than an aminoglycoside [26].

Urinary Tract Infection

Urinary tract infections (UTIs) are particularly frequent during the hospitalization, especially in trauma patients, and is often secondary to the bladder catheter insertion. Sadly, UTIs may cause the hospital stay to be prolonged and the risk of major complications and mortality to be increased. Monaghan et al. found advanced age, gender, high ISS, and indwelling urinary catheter use to be predictors of UTIs. Interestingly, the development of a UTI predicted the risk of in-hospital mortality as a patient's age increased [27].

Bochicchio et al. showed that women have a higher risk of developing a community-acquired infection than males (5 vs. 1%, respectively) and NPIs (23 vs. 15%, respectively), whereas obesity was predictive of prolonged Foley catheter maintenance, and thus, UTIs. Additionally, age is a substantial risk factor of developing a community-acquired infection which leads to a higher mortality rate compared to the younger counterparts (39 vs. 15%, respectively) [28].

In abdominal trauma patients, abdominal pressure measurements may be needed to rule out a potential acute compartment syndrome. Duane et al. showed that bladder pressure measurements with open technique (the bladder catheter requires to be disconnected from the bag at each measurement) were independent risk factors for UTIs regardless of age and ISS [29]. Conversely, with a closed system, where the saline is injected through a two-way valved sideport, the bladder pressure measurement is safe and does not represent a risk factor for UTIs [30].

E. coli is the commonest pathogen causing UTIs (nearly 80%). *Enterobacteriaceae* (*Klebsiella* species, *Serratia* species, *Citrobacter* species), *Enterobacter* species, non-fermenters (*P. aeruginosa*, gram-positive cocci, including coagulase-negative staphylococci and *Enterococcus* species), and fungi (*Candida*) are also found. In patients with indwelling bladder catheter, UTIs are usually polymicrobial [31].

Peterson et al. compared empirical therapy with quinolone antibiotics (levofloxacin 750 mg intravenously or orally once daily for 5 days vs. ciprofloxacin 400 mg intravenously and/or ciprofloxacin 500 mg orally twice daily for 10 days) to treat acute pyelonephritis or complicated UTIs. Clinical success rates and microbiologic eradication were both similar. Microbiologic eradication in catheterized patients was lower. Levofloxacin was the antibiotic, which better achieved the eradication among this group of patients [32].

According to those data, the Infectious Diseases Society of America recommends 7 days of antimicrobial treatment for catheterized patients with UTI who have an immediate resolution of symptoms, and 10–14 days of treatment for those

with a delayed response. A short regime (5 days) on levofloxacin may be considered in catheterized patients with a mild UTI [33].

Bloodstream Infections

Bloodstream infections (BSIs) in trauma patients are mainly due to the prolonged use of central venous catheter. Both during the prehospital phase and in the emergency department, triaging is a key step for the therapeutic pathway in a trauma patient. Acidosis correction and fluid restoration is often needed during these first phases. In order to guarantee an efficient fluid resuscitation and monitor the central venous pressure, a central venous line is required. Nosocomial BSIs in severely ill trauma patients are virtually deadly infections, which may be primary or secondary to other infections [34]. Nosocomial BSI is one of the most frequent infections among the NPIs and, in trauma patients, its prevalence is even higher than that among surgical patients in ICUs [3].

The most common pathogens, which cause nosocomial BSIs are gram-positive cocci (nearly 69%), followed by gram-negative bacilli (16.3%). Fungi made up 10.9% and multiple micro-organisms accounted for 3.6% [35]. Niven et al. documented that nosocomial BSIs in critically ill trauma patients determined a substantially longer hospital stay and an increase of hospital expenditure [36].

