Abdominal signs and symptoms in intensive care patients

Annika Reintam Blaser¹, Joel Starkopf¹, ², Manu L.N.G. Malbrain³

¹Department of Intensive Care Medicine, Lucerne Cantonal Hospital, Lucerne, Switzerland and Department of Anaesthesiology and Intensive Care, University of Tartu, Tartu, Estonia
²Tartu University Hospital, Tartu, Estonia
³Intensive Care Unit and High Care Burn Unit, Ziekenhuis Netwerk Antwerpen, ZNA Stuivenberg, Antwerp, Belgium

Abstract

Abdominal problems, both as a primary reason for admission or developing as a part of multiple organ dysfunction syndrome during an ICU stay, are common in critically ill patients. The definitions, assessment, incidence and outcome of different abdominal signs, symptoms and syndromes are assessed in the current review. General abdominal signs and symptoms include abdominal pain and distension, as well as other signs assessed during the physical examination (e.g. palpation, percussion). Gastrointestinal (GI) symptoms include vomiting, high gastric residual volumes, diarrhoea, GI bleeding, paralysis of the lower GI tract, bowel dilatation and absent bowel sounds. Although around half of patients suffer from these symptoms, the reported incidences of single symptoms vary within a large range due to variable definitions and case-mix. In a few studies, the total number of coincident GI symptoms was associated with increased mortality. Although acute abdomen is a well-recognized severe syndrome in emergency medicine, its incidence in ICUs is not known. Next to subjective clinical evaluation, intra-abdominal pressure, as a reproducible numerical variable, provides useful assistance in the assessment of the abdominal compartment, whereas intra-abdominal hypertension has been shown to impair the outcome of the critically ill. In conclusion, abdominal symptoms occur in half of patients in ICUs. Clinical evaluation, albeit largely subjective, remains the main bedside tool to detect abdominal problems and to assess GI function in the critically ill. IAP is a useful additional tool in the assessment of abdominal complications in ICUs.

Key words: abdominal signs, abdominal symptoms, abdominal syndromes, abdominal hypertension, vomiting, diarrhoea, bleeding, paralysis, ileus, bowel dilatation, bowel sounds, gastric residual volume

Abdominal problems are common in critically ill patient. Although they can be the initial condition requiring admission to the intensive care unit (ICU), most often they are a reflection of the severity of the underlying disease. The interactions of a critical illness with abdominal contents are complex and can present themselves in different ways. In order to perform the correct treatment, a complete initial clinical and diagnostic work up for gastro-intestinal (GI) problems, with early assessment of disease severity (looking at aetiological factors and predisposing conditions) is necessary. This should be followed by appropriate monitoring, such as intra-abdominal pressure (IAP) measurements, in order to anticipate complications and to avoid remote organ failure. This global approach should form the basis of patient management.

Within this respect, abdominal signs are an important diagnostic tool to trigger further investigations in emergency-room patients. In ICU patients they are often masked or difficult to assess in the critically ill. Although most of the GI symptoms are not specific, they have been associated
with impaired ICU outcomes [1, 2]. The current review was undertaken to clarify the assessment, incidence and impact of different abdominal signs and symptoms in intensive care patients.

**METHODS**

A MEDLINE and PubMed search was performed using the search terms ‘abdominal’, ‘gastrointestinal’ and ‘symptoms’. This search yielded many references, most of which were not relevant to the subject of this paper. The abstracts were screened and selected on the basis of relevance, methodology and number of cases. Full text articles of the selected abstracts were used to supplement the authors’ expert opinion and experience. The references were also checked to identify other relevant interesting papers.

**RESULTS AND DISCUSSION**

There is no clear nomenclature for abdominal signs, symptoms and syndromes. Different terms for diagnoses, symptoms and syndromes are sometimes used interchangeably. Many definitions are not applicable in critically ill patients, because they include the patient’s subjective description (e.g. constipation). Definitions for different abdominal symptoms and syndromes specifically in ICU patients were recently proposed by the Working Group on Abdominal Problems (WGAP) of the European Society of Intensive Care Medicine (ESICM) [3].

Various abdominal pathologies are often a primary reason for admission to an ICU (Table 1). On the other hand, abdominal problems may develop as a part of multiple organ dysfunction syndrome during an ICU stay (Table 2). These problems may lead to a certain diagnosis (e.g. Ogilvie’s syndrome, bowel ischemia, cholecystitis, peritonitis) or should be assessed as acute gastrointestinal injury (AGI) [3].

