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Severe sepsis and septic shock
Management and performance improvement

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Morbidity and mortality from sepsis remains unacceptably high. Large variability in clinical practice, plus the increasing awareness that certain processes of care associated with improved critical care outcomes, has led to the development of clinical practice guidelines in a variety of areas related to infection and sepsis. The Surviving Sepsis Guidelines for Management of Severe Sepsis and Septic Shock were first published in 2004, revised in 2008, and recently revised again and published in 2013. The first part of this manuscript is a summary of the 2013 guidelines with some editorial comment. The second part of the manuscript characterizes hospital based sepsis performance improvement programs and highlights the sepsis bundles from the Surviving Sepsis Campaign as a key component of such a program.

Introduction

Morbidity and mortality from sepsis remains unacceptably high.[1,2] Large variability in clinical practice, plus the increasing awareness that certain processes of care associated with improved critical care outcomes, has led to the development of clinical practice guidelines in a variety of areas related to infection and sepsis.[3] The Surviving Sepsis Guidelines for Management of Severe Sepsis and Septic Shock were first published in 2004, revised in 2008, and recently revised again and published in 2013.[4-6] The first part of this manuscript is a summary of the 2013 guidelines with some editorial comment. The second part of the manuscript characterizes hospital based sepsis performance improvement programs and highlights the sepsis bundles from the Surviving Sepsis Campaign as a key component of such a program.

Diagnostic Terminology

Sepsis is defined as infection plus systemic manifestations of infection[7] (**Table 1**). Severe sepsis is defined as infection plus infection induced organ dysfunction or tissue hypoperfusion[7] (**Table 2**). Sepsis induced hypotension is defined as infection induced decrease in blood pressure (systolic pressure <90 mmHg or mean arterial pressure <70 mmHg). Septic shock is defined as the requirement for vasopressors after initial fluid resuscitation fails to correct sepsis induced hypotension.[7]

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Management

Initial resuscitation

Protocolized, quantitative resuscitation of patients with sepsis-induced tissue hypoperfusion (defined as hypotension persisting after initial fluid challenge or a blood lactate concentration ≥4 mmol/L) is recommended.[8-15] For the initial resuscitation of these patients the goals during the first 6 h of resuscitation include a central venous pressure 8–12 mmHg,[16,17] a mean arterial pressure (MAP) ≥65 mmHg,[18,19] a urine output ≥0.5 mL/kg/h, and a superior vena cava venous oxygen saturation of ≥70%.[20]

In patients who are found to initially have elevated lactate levels, targeting resuscitation to normalize lactate is suggested. Normalization of lactate seems a more appropriate goal than a percent reduction in baseline elevated lactate, although the latter has been demonstrated to be an effective resuscitation target variable.[21,22] Where capability to measure central venous oxygen saturation does not exist, lactate clearance can be used as an alternative. Where both technologies are available, both targets are recommended.

Diagnosis of infection

Early diagnosis of sepsis, source of sepsis, and ideally causative organism is important.[23-25] Two sets of blood cultures (both aerobic and anaerobic bottles) should be obtained before initiation of antimicrobial therapy unless it induces a significant delay (greater than 45 min) in the administration of antimicrobials.[26,27] At least one of these blood cultures should be drawn percutaneously and one drawn through each vascular access device, unless the device was recently (less than 48 h) inserted. Imaging studies should be obtained promptly to confirm a potential infection source.

Prevention of selective oral decontamination and selective digestive decontamination should be considered as an ICU wide process to prevent the occurrence of sepsis and severe sepsis.[28-30] Oral chlorhexidine gluconate is suggested as a form of oropharyngeal decontamination to reduce the risk of ventilator-associated pneumonia in ICU patients with severe sepsis.