Infections are more likely to occur in trauma patients and may easily determine their outcomes. There is a number of risk factors, which may increase the probability of infection in trauma patients. Firstly, trauma patients are often frail and feeble patients who came from a significant "first hit" (trauma) which requires a good physiological reserve. The rational is due to the loss of integrity of physiological barriers secondary both to the traumatic event and invasive devices (such as central venous catheter, blood catheter, and thoracic drain) and to the immunosuppression, which may occur as reaction to the trauma [37•]. They are both risk factors of developing an infection. Additionally, those patients are usually hospitalized for longer than patients who are admitted for elective procedures and are usually due to massive transfusions. El-Masri et al. found that trauma patients, who received ten or more units of blood, were five times more likely to develop a nosocomial BSI [35]. It may be postulated that massive transfusions are usually secondary to great loss of blood and thus, immunoglobulins. Lastly, the trauma induces a protein catabolism. This change in association with an important total parental nutrition may determine hyperglycemia, which is a predisposing factor for infections. Therefore, a continuous glycemic monitoring with a consequent appropriate insulin therapy is advised, so too is calculating carefully the calories for day in order not to overfeed the patient [38].

Surgical Site Infections

Surgical site infections (SSIs) are defined as infections affecting either the incision or the deep tissue and may occur up to 30 days after surgery or up to 1 year in patients who receive implants. SSI is the most frequently reported surgical complication and accounts for 16% of all the nosocomial infections [39].

Surgery is usually needed in trauma patients and may consist of a cluster of surgical techniques performed by orthopedics, general surgeons, plastic surgeons, and urologists. Occasionally, more than two surgical specialties may cowork during the same operation to optimize the timing and the patient outcome. Polytrauma patients have multiple anatomical sites involved in the trauma and multiple surgical wounds are necessary. Moreover, different surgical specialties require different approaches according to the lesion site and grade and to the surgical team expertise [40••]. Lastly, after surgery, trauma patients often require to be closely monitored in ICUs where MDR pathogens are highly selected [41].

Morales et al. found that the presence of hemodynamic shock, the number of affected organs, unconsciousness, Acute Physiology and Chronic Health Evaluation (APACHE II), ISS or TRISS, prolonged mechanical ventilation, use of prophylactic antibiotics, central venous catheters, spinal cord injury, multiple transfusions, several surgical procedures, systemic inflammatory response syndrome are all risk factors for SSIs in trauma patients [42]. Additionally, Seamon et al. showed that the acidosis status and hypothermia intraoperatively (less than 35 °C) induced an increased SSI risk [43]. Richards et al. pointed out that hyperglycemia as an independent risk factor for SSI in no diabetic patients [44].

Interestingly, Herruzo-Cabrera et al. highlighted further risk factors for SSIs such as contaminated surgery, inadequate chemoprophylaxis, and a pre-surgical stay >4 days. According to these study results, pre-surgical stay and perisurgical chemoprophylaxis optimization may significantly decrease SSIs [45].

Pathogens causing SSIs depend on the patient and surgery features. *S. aureus*, coagulase-negative staphylococci, *Enterococcus* spp., and *E. coli* are the more commonly micro-organisms isolated. Though, as previously mentioned, a number of patient-related and procedure-related factors, which influence the SSI risk, were documented. A systematic attention to multiple risk factors as to optimize prevention should be obtained in each patient. Obviously, because of such a great number of risk factors, prevention should be tailored in order to decrease bacterial contamination and improve the patient's defenses. The Centers for Disease Control and Prevention guidelines for the prevention of SSIs yield that a good patient preparation, an aseptic practice, and the attention to surgical technique are crucial [46].

Intra-Abdominal Infections

Intra-abdominal infections are frequent in abdominal penetrating trauma because the abdominal wall is disrupted and occasionally the hollow organs (e.g., stomach and intestine) are perforated. The first finding may be clinical signs of peritonitis or a clear evisceration, which requires emergency surgery. Conversely, in blunt abdominal trauma, the injury of hollow organs is unusual, though these trauma may determine the outbreak of those organs. Additionally, the splanchnic circulation may be damaged because of tearing in the context of the mesentery and a consequent ischemic insult to abdominal organs may develop over the next 24–48 h [47]. In trauma patients with a positive ECO-fast for abdominal free fluid, either a damage of the splanchnic circulation or a splenic lesion should be suspected and investigated with an emergency laparotomy.