**GENERAL ABDOMINAL SIGNS AND SYMPTOMS**

**ABDOMINAL PAIN**

The type, location, severity, chronology and duration of pain give important clues as to the source of the abdominal problem [4]. Diffuse abdominal pain and tenderness is seen in most patients with acute abdominal problems although it can be subtle and masked in the elderly, and by the use of corticosteroids [5]. Abdominal pain is a key symptom of peritonitis. However, as the correct assessment of abdominal pain is not possible in the unconscious or sedated, mechanically ventilated patients, it may be therefore very difficult to reach the diagnosis of peritonitis in such patients. Visceral pain is often poorly localized because of its dull and aching character. It arises from distension or spasm of a hollow organ (e.g. obstruction or cholecystitis). Parietal pain is very well localized and sharp. It arises from peritoneal irritation (e.g. acute appendicitis). Referred pain is perceived to be

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<th>Table 1. Overview of acute abdominal diagnoses that may require ICU admission (listed in order of frequency per organ)</th>
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near the surface of the body and aching (e.g. basal pneumo-
nia). The location of an abdominal pain with regard to the
abdominal quadrants also helps to narrow the differential
diagnosis (Fig. 1). The incidence and possible associations
with the outcome of abdominal pain in the critically ill are
not known.

**ABDOMINAL DISTENSION**

Abdominal distension is a non-specific sign that may,
but does not need to originate from the gastrointestinal
tract. It can be defined as a sagittal abdominal diameter
(SAD) that is greater than the rib cage or the hip height.
Looking sideways at a patient in a supine position this would
mean that the shape of the abdomen is like a curved line
that extends above the virtual line between the xiphoid and
symphysis in a convex way. Studies have shown that obesity
and intra-abdominal hypertension (IAH) are associated with
an increased SAD [6, 7]. Causes for abdominal distension are
related to an increase in intra-abdominal volume: ascites,
bowel edema, hematomata, bowel distension, or ileus.

**Abnormal findings on percussion** include tympanism in
case of high content of air in the abdomen or in the intesti-
tines and dullness in case of large amount of fluids (e.g.
ascites).

**Abdominal discoloration** may occur as a sign associ-
ated with abdominal wall infection or with pancreatitis
(Grey-Turner's or Cullen's signs).

General abdominal signs may be important triggers for
further investigations, but are never diagnostic by themse-

**GASTROINTESTINAL SYMPTOMS**

**ABSENT OR ABNORMAL BOWEL SOUNDS**

For an assessment of bowel sounds, auscultation for at
least one minute in two quadrants, repeated at least once
within a tight time frame, should be performed [5]. Palpation
of the abdomen should be performed after auscultations as
it may stimulate subsequent bowel sounds [8]. In general
medicine, absent or abnormal (for example tinkling) bowel
sounds have been considered as an important symptom
of acute abdominal pathology. In intensive care, ausculta-
tion of bowel sounds – widely used but seldom correctly
performed — is often regarded as a method having no
particular importance. Diminished bowel peristalsis and
the absence of bowel sounds are common in mechanically
ventilated patients receiving sedatives, opiates and/or cate-
cholamines. The studies suggest that the bowel sounds
may be decreased or absent in half of intensive care patients
[1, 9–11]. The lack of uniform definition and subjectivity in
assessment are obvious reasons why only few studies have
investigated whether the presence of absent peristalsis has
at all any impact for intensive care patient. Nevertheless, two
studies have demonstrated that absence of bowel sounds
is associated with impaired outcome [1, 2].

**HYPERPERISTALSIS**

Hyperperistalsis is defined as the presence of excessive
bowel sounds. Excessive and tinkling bowel sounds are
characteristic of bowel obstruction [12]. As bowel dilatation
and/or (pseudo)obstruction are potentially life-threaten-
ing conditions [12, 13], the occurrence of hyperperistalsis
deserves particular attention. The true incidence of this
symptom in intensive care patients, as well as the impact
on outcomes, however, is not known.

