

## Acute Respiratory Distress Syndrome due to Acute Mesenteric Ischemia

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### Abstract

**Introduction:** An important and critical cause of acute respiratory failure is respiratory distress syndrome (ARDS). It can be precipitated by several clinical disorders, including pneumonia, sepsis, aspiration of gastric contents, and major trauma.

We describe a case of a patient who developed ARDS due to acute mesenteric ischemia.

**Case Report:** TL, an 89-years-old, Caucasian female, visited the emergency room after experiencing domestic maxillo-facial trauma. During the first days of hospitalization the patient presented with fever, generalized abdominal pain and nausea and diarrhea, with mild tenderness on deep palpation.

She was subjected to an abdominal computed tomography scan with contrast which detected acute mesenteric ischemia (AMI) and showed a rare anatomical variation of the celiac trunk: the common hepatic artery arose from the superior mesenteric artery and not from the celiac trunk.

She was treated by angiography and revascularization. In the following days, the patient experienced severe hypoxemia and acute lung failure. The chest x-ray showed bilateral ground glass infiltrates.

Thus, oxygen supplementation was administered until noninvasive ventilation was required.

Nevertheless, the progression of ARDS in the subsequent days led to clinical worsening and the patient's transfer to the intensive care unit.

**Conclusion:** AMI is a vascular emergency with high mortality.

This case report describes how an ischemia/reperfusion injury can cause an ARDS due to a systemic inflammatory response. Prompt diagnosis and management of AMI are essential for bettering the prognosis and decreasing the risks of mortality.

**Keywords:** ARDS; ALI; Acute Mesenteric Ischemia

### Introduction

Acute respiratory distress syndrome (ARDS) is caused by severe pulmonary injury that determines alveolar damage heterogeneously throughout the lung [1].

The diagnosis of ARDS is based on the Berlin's criteria: arterial hypoxemia with a  $\text{PaO}_2/\text{FiO}_2$  ratio (ratio of partial pressure arterial oxygen and fraction of inspired oxygen) less than 300 mmHg and less than 200 mmHg to define Acute Lung Injury (ALI), bilateral radiographic pulmonary infiltrates, no clinical evidence of cardiogenic pulmonary edema [1].

ARDS is recognized as the most severe form of acute lung injury (ALI).

The pathophysiological basis of ALI/ARDS involves the activation and modulation of various inflammatory and immune events. The ischemia creates the absence of an oxygen supply and nutrients and the restoration of blood circulation (reperfusion) causes a oxidative damage to the tissue, the release of inflammatory mediators and the influx of inflammatory cells to local and remote organs [2-4]. A common complication after intestinal ischemia (I/R) is ALI that, when severe, can lead to ARDS with high mortality [5].

In 2005, the estimate annual incidence of ALI and ARDS in adults in the US was estimated to be approximately 1:200,000 patients, and the mortality was around of 40% [6,7].

Several clinical disorder, such as pulmonary (pneumonia, which can be bacterial, viral or fungal) or non-pulmonary (peritonitis, sepsis, pancreatitis, aspiration of gastric contents, transfusion-associated lung injury, and drug reactions) have been associated with the development of ALI/ARDS [8-11].

Intestinal ischemia and reperfusion (I/R) injury can lead to ALI and after acute ARDS. Barrier dysfunction due to intestinal ischemia-reperfusion eventually effects the translocation of bacteria and toxins through a leaky gut mucosa, and amplifies or perpetuates systemic inflammation and oxidative stress, leading to multiple organ dysfunction syndrome and death in critically ill patients.

Several reviews have shown that females are protected against lung damage that is induced by intestinal I/R through an estrogen-mediated mechanism [12].

Acute mesenteric ischemia (AMI) is a potentially fatal vascular emergency, with a high mortality and it requires prompt diagnosis and treatment. The prognosis is determined by several conditions: the severity of lung injury, the extent of non-pulmonary organ dysfunction, preexisting medical conditions, and the quality of supportive care. Treatment of this life-threatening condition includes surgical resection of the necrotic bowel, restoration of blood flow to the ischemic intestine and supportive measures, fluid resuscitation and hemodynamic support. The principal therapies for ARDS - lung-protective ventilation and conservative fluid management – can reduce mortality and morbidity, respectively [13].

### Case Report

TL, an 89-years-old, Caucasian female with a medical history of dyslipidemias was admitted to the emergency room after a domestic maxillo-facial trauma. There was no relevant personal or family of history cardiovascular disease and no history of non-steroidal anti-inflammatory drug use or other previous/current medications.