As the abdominal hollow organ perforation secondary to blunt trauma is uncommon (approximately 0.3%) and usually has a late onset and vague symptoms, the diagnosis may be challenging. Watts et al. found that the most frequently involved hollow organs were the small bowel followed by the colon, the duodenum, and finally, the stomach [48]. Williams et al. showed the difficulty to determine the anatomical site of the perforation despite the use of multiple imaging modalities. In the vast majority of those patients, the anatomical site of the perforation is highlighted once the patient undergoes the emergency laparotomy [49].

A delayed diagnosis may be crucial for the patient outcome. Interestingly, Malinoski et al. documented that a delay of surgery >5 h from the admission is a preponderant prognostic factor and deaths were usually associated to abdominal-related sepsis. Further prognostic factors are the advanced age, the Abdominal Abbreviated Injury Score, and the presence of a significant extra-abdominal injury [50].

As intra-abdominal infections are associated with a high morbidity and mortality rate, a prompt recognition of risk factors and a proper evaluation of the patient and trauma should be performed to prevent as much as possible any infection source. Once the infection source is identified, it should be treated before the patient might manifest systemic symptoms. Those patients may require a surgical treatment in addition to antibiotic therapy. A close monitoring of the patient's vital parameters and infection evolution is essential to prevent a systemic instability. At this stage, the patient still has physiological reverse to undergo a surgical procedure in order to eradicate the infection source whether it was not possible to remove it earlier [51].

The treatment choice, which may be either surgical or conservative, depends on the anatomical infection source, the peritoneal inflammation degree, the septic response, and the physiological reserve. Antimicrobial therapy has a key role, especially in severely ill trauma patients because it should be offered early on, when the patient manifests the first infection symptoms. However, the micro-organism causing the infection is rarely isolated at this stage and the antibiotic therapy to deliver is empirical. The decision of antimicrobial therapy depends on three factors:

- Presumed pathogens involved and risk factors for major resistance patterns
- Clinical patient's severity
- Presumed/identified source of infection.

More commonly, the pathogens involved in community-acquired intra-abdominal infections are *Enterobacteriaceae*, *Streptococcus* spp., and anaerobes (especially *Bacteroides fragilis*). Conversely, nosocomial intra-abdominal infections are due to a broader spectrum of micro-organisms such as *Enterobacteriaceae*, *Streptococcus* spp., and anaerobes, but also *Enterococcus* spp. and *Candida* spp. [52•].

Conclusion

Infections in trauma patients are more common than in surgical patients because after the trauma (first hit), the natural barriers (e.g., skin muscles and bones) are disrupted and the physiological reserve has decreased. Additionally, invasive devices (e.g., central venous catheter, thorax drains) represent a vehicle for nosocomial micro-organisms. Lastly, those patients are more likely to require an intermediate period in ICU and longer hospitalizations than surgical patients. Considering all these factors, NPIs still represent a life-threating condition for trauma patients whether they are not promptly recognized and treated. As to reduce the infection rate, physicians should attempt to achieve two goals: (1) decrease the use of invasive devices when unnecessary and (2) monitoring closely the patient parameters in order to promptly treat the infection before systemic symptoms and sepsis. The knowledge of the risk factors and the potential pathogens may help physicians to prevent NPIs and decrease the antibiotic overuse, which induces harmful drug resistances and selects MDR micro-organisms.

