Mini-review

Clinical Sepsis Mimic

Alfonso Lagi1*, and Simone Cencetti2

1 Internal Medicine Unit, Villa Donatello Hospital, Italy
2 Internal Medicine Unit and Syncope Unit, Santa Maria Nuova Hospital, Italy

*Corresponding author: Alfonso Lagi, Internal Medicine Unit, Villa Donatello Hospital, Florence, Italy, Tel: +39 055 583182; E-mail: alfonso.lagi1@tin.it

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Abstract

Background: Managing patients with an incorrect diagnosis can make a difference in terms of complications and survival. This is true in the field of sepsis and whereas reverse is true in case of septic shock. In this review we did not discuss how to make an alternative diagnosis, but we have demonstrated very different clinical pictures which can mimic the sepsis and/or septic shock. So, we think that a kind of warning is needed to the emergency physicians to alerting their diagnostic feelings and their attention.

Methods: We started from our personal experience in the field and made a rank of the clinical picture with confounding signs and symptoms. We listed: aspiration lung diseases, diabetic ketoacidosis, adrenal insufficiency, acute hypovolemia, pulmonary embolism, acute pancreatitis, intestinal ischemia and thyroid storm. The support of PubMed and Medline has been used in the field at different voices: clinical mimics, sepsis, septic shock, differential diagnosis crossing the different files two by two.

Results: The specific and different clinical picture of the evaluated diseases has been addressed with the aim of approaching them as it is the case, or distinguishing them from sepsis, emphasizing the importance of differentiating signs and symptoms.

Conclusion: Because of significant overlap, sepsis and its mimics are often not differentiable upon initial assessment. There is an important behaviour to consider that is addressing resuscitation before differentiating sepsis and mimics. The message is to approach the patient with sepsis or mimic in a similar modality according to that the resuscitation takes precedence, followed by targeted history and examination.

Keywords: clinical mimics, sepsis, septic shock, acute pancreatitis, diabetic ketoacidosis

Introduction

Many clinical settings can be confused with sepsis for likeness of the signs and symptoms that can occur in clusters similar to each other. The aim of this review concerns the likelihood that clinical pictures different from sepsis can be misled to a false diagnosis and to a treatment delay of morbid events that are dangerous for life.

The awareness of the clinical mimics has recommended a review of the parameters used for the diagnosis of sepsis. The recent consensus "Sepsis-3", delete the old definition where sepsis was the sum of two conditions: a suspected infection and SIRS (Systemic Inflammatory Response Syndrome). The “new” sepsis definition
distinguishes sepsis and septic shock only, the latter distinct from multi-organ failure and defined by the presence of sepsis more hyperlactatemia and hypotension despite adequate fluid infusion.

Thus, sepsis becomes a life-threatening organ dysfunction caused by a dysregulated host response to infection. The hemodynamic change (compensated or uncompensated) distinguishes between an infection without sepsis and a sepsis with or without shock (Figure 1).

![Diagram of relationship between infection-sepsis-septic shock](image)

**Figure 1:** Diagram of relationship between infection-sepsis-septic shock

Despite the introduction of the concept of SOFA (Sequential Organ Failure Assessment) score which explores the progressive worsening of respiratory, cardiovascular, hepatic, renal, neurological and coagulation does not allow overcoming the clinic mimics. So, in the variable clinical pictures included into clinical mimics of sepsis there are in common symptoms, infective foci and organ failure which are confusing signs and symptoms.

**Aspiration lung disease: pneumonia and pneumonitis**

Three main characteristics describe the clinical picture of aspiration, whether the inoculum is infectious or not, the volume of the inoculum, and the acuity of onset of the clinical syndrome.

Commonly, the use of the term 'aspiration' refers to an acute lung infection which develops after a large-volume aspiration of oropharyngeal or upper gastrointestinal contents with pH high enough to avoid chemical pneumonitis. This type of aspiration deposits a large bacterial load from the oral cavity or upper gastrointestinal tract.
into the lungs. The chance of infection with these normally nonvirulent bacteria is partly because of the large inoculum. Not all aspirations cause an inflammatory response in the lung. Therefore, would be inappropriate to label these as pneumonitis and the term pneumonia seems to be more suitable. Probably, two examples are the most common: aspiration of blood as a complication of severe epistaxis or hematemesis and aspiration of enteral feedings. Blood and enteral feedings represent excellent culture media for both resident and inoculum bacteria. Generally, mucociliary clearance and resident alveolar macrophages can clear the inoculum within hours. Consequently, most episodes of aspiration with enteral nutrition are also uncomplicated [1].