Treatment of infection

Antimicrobials administered within the first hour of recognition of severe sepsis and septic shock should be the "goal" of therapy.[31-36] Although an admirable goal, this time window is not the current standard of clinical practice. Initial empiric anti-infective therapy should be broad and target all likely pathogens and

include antimicrobials that penetrate in adequate concentrations into the tissues presumed to be the source of sepsis. The antimicrobial regimen should be reassessed daily with the potential for de-escalation. Combination empirical therapy for a particular known or suspected infecting organism may be considered in certain patient groups such as neutropenic patients; patients with difficult-to-treat, multidrug resistant bacterial pathogens; patients with severe infections associated with respiratory failure and septic shock and for septic shock from bacteremic pneumococcal infections.[37-39] Empiric combination therapy should not be administered for more than 3–5 d. De-escalation to the most appropriate single drug therapy should be performed as soon as the susceptibility profile is known.

Duration of antimicrobial therapy is typically 7–10 d; however, longer courses may be appropriate in patients who have a slow clinical response, an undrainable focus of infection, bacteremia with *Staphylococcus aureus*, *Pseudomonas* ventilator-acquired pneumonia, as well as some fungal and viral infections or immunologic deficiencies, including neutropenia. Antiviral therapy should be initiated as early as possible in patients with severe sepsis or septic shock of viral origin including targeting influenza during flu outbreaks, such as H1N1.[40]

Source control is paramount.[41,42] A specific anatomical diagnosis of infection requiring consideration for emergent source control should be sought and diagnosed or excluded as rapidly as possible, and intervention be undertaken for source control within the first 12 h after the diagnosis is made, if feasible. When source control is needed the "effective" intervention associated with the least physiologic insult should be considered (e.g., percutaneous rather than surgical drainage of an abscess). If intravascular access devices are a possible source of severe sepsis or septic shock, they should be removed promptly after another vascular access has been established. When infected peripancreatic necrosis is identified as a potential source of infection, definitive intervention is best delayed until adequate demarcation of viable and nonviable tissues has occurred.

Hemodynamic support

Crystalloids are the initial fluid of choice in the resuscitation of severe sepsis and septic shock.[43] Hydroxyethyl starches are not recommended.[44-46] Albumin is suggested to be added to crystalloid fluid resuscitation when patients require substantial amounts of crystalloids.[47] Initial fluid challenge in patients with sepsis-induced tissue hypoperfusion with suspicion of hypovolemia should include a minimum of 30 mL/kg of crystalloids (a portion of this may be albumin equivalent). More rapid administration and greater amounts of fluid may be needed in some patients. Fluid challenge techniques should continue as long as there is hemodynamic improvement based either on dynamic (e.g., change in pulse pressure, stroke volume variation) or static (e.g., arterial pressure, heart rate) variables. Methods to assess intravascular volume such as echocardiography for assessment of left ventricular size or ultrasound assessment of inferior vena cava may also be used. Direct measurement of flow with assessment of effect of fluid boluses on stroke volume may be potentially useful, where that technology is available, and may include pulmonary artery catheters for thermodilution cardiac output measurement, esophageal Doppler for assessment of aortic flow and estimation of stroke volume based on arterial pressure waveform assessment using minimally invasive cardiac output measurement technologies such as LiDCO™, PiCCO®, and Flo Trac™. All of these devices have risks and some limitations.

Vasopressor therapy should initially target a mean arterial pressure (MAP) of ≥65 mmHg. Norepinephrine is the first

Table 1. Diagnostic criteria for sepsis

Infection, documented, or suspected, and some of the following:
General variables
Fever, >38.3 °C
Hypothermia (core temperature <36 °C)
Heart rate >90/min^{-1} or more than two SD above the normal value for age
Tachypnea
Altered mental status
Significant edema or positive fluid balance (>20 mL/kg over 24 h)
Hyperglycemia (plasma glucose >140 mg/dL or 7.7 mmol/L) in the absence of diabetes
Inflammatory variables
Leukocytosis (WBC >12 000 μL^{-1})
Leukopenia (WBC count <4000 μL^{-1})
Normal WBC count with greater than 10% immature forms
Plasma C-reactive protein more than two SD above the normal value
Plasma procalcitonin more than 2 SD above the normal value
Hemodynamic variables
Arterial hypotension (SBP <90 mmHg, MAP <70 mmHG, or an SBP decrease >40 mmHg in adults or less than 2 SD below normal for age)
Organ dysfunction variables
Arterial hypoxemia (PaO_2/FiO_2 <300)
Acute oliguria (urine output <0.5 mL/kg/h for at least 2 h despite adequate fluid resuscitation)
Creatinine increase >0.5 mg/dL or 44.2 μmol/L
Coagulation abnormalities (INR >1.5 or aPTT >60 s)
Ileus (absent bowel sound)
Thrombocytopenia (platelet count <100 000 μL^{-1})
Hyperbilirubinemia (plasma total bilirubin >4 mg/dL or 70 μmol/L)
Tissue perfusion variables
Hyperlactatemia (>1 mmol/L)
Decreased capillary refill or mottling