Compliance with Ethical Standards

Conflict of Interest The authors declare no conflicts of interest relevant to this manuscript.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1. Chiara O, Cimbanassi S, Pitidis A, Vesconi S. Preventable trauma deaths: from panel review to population based-studies. World J Emerg Surg. 2006;1:12.
2. Czaja AS, Rivara FP, Wang J, Koepsell T, Nathens AB, Jurkovich GJ, et al. Late outcomes of trauma patients with infections during index hospitalization. J Trauma. 2009;67(4):805–14.
3. Wallace WC, Cinat M, Gornick WB, Lekawa ME, Wilson SE. Nosocomial infections in the surgical intensive care unit: a difference between trauma and surgical patients. Am Surg. 1999;65(10):987–90.
4. Pories SE, Gamelli RL, Mead PB, Goodwin G, Harris F, Vacek P. The epidemiologic features of nosocomial infections in patients with trauma. Arch Surg. 1991;126(1):97–9.
5. Jamulitrat S, Narong MN, Thongpiyapoom S. Trauma severity scoring systems as predictors of nosocomial infection. Infect Control Hosp Epidemiol. 2002;23(5):268–73.
6. Hashmi A, Ibrahim-Zada I, Rhee P, Aziz H, Fain MJ, Friese RS, et al. Predictors of mortality in geriatric trauma patients: a systematic review and meta-analysis. J Trauma Acute Care Surg. 2014;76(3):894–901.
7. Maxwell CA, Mion LC, Mukherjee K, Dietrich MS, Minnick A, May A, et al. Preinjury physical frailty and cognitive impairment among geriatric trauma patients determine postinjury functional recovery and survival. J Trauma Acute Care Surg. 2016;80(2):195–203.
8. Bochicchio GV, Joshi M, Knorr KM, Scalea TM. Impact of nosocomial infections in trauma: does age make a difference? J Trauma. 2001;50(4):612–7. discussion 7-9
9. Serrano PE, Khuder SA, Fath JJ. Obesity as a risk factor for nosocomial infections in trauma patients. J Am Coll Surg. 2010;211(1):61–7.
10. Laird AM, Miller PR, Kilgo PD, Meredith JW, Chang MC. Relationship of early hyperglycemia to mortality in trauma patients. J Trauma. 2004;56(5):1058–62.
11. Gannon CJ, Pasquale M, Tracy JK, McCarter RJ, Napolitano LM. Male gender is associated with increased risk for postinjury pneumonia. Shock. 2004;21(5):410–4.
12. Edelman DA, Donoghue L, White MT, Tyburski JG, Wilson RF. Gastric injury increases infections in trauma patients. Am Surg. 2008;74(11):1057–61.
13. Lendemans S, Heuer M, Nast-Kolb D, Kuhne CA, Dammann M, Lefering R, et al. Significance of liver trauma for the incidence of sepsis, multiple organ failure and lethality of severely injured patients. An organ-specific evaluation of 24,771 patients from the trauma register of the DGU. Unfallchirurg. 2008;111(4):232–9.
14. Claridge JA, Crabtree TD, Pelletier SJ, Butler K, Sawyer RG, Young JS. Persistent occult hypoperfusion is associated with a significant increase in infection rate and mortality in major trauma patients. J Trauma. 2000;48(1):8–14. **discussion -5**
15. Giamberardino HI, Cesario EP, Carmes ER, Mulinari RA. Risk factors for nosocomial infection in trauma patients. Braz J Infect Dis. 2007;11(2):285–9.
16. Andermahr J, Hensler T, Sauerland S, Greb A, Helling HJ, Prokop A, et al. Risk factors for the development of pneumonia in multiple injured patients. Results of a prospective clinical trial. Unfallchirurg. 2003;106(5):392–7.
17. Andermahr J, Greb A, Hensler T, Helling HJ, Bouillon B, Sauerland S, et al. Pneumonia in multiple injured patients: a prospective controlled trial on early prediction using clinical and immunological parameters. Inflamm Res. 2002;51(5):265–72.
