

Role of bedside-focused ultrasonographic evaluation in the critical patient: a case report

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ABSTRACT

We present the case report of an 80 years old woman with late post-traumatic (of low intensity without any immediate clinical alteration) onset of dyspnoea, chest pain and hypotension, in which bedside ultrasonography has been a very useful tool in immediate diagnostic definition (acute respiratory distress syndrome – ARDS due to multiple pulmonary contusions), considering and excluding all possible alternative diagnosis with the same clinical presenta-

tion, but also providing a “real time” evaluation of therapeutic regimen (hypovolemia versus excessive fluid embalance) and, last but not least, the chance to perform a close follow up of the ultrasonographic alterations (pleural effusion, signs of alveolar consolidation) pointed out at initial diagnosis. Moreworthly, it has been possible to identify the previous mentioned lesion before they had become evident to standard X-Ray chest evaluation.

SINTESI

Presentiamo il caso clinico di una donna di 80 anni che, in seguito ad un trauma di intensità lieve senza ripercussioni cliniche avvenuto qualche giorno addietro, presentava dispnea, dolore toracico ed ipotensione; l'ecografia eseguita “al letto” ha costituito uno strumento molto utile nel conseguire una definizione diagnostica pressoché immediata (sindrome ARDS dovuta a contusioni polmonari), considerando ed escludendo nel contempo tutte le possibili diagnosi alternative con la mede-

sima presentazione clinica; è stata possibile inoltre una valutazione in tempo reale dell'efficacia della terapia (ipovolemia versus sovraccarico volumico) e non ultimo per importanza, è stato eseguito un attento monitoraggio delle alterazioni ecografiche (versamento pleurico, segni di consolidamento polmonare) evidenziati alla diagnosi iniziale. Inoltre abbiamo rilevato che l'identificazione delle lesioni sopra menzionate è stato più precoce rispetto alla radiografia del torace standard.

Introduction

Bedside-focused ultrasonographic evaluation is a very useful tool, in addition to clinical examination, in order to define critical conditions eligible for a rapid diagnostic definition and an intensive therapeutic approach; herein we present a case report in which the coexistence of signs and symptoms common to different pathological conditions leads us to formulate multiple diagnostic hypotheses and to quickly resolve them with the aim of bedside-focused ultrasonography evaluation.

Case report

An 80-year-old caucasian woman was admitted to our hospital because of progressive dyspnea and rapid onset of chest pain – defined by “borderline” features suggestive of acute coronary syndrome¹ – but absent of fever, cough and any other symptoms. Her past medical history was unremarkable except for chronic haemodynamically stable atrial fibrillation not requiring antiarrhythmic drugs but treated with anticoagulant therapy. She referred of recent left thoracic trauma five days before and presented signs of wall thoracic ecchymosis. Upon clinical examination, airflow was adequate but she presented severe hypotension (80/50 mmHg), heart rate 110/min, Glasgow Coma Scale 15/15, and dyspnea (respiratory rate 32/min, SO₂ 80% with FiO₂ 0.21). Clinical, rectal and neurological examinations were normal without any evidence of gastrointestinal blood loss. Axillary temperature was 36.5°C, while her electrocardiogram revealed atrial fibrillation with normal ventricular rate and morphology. An arterial blood gas specimen (performed with Radiometer-ABL) revealed pH 7.35, PO₂ 35 mmHg, PCO₂ 48 mmHg, HCO₃ 25 mEq/l with calculated PaO₂/FiO₂ = 166 (with haemoglobin 9.0 mg/dl and lactate 2.6 mmol/l). Chest X-Ray examination did not detect any pathological findings in the lungs and ribs. Clinical suspicion of pulmonary embolism was unlikely according to Well's Score and Ginevra score.