WBC, white blood cell; SBP, systolic blood pressure; MAP, mean arterial pressure; INR, international normalized ratio; aPTT, activated partial thromboplastin time. Diagnostic criteria for sepsis in the pediatric population are signs and symptoms of inflammation plus infection with hyper- or hypothermia (rectal temperature 38.5 °C or <35 °C), tachycardia (may be absent in hypothermic patients), and at least one of the following indications of altered organ function: altered mental status, hypoxemia, increased serum lactate level, or bounding pulses. Used with permission from reference 6 as adapted from reference 125.

Table 2. Severe sepsis

Severe sepsis definition = sepsis-induced tissue hypoperfusion or organ dysfunction (any of the following thought to be due to the infection)
Sepsis-induced hypotension
Lactate above upper limits laboratory normal
Urine output <0.5 mL/kg/h for more than 2 h despite adequate fluid resuscitation
Acute lung injury with PaO_2/FiO_2 <250 in the absence of pneumonia as infection source
Acute lung injury with PaO_2/FiO_2 <200 in the presence of pneumonia as infection source
Creatinine >2.0 mg/dL (176.8 μmol/L)
Bilirubin >2 mg/dL (34.2 μmol/L)
Platelet count <100 000 μL
Coagulopathy (international normalized ratio >1.5)

Used with permission from reference 6 as adapted from reference 125.

choice vasopressor.[48-50] When norepinephrine fails to achieve the MAP target, epinephrine added to and potentially substituted for norepinephrine may be needed to maintain adequate blood pressure.[51,52] Alternatively, vasopressin up to 0.03 units/minute can be added to norepinephrine with the intent of either raising MAP or decreasing norepinephrine dosage.[53] Low dose vasopressin is not recommended as the single initial vasopressor therapy and is not recommended to be used at doses higher than 0.03–0.04 units/minute unless used for salvage therapy (failure of other vasopressors to achieve adequate MAP). Dopamine as an alternative vasopressor agent to norepinephrine is in general discouraged but may be used in highly selected patients groups (e.g., patients with low risk of tachyarrhythmias and absolute or relative bradycardia).[49] Phenylephrine is not recommended in the treatment of septic shock except in circumstances where (a) norepinephrine is associated with serious arrhythmias, (b) cardiac output is known to be high and blood pressure persistently low and difficult to maintain with vasopressor, or (c) as salvage therapy when combined inotrope/vasopressor drugs and low-dose vasopressin have failed to achieve MAP target. Low-dose dopamine should not be used for renal protection.[54] All patients requiring vasopressor therapy should have an arterial catheter placed as soon as practical if resources are available.

During initial resuscitation dobutamine may be used to increase oxygen delivery in the presence of ongoing signs of hypoperfusion (such as lactic acidosis), despite achieving adequate intravascular volume and adequate MAP in patients with $ScvO_2$ <70%. Following initial resuscitation of patients with sepsis induced hypoperfusion, where tissue hypoperfusion persists, a trail of dobutamine infusion up to 20 μg/kg/min may be administered singularly or added to vasopressor (if in use) in the presence of (a) myocardial dysfunction as suggested by elevated cardiac filling pressures and low cardiac output, or (b) ongoing signs of hypoperfusion, despite achieving adequate intravascular volume and adequate MAP.