18. Bergeron E, Lavoie A, Clas D, Moore L, Ratte S, Tetreault S, et al. Elderly trauma patients with rib fractures are at greater risk of death and pneumonia. J Trauma. 2003;54(3):478–85.
19. Tejada Artigas A, Bello Dronda S, Chacon Valles E, Munoz Marco J, Villuendas Uson MC, Figueras P, et al. Risk factors for nosocomial pneumonia in critically ill trauma patients. Crit Care Med. 2001;29(2):304–9.
20. Melsen WG, Rovers MM, Bonten MJ. Ventilator-associated pneumonia and mortality: a systematic review of observational studies. Crit Care Med. 2009;37(10):2709–18.
21. • Melsen WG, Rovers MM, Koeman M, Bonten MJ. Estimating the attributable mortality of ventilator-associated pneumonia from randomized prevention studies. Crit Care Med. 2011;39(12):2736–42. **The referenced article based on 58 randomized studies provides accurate and reliable data about the attributable mortality rate of ventilator-associated pneumonia.**
22. Magnotti LJ, Croce MA, Fabian TC. Is ventilator-associated pneumonia in trauma patients an epiphenomenon or a cause of death? Surg Infect. 2004;5(3):237–42.
23. Miller CD, Rebuck JA, Ahern JW, Rogers FB. Daily evaluation of macroaspiration in the critically ill post-trauma patient. Curr Surg. 2005;62(5):504–8.
24. Jones RN. Microbial etiologies of hospital-acquired bacterial pneumonia and ventilator-associated bacterial pneumonia. Clin Infect Dis. 2010;51(Suppl 1):S81–7.
25. Bergallo C, Jasovich A, Teglia O, Oliva ME, Lentnek A, de Wouters L, et al. Safety and efficacy of intravenous tigecycline in treatment of community-acquired pneumonia: results from a double-blind randomized phase 3 comparison study with levofloxacin. Diagn Microbiol Infect Dis. 2009;63(1):52–61.
26. American Thoracic Society, Infectious Diseases Society of America. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med. 2005;171(4):388–416.
27. Monaghan SF, Heffernan DS, Thakkar RK, Reinert SE, Machan JT, Connolly MD, et al. The development of a urinary tract infection is associated with increased mortality in trauma patients. J Trauma. 2011;71(6):1569–74.
28. Bochicchio GV, Joshi M, Shih D, Bochicchio K, Tracy K, Scalea TM. Reclassification of urinary tract infections in critically ill trauma patients: a time-dependent analysis. Surg Infect. 2003;4(4):379–85.
29. Duane TM, Brown H, Wolfe LG, Malhotra AK, Aboutanos MB, Ivatury RR. Bladder pressure measurements are an independent predictor of urinary tract infection in trauma patients. Surg Infect. 2011;12(1):39–42.
30. Duane TM, Young A, Weber W, Wolfe LG, Malhotra AK, Aboutanos MB, et al. Bladder pressure measurements and urinary tract infection in trauma patients. Surg Infect. 2012;13(2):85–7.
31. Nicolle LE. Catheter-related urinary tract infection. Drugs Aging. 2005;22(8):627–39.
32. Peterson J, Kaul S, Khashab M, Fisher AC, Kahn JB. A double-blind, randomized comparison of levofloxacin 750 mg once-daily for five days with ciprofloxacin 400/500 mg twice-daily for 10 days for the treatment of complicated urinary tract infections and acute pyelonephritis. Urology. 2008;71(1):17–22.
33. Hooton TM, Bradley SF, Cardenas DD, Colgan R, Geerlings SE, Rice JC, et al. Diagnosis, prevention, and treatment of catheter-associated urinary tract infection in adults: 2009 international clinical practice guidelines from the Infectious Diseases Society of America. Clin Infect Dis. 2010;50(5):625–63.