Chemical pneumonitis is linked with a clinical picture mimic of sepsis and septic shock. Mendelson first described this specific entity in the anaesthesia literature in 1946 in a series of women who aspirated during obstetric anaesthesia [2]. Animal experiments helped in differentiating the pathophysiology of chemical pneumonitis from subclinical aspiration based on pH and the volume of gastric material needed to stimulate an immediate and severe inflammatory reaction. Based on experiments using human gastric secretions and rabbit lungs, a pH less than 2.4 was required to cause vigorous inflammation. At higher pH, the reaction microscopically seen was more similar to the changes caused by the instillation of water into the lungs [3]. In theory, an average 70-kg patient would need to aspirate more than 20 ml of gastric contents to induce chemical pneumonitis assuming a gastric pH of 1 [4].

It’s a picture that can become dramatic and fast-moving when a high volume is aspirated, over 20 ml. The symptoms and respiratory signs call attention to the doctor: varying from bronchoconstrictory acute symptoms to signs as subtle as tachypnea, dyspnea, cough. Progressive respiratory failure is the dominant syndromic picture, expressed by hypoxia and radiological scattered outbreaks morphologically compatible with Acute Lung Injury and/or Acute Respiratory Distress Syndrome (ARDS). Afterwards, signs and symptoms of shock can appear. The wide range of severity from transient hypoxemia to ARDS has become apparent. Prospective studies of ARDS suggest that 16.5% of patients thought to have experienced aspiration developed ARDS [5]. If ARDS occurs, a particularly severe subtype with a high mortality ensues [6]. In this case the confusing signs and symptoms are the presence of a clinical picture characterized by pulmonary crazy – paving signs associated with respiratory failure and shock with multi-organ failure.

In the clinical setting, we must be careful to dysphagia, typically from neurologic disease (dementia, Parkinson disease, multiple sclerosis, post stroke), which is considered the most important risk factor for aspiration. Although sedatives may suppress the patient’s mental status sufficiently to lead to aspiration, antipsychotic medications may actually affect the swallowing mechanism by inhibiting dopamine and therefore lead to aspiration. Probably the highest risk of aspiration occurs in the severe alcohol abuse population. Acute alcohol ingestion has multifactorial risks for aspiration, increased risk of vomiting, and direct effects of alcohol on normal neutrophil function.

**Diabetic Ketoacidosis**

The real incidence of Diabetic Ketoacidosis (DKA) is difficult to establish. Population based studies range from 4.6 to 8 episodes per 1,000 patients with diabetes. DKA remains a significant clinical problem and a life-threatening complication despite improvements in diabetes care. DKA is a complex disordered metabolic state characterised by hyperglycaemia, acidosis, and ketonaemia. It occurs because of absolute or relative insulin deficiency that is accompanied by an increase in counter regulatory hormones (i.e. glucagon, cortisol, growth hormone, catecholamines). This type of hormonal imbalance enhances hepatic gluconeogenesis and glycogenolysis resulting in severe hyperglycaemia. Enhanced lipolysis increases serum free fatty acids that are then metabolised as an alternative energy source in the process of ketogenesis. This result in accumulation of large quantities of ketone bodies and
subsequent metabolic acidosis. DKA has been considered to be indicative of type 1 diabetes, but increasingly there are cases of ketone-prone type 2 diabetes being recognised. However, the diagnosis and initial treatment is the same.

There are several mechanisms responsible for fluid depletion in DKA. These include osmotic diuresis due to hyperglycaemia, vomiting, commonly associated with DKA, and finally inability to take fluids due to a diminished level of consciousness. Electrolyte shift, and depletion are in part related to the osmotic diuresis. Hyperkalaemia and hypokalaemia need particular attention [7].

The beginning of DKA has to make it think to an infective, acute, undercurrent disease and is characterized by the appearance of symptoms of respiratory compensation like tachypnoea and dyspnoea. Hyperglycaemia and polyuria contributes to hypovolemia and hypotension. Frequently, latent organ failure is associated with diabetes, so that hypovolemia increases it and the signs of that organ failure appears, in particular is the case of renal failure. Finally, the drowsiness and mental confusion quickly occur because of the complex metabolic derangement. In conclusion, signs and symptoms that describe the clinical picture can be confused with septic shock: mental confusion, hypotension, metabolic acidosis, renal failure and the infective focus.