**VOMITING (EMESIS)**

Vomiting or emesis should be defined in ICU patients as
the occurrence of any visible regurgitation of gastric content
irrespective of the amount [3]. In spontaneously breathing
patients, it is characterized by a contraction of the abdomi-
nal muscles, descent of the diaphragm, and opening of the gastric cardia, resulting in a forceful expulsion of stomach contents from the mouth [14]. In mechanically ventilated patients the forcefulness of the act is often undetectable; therefore, vomiting and regurgitation (effortless passage of gastric contents into the mouth) should be assessed together. The incidence of vomiting highly depends on the case mix of an ICU. Concomitant chemotherapy, recent anaesthesia and surgery, as well as abdominal and/or central nervous system (CNS) diseases are well known predisposing factors for vomiting [15]. Data in the general cohort of critically ill patients are scarce. Many studies investigating nutritional support do not report the incidence of vomiting [16]. In others, incidences ranging from 10 to 20% have been reported [1, 10, 15, 17–20]. Increased IAP may interfere with vomiting. Patients with IAH have been demonstrated to suffer from vomiting/regurgitation in 48.5% of ICU days, in contrast to 28.3% in patients without IAH [21]. Serious complications...
such as aspiration pneumonia, dehydration, malnutrition, and disruption of the surgical site are often attributed to vomiting and regurgitation [15, 22]. Whether vomiting has an association with ICU mortality and length of stay is not clear. Mentec et al. [10] have shown that vomiting and upper digestive intolerance are associated with nosocomial pneumonia and mortality in nasogastric tube-fed intensive care unit patients. In our previous studies, no difference in mortality between patients with and without vomiting/regurgitation was found [1, 2].

GASTRIC RESIDUAL VOLUME (GRV)

The GRV should be regularly measured according to most of the feeding protocols. The latest study, however, showed no increase in complications, but better achieved caloric goals in patients without GRV monitoring [23]. This finding might change this protocolized approach in the future. The technique for GRV measurement is neither unified nor validated [24−26] and therefore has a wide variation — for example, active aspiration vs. passive outflow of gastric contents, replacement vs. discarding the contents — can be found. Delayed gastric emptying occurs in up to 30–50% of critically ill patients [13] and can result in an increased GRV. Depending on the threshold used for high GRV, it may occur in 5 to 30% of intensive care patients [13, 14, 20]. A GRV above 150 mL has been observed in 28% of medical ICU patients [17]. If 500 mL is taken as the threshold, the incidence is about 5% [2]. The ESICM working group recommends one to consider GRV high if a single volume is > 200 mL [17, 25, 27, 28] or a total gastric aspirate volume >1,000 mL per 24 hours [3]. Although high GRV requires specific attention, it should not automatically result in the discontinuation of enteral feeding. High GRV has been associated with an increased risk of pulmonary aspiration and impaired outcomes in some earlier studies [10]. However, a delay in enteral feeding has similarly been linked to poor results [29]. Therefore, balanced decisions should be made at which levels of GRV enteral feeding has to be withheld. Montejo et al. have demonstrated that increasing the cut-off for normal GRV to 500 mL is not associated with increased adverse effects of EN, gastrointestinal complications or in outcome variables [30]. In a prospective study of 377 patients, we did not find a difference in the incidence of high GRV (above 500 mL) between survivors and non-survivors [2].

GI BLEEDING

Gastrointestinal bleeding is defined as any bleeding into the GI tract lumen, confirmed by macroscopic presence of blood in vomited fluids, gastric aspirate or stool [3]. Asymptomatic, endoscopically evident mucosal damage occurs in the majority of ICU patients [11]. Clinically evident GI bleeding, reflecting considerable damage to GI mucosa may be seen in 5–25% of ICU patients [2, 11]. Overt bleeding associated with hemodynamic compromise or the need for blood transfusions, occurs in 1.5–4% of mechanically ventilated patients [11]. GI bleeding itself may increase mortality rates [2], while the risk factors for GI bleeding are mechanical ventilation for > 48 hours and coagulopathy [31, 32]. Several studies have confirmed the benefit of stress ulcer prophylaxis [33−36] while the use of H₂ blocker or proton pump inhibitors is recommended for patients who have risk factors for bleeding [37].

DIARRHOEA

Diarrhoea is defined as having three or more loose or liquid stools per day with a stool weight > 200–250 g per day (or > 250 mL per day) [3]. Due to the lack of a standardized definition, especially in earlier studies, the reported incidences of diarrhoea vary over a very wide range from 2 to 95% [38]. Diarrhoea is, however, a significant problem in intensive care patients which interferes with the adequacy of enteral nutrition [39]. Besides enteral nutrition itself, hypo-albuminemia, intestinal ischaemia, as well as medication use (antibiotics, laxatives) have been demonstrated as risk factors for diarrhoea [39, 40]. The main consequence of diarrhoea is malnutrition, which, if not properly handled, can increase mortality rates [39]. Severe diarrhoea can also result in other serious problems, such as haemodynamic instability due to fluid and electrolyte losses. The severity of the symptoms partly depends on the aetiology of the diarrhoea. Clostridium difficile infection is most common nosocomial cause of diarrhoea, and may occur in up to 4% of intensive care patients [41].