Steroid therapy

Intravenous corticosteroids are not recommended in the treatment of adult septic shock if adequate fluid resuscitation and vasopressor therapy is able to restore hemodynamic stability.[55-59] In case this goal is not achieved, intravenous hydrocortisone alone at a dose of 200 mg per day (50 mg q6h IV or 50 mg IV followed by 24 h continuous infusion to minimize swings in glucose) for up to 7 d is suggested.[60,61] It is not necessary to use the ACTH stimulation test to identify adults with septic shock who should receive hydrocortisone. Instead, bedside clinical assessment as described above should be used. In patients treated with hydrocortisone for septic shock tapering should be performed when vasopressors are no longer required and steroids may be delivered for up to 7 d.[62] Steroids should not be administered for the treatment of sepsis in the absence of shock.

Other supportive therapy of severe sepsis

Once tissue hypoperfusion has resolved and in the absence of extenuating circumstances, such as myocardial ischemia, severe hypoxemia, acute hemorrhage, or ischemic heart disease, red blood cell transfusion should occur only when hemoglobin concentration decreases to <7.0 g/dL.[63] The anemia of severe sepsis should not be treated with erythropoietin unless another indication exists.[64,65] Fresh frozen plasma should not be used to correct laboratory clotting abnormalities in the absence of bleeding or planned invasive procedures.[66,67] Antithrombin is not indicated to treat severe sepsis.[68,69] In patients with severe sepsis, and without significant risk of bleeding or with planned invasive procedures or active bleeding, transfusion threshold for platelets is <10 000/mm^3.[70] Platelets should be transfused when <20 000/mm^3 if the patient has a significant risk of bleeding and platelet counts ≥50 000/mm^3 should be maintained in the presence of active bleeding or if surgery or invasive procedures are needed. Immunoglobulins are not recommended in adult patients with severe sepsis or septic shock.[71] Possibly exceptions include toxic shock syndrome or severe life threatening H1N1 ARDS. There is no current data that would support the use of intravenous selenium for the treatment of severe sepsis.

In the patient with sepsis induced acute respiratory distress syndrome (ARDS), ARDSnet lung protective strategy is recommended to include targeting 6 mL/kg predicted body weight (PBW) tidal volume and a plateau pressure ≤30 cm H_2O.[72] When a tidal volume of 6 mL/kg/PBW results in plateau pressure >30 cm H_2O then tidal volume is decreased to as low as 4 mL/kg in 0.5 ml/kg/PBW increments in order to achieve a <30 cm H_2O plateau pressure target. Plateau pressures higher than 30 cm H_2O may be allowed in patients with increased chest wall or abdominal elastance (morbid obesity or anasarca). A level of positive end-expiratory pressure (PEEP) should be applied to avoid alveolar collapse at end expiration (atelectotrauma).[73] Strategy based on higher rather than lower levels of PEEP is suggested for patients with sepsis-induced moderate or severe ARDS.[74-77] Recruitment maneuvers are suggested in sepsis patients with ARDS induced severe refractory hypoxemia.[78,79] Prone positioning is suggested to be used in sepsis-induced ARDS patients with a PaO_2/FiO_2 ratio ≤100 mmHg in facilities that have experience with such practices.[80,81]

A conservative rather than a liberal fluid strategy is recommended for patients with established sepsis-induced ARDS who do not have evidence of tissue hypoperfusion.[82] Utilizing a CVP target of <4 mmHg is equally effective as using a pulmonary artery catheter to target a pulmonary artery occlusive pressure of <8 mmHg. In the absence of bronchospasm, β 2-agonists should not be used in patients with sepsis- induced ARDS.[83] Neuromuscular blocking agents (NMBAs) should be avoided in the septic patient without ARDS;[84,85] however, a short course of NMBA is suggested (for not greater than 48 h) in the patient with early sepsis induced ARDS and a PaO_2/FiO_2 <150 mmHg.[86]

When two consecutive glucose levels >180 mg/dL are encountered a continuous infusion of insulin should be instituted, targeting an upper blood glucose ≤180 mg/dL.[87] Hypoglycemia should be avoided.[88] Blood glucose values should be monitored every 1–2 h until glucose values and insulin infusion rates are stable and then every 4 h thereafter.[88] Glucose levels obtained with point-of-care testing of capillary blood should be interpreted with caution, as such measurements may not accurately estimate arterial blood or plasma glucose values.[89-91]