Adrenal insufficiency

Usually, adrenal crisis occurs in subjects with chronic adrenal failure who are under severe physiologic stress, such as infection, trauma, burns, surgery, and myocardial infarction. A severe physiologic stress can rapidly deplete the already limited patient’s cortisol reserves, making the subjects unable to mount an adequate stress response. The incoming of acute adrenal failure is a brisk and dramatic event.

Its incidence in the western world is low, 40-60 cases per million with the diagnosis peaks during the fourth to sixth decades of life. This endocrine disorder can occur as an isolated process or as part of an autoimmune polyendocrine syndrome known as the polyglandular autoimmune syndrome types I and II.

Adrenal crisis is a great imitator and it often presents with a constellation of nonspecific signs and symptoms that can mislead even the most diligent emergency physician. The hallmark of adrenal crisis is hypotension, shock (90% of the cases) refractory to fluid resuscitation and vasopressors. Patients have nonspecific symptoms including fever (65%), abdominal pain (22%), nausea, vomiting (47%), lethargy, malaise, weakness, and confusion (42%) [8,9]. When acute adrenal insufficiency is revealed by an infectious disease in the background, the clinical picture has many elements in common with septic shock.

In this case the confusing signs are fever, infection, hypotension, drowsiness and mental confusion. Furthermore, vomiting and hypotension may cause renal failure so that SOFA score is satisfied.

Acute Hypovolemia

Hypovolemic shock refers to a rapid fluid loss which results in multiple organ failure due to inadequate circulating volume and subsequent inadequate perfusion. In these cases, the critical point is the endothelium suffering. It plays a critical role in vascular pathophysiology. The functions of the endothelium change after the hypovolemic shock because of the ischemia of the endothelial cells and the reperfusion due to resuscitation with fluids. In virtue of oxygen deprivation, endothelial cell apoptosis is also induced following the hypovolemic shock. The organ failure is the effect of this damage [10]. Without fluid and blood resuscitation and/or correction of the underlying pathology that cause the loss of fluids, a multiple organ failure soon follows. Furthermore, the diagnosis is easy if the loss of fluid is identified. Otherwise, in relation to the percentage of the volume loss [11], the confusing signs and symptoms are hypotension, tachycardia, tachypnoea and different organ failure as kidney, lung and significant changes in mental status, such as confusion or agitation.
Pulmonary embolism

It is a frequent occurrence, characterized by a broad clinical spectrum from pauci-symptomatic to sudden death. The association between pulmonary embolism (PE) and shock may vary with the extent of pulmonary embolism and underlying cardiopulmonary impairment. Tachypnea and tachycardia are common but nonspecific findings. Signs and symptoms of both deep venous thrombosis and pulmonary embolism may be highly suggestive but are neither sensitive nor specific. The possibility of massive pulmonary embolism should be considered in patients who have a sudden onset of near syncope or syncope, hypotension, extreme hypoxemia, electromechanical dissociation or cardiac arrest [12]. Low-grade fever is not uncommon in PE, and high fever, although rare, may occur. Fever is related with complication of PE, pulmonary haemorrhage or infarction [13]. In conclusion, PE may be an imitator of septic shock in specific cases only. It occurs when PE has begun some days before, as an aspecific disease complicated with pulmonary infarction or haemorrhages with secondary pneumonia or abscess. In this case then, the confusing signs and symptoms with septic shock are hypotension, tachycardia and tachypnea in presence of fever.

Acute Pancreatitis

Acute pancreatitis is an inflammation of the pancreas; sometimes, it is associated with a systemic inflammatory response that can impair the function of other organs or systems. The distant organ or system dysfunction may resolve or may progress to some organ failure.

The most important risk factors for pancreatitis in adults are gallstones and excessive alcohol use [14]. Other causes include metabolic aberrations (e.g. hypertriglyceridemia), duct obstruction (e.g. related to a neoplasia or pancreas divisum), medications (e.g. azathioprine, thiazides, and oestrogens), and trauma. The clinical picture is different.

There are abdominal symptoms and signs: patients mention a severe and constant abdominal pain (resembling peritonitis), usually the onset is sudden and associated with vomiting. Abdominal examination shows epigastric tenderness, with guarding. Differential diagnosis includes abdominal infection like cholecystitis, diverticulitis and peritonitis.