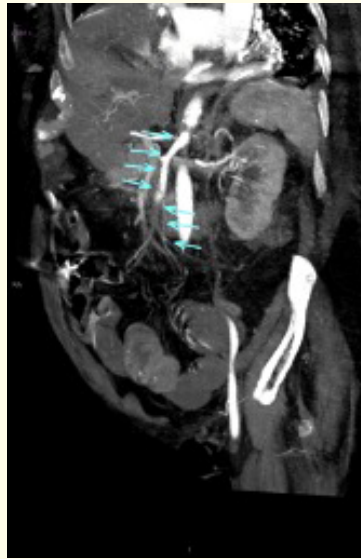
The initial physical examination showed small abrasions and lacerations on the facial skin and no vomiting, seizures, or ear bleed. There was no other neurological or bone complaints. The patient was conscious and oriented. The computed tomography (CT) of the skull noted only a small fracture of the zygomatic bone was highlighted. The x ray was negative for infiltrates and pleural problems.

After 4 days the patient presented with fever, generalized abdominal pain, nausea and diarrhea. It could not be resolved with painkillers. She denied any chills, cough, or chest pain.

On the physical examination, hypoactive bowel sound were noted, accompanied by tenderness of the entire abdomen, especially the periumbilical area. Muscle guarding was not significant. The Murphy sign reveled no significant tenderness, nor was there any in McBurney's point. Bilateral knocking pain was negative. In the extremities, no significant peripheral arterial occlusive sign was noted. The patient initiated antibiotic therapy with carbapenems (meropenem 2gr x 3/daily) and tigecycline at initial dose of 100 mg, followed by 50 mg every 12 hours and metoclopramide.

Blood cultures for aerobic and anaerobic bacteria were negative. The patients maintained 95% O<sub>2</sub> saturation on room air.

Her abdominal CT (Figure 1) revealed complete acute occlusion of the superior mesenteric artery, several centimeters downstream of the origin.

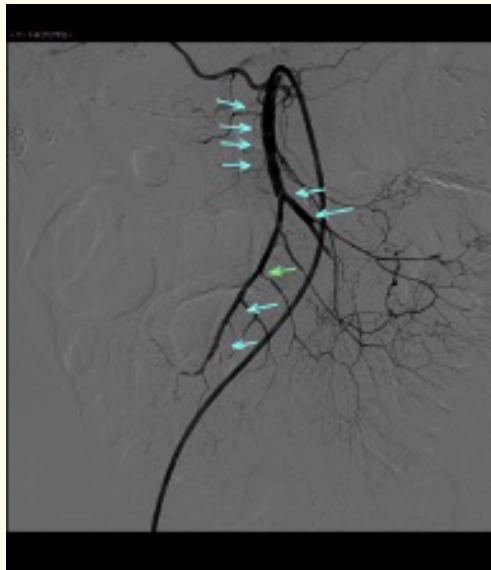


**Figure 1:** Image of abdominal CT, arterial phase, reconstruction by maximum intensity projection of superior mesenteric artery: patency of first tract (arrow from left) and complete acute occlusion several centimeters downstream of the origin (arrow from right).

After the CT scan, the patient underwent angiographic revascularization by mechanical thromboaspiration with success (Figure 2 and 3). The laboratory evaluation is listed in table 1 guiding the correction of an electrolyte imbalance and blood transfusion.



**Figure 2:** Image of diagnostic angiography of superior mesenteric artery: confirmation of patency of first tract (arrow from left) and complete acute occlusion several centimeters downstream of the origin (arrow from right). Note that the hepatic artery arises as first collateral on the left.

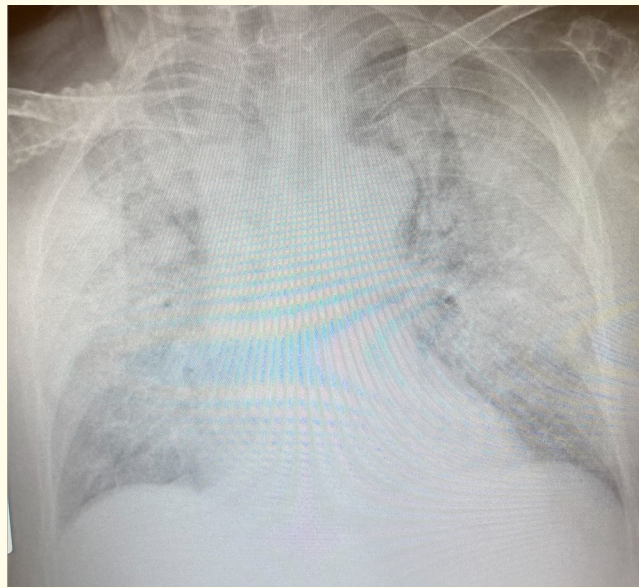


**Figure 3:** Angiographic image of superior mesenteric artery after mechanical thromboaspiration: complete patency of the artery.