Moreover, one-in-five of these patients may develop fulminant colitis with a mortality rate of nearly 60% [42, 43].

PARALYSIS OF THE LOWER GI TRACT (PARALYTIC ILEUS)

Paralysis is the inability of the bowel to pass stool due to impaired peristalsis. Clinical signs include the absence of stool for three or more consecutive days without mechanical obstruction. Bowel sounds may or may not be present. Paralytic ileus is inevitable after abdominal surgery and lasts usually 3 to 5 days [13]. In addition to surgery, other factors such as mechanical ventilation, increased intracranial or intra-abdominal pressure, sedation or sepsis may contribute to development of paralytic ileus [13]. Importantly, in analgo-sedated, mechanically ventilated patients, GI paralysis may be the only sign of ongoing peritonitis. Further risk factors include volume overload [44, 45], hypotension, and use of drugs with known inhibitory effects on GI motility (catecholamines, opioids). In most severe cases, paralytic ileus may lead to severe bowel dilatation, needing immediate interventions. Paralytic ileus is a risk factor of IAH.
[46], which in turn is associated with impaired survival. The mortality of Ogilvie’s syndrome is reported to be between 10–15% in patients without complications and up to 50% if complications such as ischemia or perforation occur [47, 48].

**BOWEL DILATATION**

Bowel dilatation is present if the colonic diameter is > 6 cm (> 9 cm for caecum) or small bowel diameter is > 3 cm, diagnosed either on a plain abdominal x-ray or CT scan [3]. Although bowel dilatation is a common sign of obstruction at any level of the GI tract, it may also appear without an obstruction (as in toxic megacolon or acute colonic pseudo-obstruction = Ogilvie’s syndrome). The most common predisposing conditions for Ogilvie’s syndrome are non-operative abdominal trauma, infections and cardiac diseases [47, 48]. Complex measures including minimal invasive surgery, epidural anaesthesia, restrictive fluid management, early enteral feeding and mobilisation (ERAS protocol) are effective in order to avoid/reduce the duration of postoperative GI paresis and enhance one’s recovery after elective abdominal surgery [49].

An increasing number of GI symptoms is associated with impaired outcome [1, 2]. Therefore, several GI symptoms occurring together should be considered as a sign of gastrointestinal dysfunction, possibly contributing to impaired outcome.

**ABDOMINAL SYNDROMES**

**ACUTE ABDOMEN**

“Acute abdomen” is a syndrome needing immediate intervention. Some of the conditions leading to “acute abdomen” rarely occur in ICU occupants (aortic rupture, ruptured ectopic pregnancy), whereas the others may develop at any stage during the ICU stay (bowel ischemia, ulcer perforation). The symptoms associated with “acute abdomen” are abdominal pain, abdominal distension, nausea, fever, rigidity and/or involuntary contraction of the abdominal muscles in response to palpation [50]. Even if the symptoms that may refer to the presence of “acute abdomen” are the most important to be recognized immediately, data about the incidence of these symptoms in ICUs is scarce [5]. In a patient with abdominal sepsis, an often-overlooked clinical sign is altered mental status. This alteration initially may be so subtle that only close relatives can detect it. In ongoing sepsis, alterations in mental status can range from agitation, anxiety, somnolence, delirium, stupor, epileptic insult to coma. This is called septic or metabolic encephalopathy. Anorexia and nausea are frequent and may precede the emergence of abdominal pain by some time. Vomiting can be caused by hollow viscus obstruction or peritoneal inflammation [51]. Other important elements are: the amount of gastric residuals, the aspect of gastric contents (feculent, bloody, bilious), and the presence of hematemesis. Diarrhoea is another clinical sign and stool consistency can be changed (or mixed with blood) by alterations in gut mucosal flora or gut hypoperfusion. Fever is clearly the most common manifestation of abdominal sepsis, although it can be masked in immunocompromised patients with neutropenia or under corticosteroid therapy. A patient with severe sepsis can also present one with hypothermia. Some believe that the skin is the mirror of the “inner human being”. If the skin is mottled the intestinal mucosa probably is too. One should look at the turgor of the skin and the presence or absence of sudor or sweating, and local or generalised signs of inflammation, the presence of central or peripheral cyanosis (livedo reticularis). One should assess whether the extremities are cold or warm and if the capillary refill is normal (less than two seconds). The outcome of patients with an acute abdomen depends on many issues. The development of GI complications during an ICU stay has been repeatedly shown to increase mortality rates [2, 52–54]. Timely recognition of possible abdominal complications is therefore of utmost importance, independent of whether the patient is admitted because of acute abdomen or develops it as a complication during ICU treatment. Together with a clinical evaluation, diagnostic procedures must always be performed with biochemical and radiological (X-ray, ultrasound or CT) analysis. Cultures should always be taken in case of suspicion of abdominal sepsis. After initial assessments, the clinician should be able to distinguish promptly abdominal problems necessitating immediate surgery. Moreover, when a surgical intervention is planned, it should not be deferred [55].