There are systemic symptoms and signs: tachycardia, leucocytosis, fever and tachypnea (signs of SIRS) are the framework of pancreatitis [15]. One hundred fifty-five of 252 patients (62%) had SIRS on day 1. A total of 187/252 patients (74%) had SIRS during the first 5 days of hospitalization [16].

The clinical diagnosis of acute pancreatitis is based on the typical abdominal pain and nausea, combined with elevated serum levels of pancreatic enzymes. This is a characteristic sign and it is not present in septic shock. According to the SOFA score, the involvement of digestive system in sepsis results in the presence of a liver disease and high transaminases and/or bilirubin.

In a setting of pancreatitis there were 38 (15%) patients with organ failure during the first 7 days of hospitalization [16].

Therefore, the clinical mimic of pancreatitis vs septic shock is referred to an infection associated with SIRS at the beginning. In the next days, the picture of septic shock is completely displayed when the failure of an organ occurs. Pulmonary, renal or hepatic damage by radiological outbreaks morphologically compatible with acute ARDS, increased creatinine or transaminases. Finally, signs of mental confusion may be associated with the fever and increased lacticidemia related with hypoperfusion of peripheral tissues.
Intestinal ischemia

The splanchnic circulation receives approximately 25% of the resting and 35% of the postprandial cardiac output. Acute mesenteric ischemia (AMI) can be categorized into three specific types based on its cause and different clinical implications: arterial embolism, arterial thrombosis, and venous thrombosis.

Arterial emboli are the most frequent cause of AMI and are responsible approximately for the half of the cases. Most mesenteric emboli originate from a cardiac source and hit the superior mesenteric artery, distally to the origin of the middle colic artery. Usually, the onset of symptoms is dramatic because of the poorly developed collateral circulation. It is characterized by the abrupt beginning of severe abdominal pain associated with diarrhoea, which may become bloody: the prognosis is poor within few hours or days.

Acute mesenteric thrombosis accounts for one-third of all ischemic events. Almost all mesenteric ischemia due to arterial thrombosis occur in setting of severe atherosclerotic disease. Patients can tolerate major visceral artery obstruction, because the slow progressive nature of atherosclerosis allows the development of collaterals. Bowel ischemia or infarction ensues when the last remaining visceral artery or an important collateral artery occludes. The extent of bowel ischemia or infarction is typically greater than that with embolism, extending from the duodenum to the transverse colon.

Mesenteric venous thrombosis representing up to one fifth of all patients with mesenteric ischemia. Mortality depends on the type of thrombosis (acute vs chronic) and the extent of venous involvement.

The colon is predisposed to ischemia because of its less developed microvasculature plexus in comparison to those of the small bowel. Two major arteries supply most of the blood to the colon: the superior mesenteric artery (which supplies the ascending and transverse colon) and the inferior mesenteric artery (which supplies the descending and sigmoid colon). The internal iliac arteries supply the rectum. The colon is protected from ischemia by a collateral blood supply by a system of arcades connecting the two major arteries. However, the anatomy is highly variable, and, in some people, certain areas are more vulnerable.

Many conditions may predispose to intestinal ischemia leading to occlusive vascular disease like hypoperfusion due to transient hypotension in the perioperative period or shock due to a variety of causes (e.g. hypovolemia or sepsis) can result in intestinal ischemia [17].

The clinical presentation varies, depending on the severity and extent of the disease. None of the symptoms and signs is specific. Most patients present with a sudden onset of crampy abdominal pain, diarrhoea and an urge to defecate. The pain is mild, located over the affected bowel, usually to the left side of the lower abdomen and hypogastrium, followed by mild rectal bleeding within 24 h. The blood may be bright red or maroon, frequently mixed with the stools. In these cases, the diagnosis is not difficult but in cases of severe ischemia with transmural infarction and necrosis, marked tenderness with peritoneal signs may be present on physical examination accompanied by metabolic acidosis and septic shock.

The clinical picture of colon ischemia is not specific and is highly variable. Diagnosis requires a high index of clinical suspicion. The chronology of symptoms and the clinical situations upon which these symptoms appear must be taken into account. Common clinical conditions should be excluded. The differential diagnosis includes infectious colitis, inflammatory bowel disease, pseudomembranous colitis, diverticulitis and colon carcinoma. Severe forms may be difficult to distinguish from AMI.