Continuous renal replacement therapies and intermittent hemodialysis are equivalent in patients with severe sepsis and acute renal failure.[92-96] The use of continuous renal replacement therapies to facilitate management of fluid balance in hemodynamically unstable septic patients is an acceptable approach. Sodium bicarbonate given to septic patients with tissue hypoperfusion and a pH ≥7.15 should not be expected to improve hemodynamics or decrease vasopressor requirement when compared with equimolar quantities of crystalloid.[97,98]

Deep vein thrombosis and stress ulcer prophylaxis are both recommended in the patient with severe sepsis.[99-105] Deep vein thrombosis prophylaxis should be given with either daily low-molecular weight heparin (LMWH) or unfractionated heparin (UFH) thrice daily. If creatinine clearance is <30 mL/min and LMWH is given, either dalteparin or another form of LMWH with a low degree of renal metabolism or unfractionated heparin should be used. Severely septic patients with a contraindication to heparin use (e.g., clinically significant thrombocytopenia, severe coagulopathy, active bleeding, recent intracerebral hemorrhage), should receive mechanical prophylactic treatment such as graduated compression stockings or intermittent compression devices, unless contraindicated. It is suggested that patients with severe sepsis receive both pharmacologic therapy and intermittent pneumatic compression devices when there are no contraindications to the use of either therapies in patients with severe sepsis. Stress ulcer prophylaxis is strongly recommended with either an H2 blocker or a proton pump inhibitor. Proton pump inhibitors have a weak preference over H2 blockers.[106,107] In the absence of risk factors, no stress ulcer prophylaxis should be given.

Within 48 h after a diagnosis of severe sepsis/septic shock administer oral or enteral feedings, as tolerated, rather than either complete fasting or provision of only intravenous glucose.[108-111] Low dose feeding beginning with 500 calories per day (intravenous glucose plus enteral feeding) advanced as tolerated, is recommended over initial mandatory full caloric feeding (addition of TPN to achieve) in the first week. There is no indication for specific immunomodulating supplementation in patients with severe sepsis.[112-114]

In patients with severe sepsis and septic shock, it is important to discuss goals of care and prognosis with patients and families.[115-117] As appropriate, the goals of care, including any end of life care planning or the use of palliative care principles should be accomplished. Although goals of care should be addressed as early as feasible, this should occur no later than 72 h following ICU admission.

See **Tables S1–3** for concise summations of SSC guidelines recommendations.

Sepsis performance improvement programs[118-120]

Guidelines have little immediate impact on bedside behavior in the management of disease processes. Guidelines, however, serve as a resource document for creation of treatment protocols that when coupled with audit and feedback as part of a formal hospital based performance improvement initiative can change bedside practice. Bundles represent a number of treatment goals to be achieved in a disease process over a set time period and function as measurable quality indicators. When chart audit scores performance on bundle goals, and is followed by feedback to the treating clinicians (audit and feedback) bedside behavior is likely to change in line with guideline recommendations.

Sepsis bundles are created to act as a cohesive unit to ensure all steps of care are consistently delivered.[121-124] The Surviving Sepsis Campaign and the Institute for Healthcare Improvement collaborated to apply the sepsis guidelines of 2004 to assemble two sepsis bundles, the 6-h resuscitation and 24-h management bundles. Following the creation of the 2012 guidelines, the bundles were revised, creating a 3-h and a 6-h bundle (**Fig. 1**). A free standardized database, provided by the Surviving Sepsis Campaign, allows hospitals to enter de-identified patient data and track sepsis bundle performance and outcomes. Participating hospitals are urged to transmit their Health Insurance Portability and Accountability Act (HIPAA) compliant data to a central repository at the Society of Critical Care Medicine for aggregate analysis. Queries of data and graphical display of bundle indicator performance can be retrieved locally using the electronic database. Patients are identified for entry into the database based on a standardized screening tool (**Fig. 2**). Steps to implement a sepsis protocol are shown in **Table 3**.