34. Hurr H, Hawley HB, Czachor JS, Markert RJ, McCarthy MC. APACHE II and ISS scores as predictors of nosocomial infections in trauma patients. Am J Infect Control. 1999;27(2):79–83.
35. El-Masri MM, Hammad TA, McLeskey SW, Joshi M, Korniewicz DM. Predictors of nosocomial bloodstream infections among critically ill adult trauma patients. Infect Control Hosp Epidemiol. 2004;25(8):656–63.
36. Niven DJ, Fick GH, Kirkpatrick AW, Grant V, Laupland KB. Cost and outcomes of nosocomial bloodstream infections complicating major traumatic injury. J Hosp Infect. 2010;76(4):296–9.
37.• Brown CV, Rix K, Klein AL, Ford B, Teixeira PG, Aydelotte J, et al. A comprehensive investigation of comorbidities, mechanisms, injury patterns, and outcomes in geriatric blunt trauma patients. Am Surg. 2016;82(11):1055–62. **The above reference is an interesting overview about risk factors and outcomes in geriatric trauma. Elderly represents a frail subset of population which is complex to treat but that is steady growing.**
38. Dissanaike S, Shelton M, Warner K, O'Keefe GE. The risk for bloodstream infections is associated with increased parenteral caloric intake in patients receiving parenteral nutrition. Crit Care. 2007;11(5):R114.
39. Emori TG, Gaynes RP. An overview of nosocomial infections, including the role of the microbiology laboratory. Clin Microbiol Rev. 1993;6(4):428–42.
40.•• Oliver M, Dinh MM, Curtis K, Paschkewitz R, Rigby O, Balogh ZJ. Trends in procedures at major trauma centres in New South Wales, Australia: an analysis of state-wide trauma data. World J Surg. 2017. **The referenced article describes the current trend in trauma management. Interventional radiology has increased over the past years whereas emergency surgery has decreased, so too will surgical wound and infections.**
41. Giuffre M, Geraci DM, Bonura C, Saporito L, Graziano G, Insinga V, et al. The increasing challenge of multidrug-resistant gram-negative bacilli: results of a 5-year active surveillance program in a neonatal intensive care unit. Medicine (Baltimore). 2016;95(10):e3016.
42. Morales CH, Escobar RM, Villegas MI, Castano A, Trujillo J. Surgical site infection in abdominal trauma patients: risk prediction and performance of the NNIS and SENIC indexes. Can J Surg. 2011;54(1):17–24.
43. Seamon MJ, Wobb J, Gaughan JP, Kulp H, Kamel I, Dempsey DT. The effects of intraoperative hypothermia on surgical site infection: an analysis of 524 trauma laparotomies. Ann Surg. 2012;255(4):789–95.
44. Richards JE, Kauffmann RM, Zuckerman SL, Obremskey WT, May AK. Relationship of hyperglycemia and surgical-site infection in orthopaedic surgery. J Bone Joint Surg Am. 2012;94(13):1181–6.
45. Herruzo-Cabrera R, Lopez-Gimenez R, Diez-Sebastian J, Lopez-Acinero MJ, Banegas-Banegas JR. Surgical site infection of 7301 traumatologic inpatients (divided in two sub-cohorts, study and validation): modifiable determinants and potential benefit. Eur J Epidemiol. 2004;19(2):163–9.
46. Owens CD, Stoessel K. Surgical site infections: epidemiology, microbiology and prevention. J Hosp Infect. 2008;70(Suppl 2):3–10.
47. Wang YC, Hsieh CH, Fu CY, Yeh CC, Wu SC, Chen RJ. Hollow organ perforation in blunt abdominal trauma: the role of diagnostic peritoneal lavage. Am J Emerg Med. 2012;30(4):570–3.
48. Watts DD, Fakhry SM. Incidence of hollow viscus injury in blunt trauma: an analysis from 275,557 trauma admissions from the East multi-institutional trial. J Trauma. 2003;54(2):289–94.
49. Williams MD, Watts D, Fakhry *S.* Colon injury after blunt abdominal trauma: results of the EAST Multi-Institutional Hollow Viscus Injury Study. J Trauma 2003;55(5):906–912.
50. Malinoski DJ, Patel MS, Yakar DO, Green D, Qureshi F, Inaba K, et al. A diagnostic delay of 5 hours increases the risk of death after blunt hollow viscus injury. J Trauma. 2010;69(1):84–7.
51. Kirkpatrick AW, Baxter KA, Simons RK, Germann E, Lucas CE, Ledgerwood AM. Intra-abdominal complications after surgical repair of small bowel injuries: an international review. J Trauma. 2003;55(3):399–406.
52.• Sartelli M, Viale P, Koike K, Pea F, Tumietto F, van Goor H, et al. WSES consensus conference: guidelines for first-line management of intra-abdominal infections. World J Emerg Surg. 2011;6:2. **A comprehensive article which methodologically addresses intra-abdominal infections according to pathogen and organ involved.**