In general, patients with embolism or thrombosis of superior mesenteric artery have an acute onset of symptoms and a rapid deterioration in their clinical condition, whereas those with mesenteric venous thrombosis have a more gradual onset and a more protracted clinical course.
Acute mesenteric ischemia should particularly be considered in the differential diagnosis when a patient is older than 60 years; has a history of atrial fibrillation, recent myocardial infarction, congestive heart failure, arterial emboli; and is initially seen for abdominal pain that is out of proportion to that suggested by physical examination. High levels of serum amylase, aspartate aminotransferase, lactate dehydrogenase, and creatine phosphokinase are frequently observed at presentation, but none is sufficiently sensitive or specific to be diagnostic.

Therefore, owing to different clinical presentations, many signs and symptoms confusing with septic shock are present after severe ischemia such as hypotension, tachycardia, fever and foci of infection as effect of colonic bacterial growth. Dehydration and excessive fluid loss from third-spacing of fluid lead to mental confusion, tachypnea, and circulatory collapse. Laboratory findings include metabolic acidosis with elevated anion gap and lactate levels, leukocytosis, and hemoconcentration.

In particular, the clinical examination and its conclusion can bring to think that patients are suffering from an infectious disease inside the abdomen. Furthermore, proves of infection can be found in the stool cultures as well as in the blood, which is the outcome of bacterial translocation through intestinal wall [18]. At this point it is easy to find signs of multiorgan failure [19].

Thyroid storm

Thyrotoxicosis is a common condition associated with symptomatic excess of circulating thyroid hormones. In Europe, it affects annually around 1 in 2000 people. Although thyrotoxicosis typically presents with weight loss, heat intolerance, and palpitations, there are a large variety of additional features, which manifest more variably with advancing in age. Graves' disease is the most common cause, accounting for the vast majority of cases. Secondly, there is the toxic nodular goitre, more common in people aged over 60 years. Rarely, other causes, such as thyroiditis (subacute, silent, or post-partum) that release pre-formed thyroid hormones into the circulation as a result of inflammatory destruction of the follicles. Finally, there is also the gestational hyperthyroidism occurring in the first trimester of pregnancy owing to increased secretion of thyroid hormone in response to placental β human chorionic gonadotrophin, which is structurally similar to TSH. Several drugs, like amiodarone, iodine, lithium, active retroviral therapy and tyrosine kinase inhibitors, can cause thyrotoxicosis in different ways. The diagnosis of thyrotoxicosis is done on the clinical symptoms and signs (sweating, weight loss, heat intolerance, palpitations, breathless, tremor, anxiety, and tiredness) and laboratory data (increased serum free thyroxine levels and/or triiodothyronine in the presence of fully suppressed serum TSH levels).

Among patients suffering from thyrotoxicosis, a particular clinical picture is described as ‘thyroid storm’ (TS) [20]. It is life threatening. Next time this condition is referred as thyrotoxic crisis but till today physicians continue to have difficulty in diagnosing the condition, and groups around the world are attempting to establish clear diagnostic criteria based on universal clinical parameters.

The condition arises in few thyrotoxic patients, no more than 1-2% [21], and is manifested by the decompensation of multiple organs, often triggered by severe stress. Since its pathophysiologic mechanisms have not been clarified, the diagnosis of TS is based on clinical manifestations. Some of the characteristic clinical features include unconsciousness, high fever, heart failure, diarrhoea, and jaundice.

Differences arise in the strength and not in the quality of symptoms and signs, between patients with TS and those with thyrotoxicosis without TS. In particularly there was no difference in the values of thyroid hormones, T3 and T4, as if the difference between TS and thyrotoxicosis was the effect of increased tissue sensitivity to the action of thyroid hormones [22]. So, the clinical picture of TS is characterized by at least one manifestation of central nervous system dysfunction (restlessness, delirium, mental aberration/psychosis, somnolence/lethargy, convulsion, coma.
including a score of 14 or lower on the Glasgow Coma Scale) and at least three combinations of fever or tachycardia, or heart failure or GI/hepatic manifestations [22].

Multiple organ failure was the most common cause of death in much severe forms, followed by heart failure, respiratory failure, arrhythmia, disseminated intravascular coagulation, gastro-intestinal perforation, hypoxic brain syndrome, and sepsis. Even with early diagnosis, the overall mortality remains high, between 10% to 30%.

It’s clear that signs and symptoms confusing with septic shock are in common with TS because some SOFA parameters appear in the clinical picture. The diagnosis becomes straightforward when the physician has the tendency to resort to the dose of thyroid hormones.