Variables	Results	Normal range
White blood cell (Netrophils)	14.000	4.8 - 10.2 u/l
	93	42.2% - 75.2%
Hemoglobin	8.0	12.0 - 16.0 g/dl
Platelet count	27.000	130.000 - 400.000 u/l
D-dimer	6.58	< 0.5 ng/ml
PT	65	70 - 130%
Fibrinogen	178	180 - 400 g/dL
C-reactive protein	95	0 - 5 mg/dL
SGOT	151	5 - 34 U/L
SGPT	124	0 - 55 U/L
Total Bilirubin	5.71	0.0 - 1.2 mg/dL
Direct Bilirubin	3.86	0.0 - 0.5 mg/dL
Na <sup>+</sup>	168	136 - 145 mEq/l
K <sup>+</sup>	3.2	3.4 - 4.4 mEq/l
LDH	1126	125 - 220 U/L
Procalcitonin	2.35	< 0.1 ng/mL
Glucose	148	70 - 110 mg/dL
Creatinine	1.39	0.55 - 1.02 mg/dL

**Table 1:** Results of the initial laboratory tests.

Several days later the patient experienced initial lung failure with low blood oxygen. The blood gas analysis showed a pattern of hypoxic-hypocapnic respiratory failure with metabolic acidosis and elevated lactate levels: pH: 7.28 (n.v 7.35 - 7.45), PaO<sub>2</sub>: 50 mmHg (n.v 75 - 100); PaCO<sub>2</sub>: 30 mmHg (n.v 35 - 45) and lactate 3,3 mmoli/Lm (n.v. 0,3 - 2,5). She started oxygen therapy; the chest x-ray showed bilateral radiographic pulmonary infiltrates (Figure 4). Consequently, the patient presented with signs of respiratory fatigue and was thus treated with a non-invasive ventilation (NIV) in total full face which provided initial benefit.



**Figure 4:** Chest x-ray: bilateral radiographic pulmonary infiltrates.

Nevertheless, the progression of the ARDS in the following days led the patient to clinical worsening and the patient's transfer to an intensive care unit.

### Discussion

ARDS is recognized as a major clinical problem worldwide and it is associated with high morbidity and mortality [6,14,15].

The physiopathology of ARDS can be divided into several levels. With regard to the alveolar capillary unit, an insult is applied to the lung, resulting in diffuse damage to this blood-gas barrier and, thus impairing gas exchange. The host's inflammatory response determines the development and the progression of ARDS. The inflammatory process leads to alveolar and interstitial edema, reduced alveolar fluid clearance, impairment of surfactant production and function and lung fibrosis; the circulation of inflammatory mediators from damaged lung tissue determines systemic inflammation and can lead to multiple organ failure.

ARDS has many risk factors: pulmonary infection or aspiration, but also extra-thoracic conditions such as sepsis, trauma, massive transfusion, drowning, drug overdose, fat embolism, inhalation of toxic fumes, and pancreatitis, that through inflammatory cascade culminate in pulmonary injury [5]. Even acute intestinal ischemia and reperfusion injury can lead to ARDS [6].

In this case report we describe the critical evolution of an AMI: septicemia due to infarction of the bowel and ARDS as a consequences of the massive inflammatory response.

AMI is a rare but emergent disease with a high mortality rate. Its complications, such as sepsis and multiple organ failure, have a high incidence in patients in whom the diagnosis is delayed.

Sepsis and ARDS have similar underlying mechanisms, characterized by inflammation and endothelial dysfunction. In addition, severe sepsis is the most common etiology of ARDS, and patients with sepsis-induced ARDS have higher case fatality rates than those with other risk factors of ARDS.

However, its nonspecific presentation can confuse physicians and delay a timely diagnosis. A delayed or missed diagnosis can lead to bowel necrosis and multiple organ failure. A sound diagnostic tool is important for shortening the time to diagnosis. Computed tomography and ultrasonography have been reported to be effective diagnostic methods, but vascular intervention (angiography) is the standard allowing early treatment to restore the intestinal blood flow [16]. In our case the patient presented elevated lactate (frequently associated in mesenteric ischemia) [16-18] and D dimer levels, even if further studies are needed on D-dimer use in mesenteric ischemia.

In our case the patient was treated with NIV, which provided an initial benefit. Limitations with the use of NIV in ARDS have been reported in recent guidelines. A trial session of NIV can be offered to a patient with hypoxemic respiratory failure, such as community acquired pneumonia and early ARDS, provided that he is managed and monitored by an experienced clinical team [19].

### Conclusion

Intestinal ischemia and reperfusion injury can lead to ARDS from which patients have high mortality. Prompt diagnosis and management of these conditions are essential. Endovascular management can be considered as a primary modality of treatment in selected cases. A multidisciplinary and multimodal management approach is needed to increase the survival.

### Conflict of Interests

All authors declare that they have no competing interests.

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