Achieving performance improvement goals requires ongoing data collection and feedback. Protocols can be successful in changing bedside behavior only with the application of education and commitment of physician, nursing, and other health care professional champions from key areas of the hospital (ICU, ED, and hospital floors). Success of severe sepsis performance improvement programs require, not only champions but also multidisciplinary commitment from physicians, nurses, pharmacy, respiratory, and administration. Programs must be multispecialty as well, and include medicine, surgery, emergency medicine, and others. Establishing support from key ICU, ED, and floor leaders is crucial. Interdepartmental communication and collaboration facilitate seamless steps in the continuum of care, and give the best chance of success. And ultimately behavior is changed with audit and feedback.

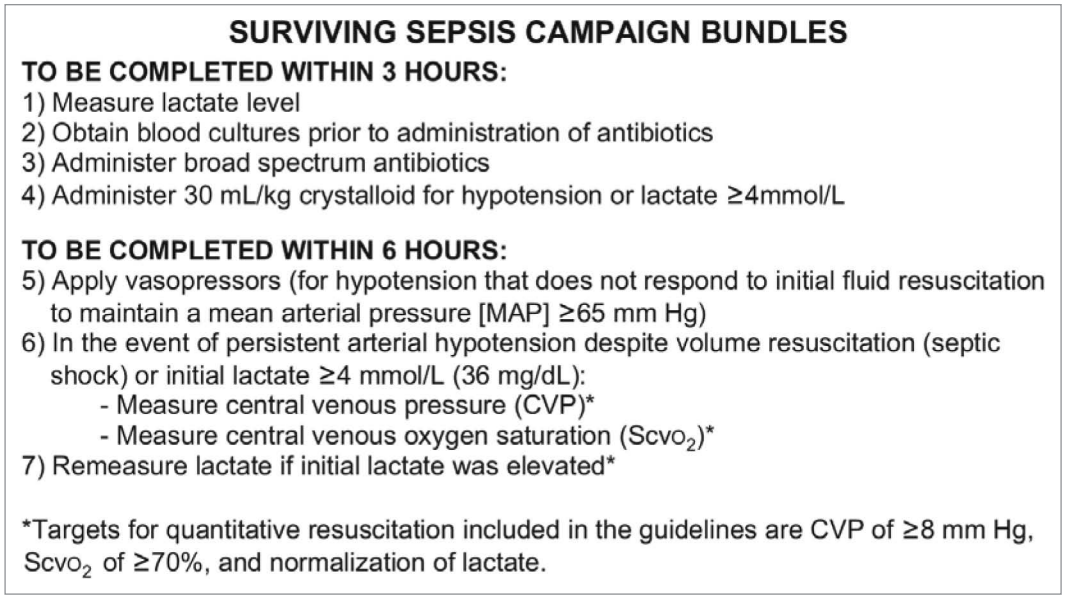

Figure 1. Surviving sepsis campaign bundles. Used with permission from reference 6.

Programs typically start with a hospital-wide education initiative, centered around early identification and familiarity with the treatment protocol that will be applied once the patient is identified. Educational sessions are conducted by members of the sepsis performance improvement leadership team. Education may be provided through departmental conferences, staff meetings, and unit-based in-services. Baseline data may or may not be collected prior to initiation of the formal performance improvement initiative. Data collection typically occurs Monday through Friday morning with a review of patients admitted to the ICU service over the last 24 h, applying the screening tool to ascertain if the patient qualifies for entry into the severe sepsis database. Performance is assessed periodically, typically quarterly through query of the database. The SSC software allows performance to be plotted and displayed over time with tables and linear or bar graphs. This display functions as the feedback tool. Evaluation of process change requires consistent data collection, measurement of indicators and feedback in order to facilitate performance improvement. Ongoing educational sessions to reinforce early identification and treatment steps continue in line with the protocol are needed. When roadblocks are encountered in process improvement a plan, do, study, act process (PDSA cycle) is employed to study the reasons for failure and to implement changes to improve process performance. This process includes initiation of a plan of action, studying results and when problems are identified, altering the plan to solve the problem. Since performance is being judged based on the time to accomplish the indicator, it is necessary to have a time zero (T0) representing when the clock starts ticking for scoring indicator compliance in treatment of severe sepsis. For ED admissions T0 is triage time. For patients presenting with severe sepsis in units other than the ED, T0 is the time that the chart reveals variables allowing the identification of the patient as having severe sepsis.