Liver failure (LF) type A

Hepatic encephalopathy is distinguished in three types of A, B and C, depending on whether they are associated with Acute liver failure, porto-systemic Bypass or liver Cirrhosis respectively.

This section refers to a complex clinical picture characterized by the association of a sudden impairment of hepatic function (defined by jaundice and coagulopathy) with a variable encephalopathy picture (cerebral edema with its signs or symptoms of endocranial hypertension), often associated with acidosis, kidney failure, sepsis and cardiovascular collapse [23]. It is a "potentially reversible pathology due to an important liver injury associated with the appearance of encephalopathy within eight weeks of the first symptom (jaundice) and the absence of a pre-existing chronic liver disease”.

In Europe, the most common cause is viral: hepatitis B virus associated with or without the delta [24] antigen and hepatitis A virus. Paracetamol intake in UK [25] and the US is followed. The hepatitis C virus rarely causes acute hepatic failure in developed countries while it is in the east with the hepatitis E virus. It should be noted that more than 800 pharmacological molecules potentially capable of causing hepatic type failure A [26].

Among the signs and symptoms of LF there are the signs of acute encephalopathy (cognitive alterations prevalent to the asterixis that is rarer) to which corresponds to cerebral edema (identifiable with cerebral imaging) associated with jaundice and hyper-ammonia. The onset of encephalopathy can be gradual, or sudden, and only occasionally precedes the onset of jaundice. The clinical picture does not differ significantly from that of encephalopathy of cirrhosis, but some signs are confusing because significant extrapyramidal changes, typically found in chronic liver encephalopathy there are not detected, or ascites defining acute LF during chronic liver disease. Hyper-lactacidemia reflects both the degree of liver dysfunction and the level of tissue oxygenation, helping to define, along with other parameters, the prognosis [27,28].

The gradual progression of LF becomes dramatic for an advancing multi-organ failure, associated with hypotension and tachycardia going to end to clinical picture of shock secondary to dehydration and metabolic acidosis. Fever is more frequent in patients with acute liver failure by drug ingestion. The concomitant infection complicates the prognosis and the differential diagnosis with septic shock as it contributes to the appearance of fever and indicates a possible septic outbreak. The origin of the infection is possible in every site, but especially in the respiratory tract, the urogenital apparatus or the colon.

Conclusion

In this review we did not discuss how make an alternative diagnosis, but we liked to demonstrate that very different clinical pictures can mimic the sepsis and/or septic shock. Managing patients with an incorrect diagnosis can make a difference in terms of complications and survival. Because of significant overlap, sepsis and its mimics are often not differentiable upon initial assessment. There is an important behaviour to consider that is addressing resuscitation before differentiating sepsis and mimics. Airway assessment, with establishment of definitive airway if
needed, in conjunction with evaluation of breathing adequacy and circulatory status are paramount. Obtaining intravenous access and appropriate diagnostic studies is necessary. These studies include a complete blood cell count, a basic metabolic panel, urinalysis, a chest radiograph, blood cultures, and lactate. Initiation of fluid bolus should be conducted to improve perfusion and preload. If sepsis is suspected, broad-spectrum antimicrobials should be initiated [29]. In the setting of patients with hypotension lactate elevations is a marker associated with increased mortality, especially in a progressive and proportional way to its growth [30,31].

Once resuscitation has been initiated, the focused history and examination can then be completed, evaluating for other conditions and sources. This can allow the doctor to target resuscitation and management to the clinical condition. With completion of the initial assessment and first stages of resuscitation, the next task is to search for a potential source in the setting of sepsis. The aim is to finalize the possible information for diagnostic purposes on history, physical examination and laboratory examinations to make a proper diagnosis of sepsis. The LUCCAASS mnemonic will assist the search for source: lung (pneumonia), urine (cystitis/pyelonephritis), cardiac (endocarditis), central nervous system (CNS) (meningitis, encephalitis), abdominal (abscess, cholecystitis), arthritis (septic arthritis), spine (osteomyelitis, abscess), and skin (cellulitis, IV line/peripherally inserted central catheter infection) [32,33]. There may be situations in which no infective source is found: this happens around in 40% of the cases [34].

The message is to approach the patient with sepsis or mimic in a similar modality according to that the resuscitation takes precedence, followed by targeted history and examination. Consideration of sepsis versus its mimics can be completed while resuscitation is underway. Once an aetiology is discovered, treatment and management can be targeted.

Because sepsis is a time dependent disease, the future perspectives are in a rapid and correct diagnosis which allows to gain time.

References