**PSEUDO-ACUTE ABDOMEN**

One must always keep in mind other nonabdominal causes of acute abdomen such as inferior myocardial infarction, basal pneumonia, diabetic ketoacidosis, uremia, porphyria, adrenal insufficiency, electrolyte disturbances, lead poisoning, sickle cell crisis, or hemolysis. These conditions are sometimes referred to as “pseudo acute abdomen”. Figure 1 summarizes all other conditions that can present as an acute abdomen.

**INTRA-ABDOMINAL HYPERTENSION (IAH)**

Intra-abdominal hypertension (IAH) is defined as a sustained or repeated IAP, equal or above 12 mm Hg [56, 57]. IAH should also be considered present if the mean of the IAP measurements of the day is 12 mm Hg or higher provided that at least 4 measurements were performed [58]. The most severe manifestation of IAH is abdominal compartment syndrome (ACS), defined as a sustained IAP > 20 mm Hg that is associated with new organ dysfunction / failure [56, 57]. Unlike the symptoms reflecting abdominal compartment, IAH is clearly defined and IAP is a measurable variable. An
increase in IAP influences other organ systems and vice versa [59–66]. At the same time, the exact associations between IAH and gastrointestinal (GI) function/dysfunction are not clear [21]. Although IAP monitoring is strongly supported in mechanically ventilated patients with severe burns, severe trauma, severe acute pancreatitis, liver failure or ruptured aortic aneurysm [67], it should also be considered early in patients with a largely positive fluid balance and other conditions associated with decreased abdominal wall compliance or increased intra-abdominal volume [56]. The ICU patients in whom IAP measurements were not initiated on admission should undergo careful bedside evaluation with a low trigger for starting IAP monitoring [67]. The true incidence of IAH in ICU patients is not known, as no study included all consecutive patients. IAH may occur in 24–38% of patients with an ICU stay >24 hours [46, 68–73], whereas a higher incidence is reported in sub-groups of severe burns (37%) [74], in ruptured abdominal aortic aneurysm (55%) [75], in severe acute pancreatitis (78%) [76] and in severe sepsis (77%) [77]. IAH is reported to occur in 12% of patients after major elective abdominal surgery [5, 78] and in 46% after emergency abdominal surgery needing intensive care [79]. Several studies have reported higher mortality in patients with IAH compared to patients without this syndrome [46, 70, 80], whereas some recent studies do not confirm this finding [2, 69]. Outcomes independently associated with IAH are difficult to determine, as IAH commonly develops in more severe patients [46, 70, 80]. Moreover, there are many interactions with other organ systems and applied treatments. The fact that recent studies do not show independent effects of IAH on mortality might be a reflection of success in the management of IAH during recent years.

CONCLUSIONS

Abdominal symptoms occur in half of the patients in an ICU. Clinical evaluation, albeit largely subjective, remains the main bedside tool to detect abdominal problems and to assess GI function in the critically ill. IAP is a useful additional tool in the assessment of abdominal complications in an ICU. The independent impact of different abdominal symptoms and syndromes remains to be identified. In order to achieve this, a standardized assessment of abdominal signs and symptoms in the critically ill is warranted.

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Saccharomyces boulardii

Risk factors for gastrointestinal failure

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Corresponding author:
Annika Reintam Blaser
Clinic of Anaesthesiology and Intensive Care,
University of Tartu, Tartu University Hospital,
Puusepa 8, 51014 Tartu, Estonia
e-mail: annika.reintam.blaser@ut.ee

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