Conclusion

Only with early diagnosis and expedited treatment based on evidence based medicine can sepsis morbidity and mortality be decreased. Sepsis guidelines create a base to allow change in healthcare practitioner behavior, but lead to only modest slow change in bedside behavior. Change comes when institutions initiate a formal performance improvement program with a formal treatment protocol, education on early identification of severe sepsis patients, followed by audit of performance and periodic feedback to the healthcare professionals taking care of these patients.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

Supplemental Materials

Supplemental materials may be found here:
www.landesbioscience.com/journals/virulence/article/27409

Evaluation for Severe Sepsis Screening Tool

Instructions: **Use this optional tool to screen patients for severe sepsis in the emergency department, on the medical/surgical floors, or in the ICU.**

1. Is the patient's history suggestive of a new infection?

- ☐ Pneumonia, empyema
- ☐ Urinary tract infection
- ☐ Acute abdominal infection
- ☐ Meningitis
- ☐ Skin/soft tissue infection
- ☐ Bone/joint infection
- ☐ Wound infection
- ☐ Blood stream catheter infection
- ☐ Endocarditis
- ☐ Implantable device infection
- ☐ Other infection _____

___ Yes ___No

2. Are any two of following signs & symptoms of infection both present and new to the patient? Note: laboratory values may have been obtained for inpatients but may not be available for outpatients.

- ☐ Hyperthermia > 38.3 °C (101.0 °F)
- ☐ Hypothermia < 36 °C (96.8 °F)
- ☐ Altered mental status
- ☐ Tachycardia > 90 bpm
- ☐ Tachypnea > 20 bpm
- ☐ Leukocytosis (WBC count >12 000 µL–1)
- ☐ Leukopenia (WBC count < 4000 µL–1)
- ☐ Hyperglycemia (plasma glucose >140 mg/dL) or 7.7 mmol/L in the absence of diabetes

___ Yes ___No

If the answer is yes, to both questions 1 and 2, *suspicion of infection* is present:

- ✓ Obtain: **lactic acid**, **blood cultures**, CBC with differential, basic chemistry labs, bilirubin.
- ✓ At the physician's discretion obtain: UA, chest x-ray, amylase, lipase, ABG, CRP, CT scan.

3. Are any of the following organ dysfunction criteria present at a site remote from the site of the infection that are NOT considered to be chronic conditions? Note: in the case of bilateral pulmonary infiltrates the remote site stipulation is waived.

- ☐ SBP < 90 mmHg or MAP <65 mmHg
- ☐ SBP decrease > 40 mm Hg from baseline
- ☐ Creatinine > 2.0 mg/dl (176.8 mmol/L) or urine output < 0.5 ml/kg/h for 2 h
- ☐ Bilirubin > 2 mg/dl (34.2 mmol/L)
- ☐ Platelet count < 100 000 µL
- ☐ Lactate > 2 mmol/L (18.0 mg/dl)
- ☐ Coagulopathy (INR >1.5 or aPTT >60 secs)
- ☐ Acute lung injury with PaO2/FiO2 <250 in the absence of pneumonia as infection source
- ☐ Acute lung injury with PaO2/FiO2 <200 in the presence of pneumonia as infection source

___ Yes ___No

If *suspicion of infection* is present AND *organ dysfunction* is present, the patient meets the criteria for SEVERE SEPSIS and should be entered into the severe sepsis protocol.

Date: ____/____/____ (circle: dd/mm/yy or mm/dd/yy) **Time: ____: ____ (24 h. clock)**

Version 7.2.13

Figure 2. Evaluation for severe sepsis screening tool. Online at http://www.survivingsepsis.org/SiteCollectionDocuments/ScreeningTool.pdf.

Table 3. Steps to implementing a sepsis protocol

• Obtain administrative support
• Evaluate inter-departmental interactions
• Develop and relay a firm understanding of the goals
• Establish a formal interactive relationship with the emergency department and the critical care unit
• Collaborate with the general/internal medicine team
• Identify champions/unit protocol leaders
• Provide a unit/hospital system wide education campaign

Used with permission from reference 126.

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