Reservoir 100% oxygen supply and fluid challenge with normal isotonic saline at a rate of 250 ml/30 minutes were started and a vesical catheterism was performed in order to monitor diuresis. Awaiting emocromocitometric and other clinical biochemical examinations, we performed bedside-focused emergency ultrasonography evaluation. Lack of collapse of inferior vena cava with inspiration ruled out a hypovolemic condition (Figures 1 and 2), the presence of free peritoneal fluid was excluded, while cardiac transthoracic exploration of the heart excluded acute left ventricular failure, cardiac tamponade and acute right ventricular dilatation due to severe pulmonary hypertension; chest ultrasonography detected a non-homogeneous pattern of multiple B-lines arising from the pleural line, mostly in the left emithorax, with confluent consolidations zone and the presence of focal signs of parenchymal distruption with localized pleural effusion

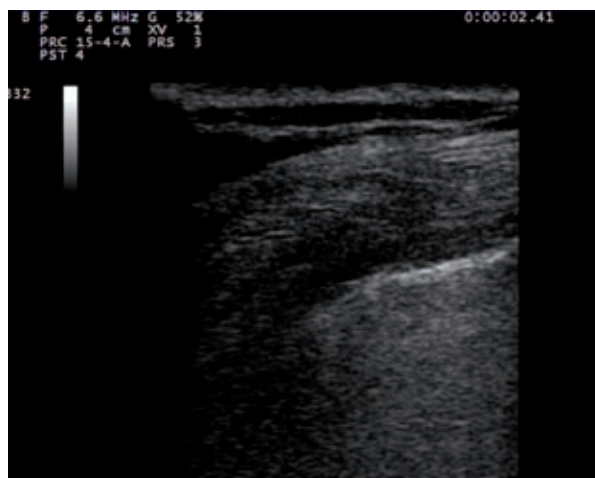


Fig. 1 - Ultrasonography at admission.



Fig. 2 - Ultrasonography after three days.

(Figures 3, 4 and 5), which is a picture of classic alveolar-interstitial syndrome pattern according to applying classic guidelines², while an alternative diagnosis of cardiogenic pulmonary edema, pneumothorax, pericardial tamponade and pulmonary embolism was excluded.

Hematologic and chemistry laboratory values were: Ht 29,5%, Hb 92 g/l, white cell count 14,000/mm³, neutrophils 12,700/mm³, platelet count 133,000/mm³, INR 2.45, fibrinogen 2.75 g/l, D-Dimer 142 µg/ml, total bilirubin 1,32 mg/dl, direct bilirubin 0,3 mg/dl, albumin 2.9 g/l, ferritin 45 mg/dl, CRP 11 mg/l. Conventional I-troponin monitoring was normal as well as myoglobin (Table 1). The main diagnostic hypothesis was consistent with “acute respiratory distress syndrome” (ARDS) secondary to a pulmonary contusion. Therefore the diagnosis was confirmed by a thorax and abdominal contrasted-enhanced computed tomography

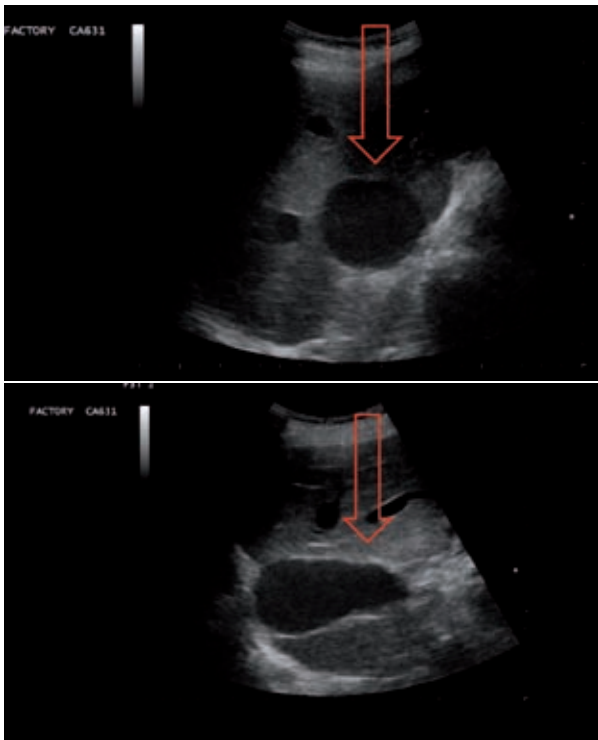


Fig. 3, 4 - A dilatated IVC (IVC diameter > 20 mm) rules out hypovolemia; IVC is measured in the sub-xiphoid space in the long and trasversal axis, using the liver as a sonographic window.

Table 1

Hematologic and chemistry laboratory values.

	Admission	III day	2 weeks after
Hb (g/l)	92	60	104
MCV (fl)	71	76	78
White cell (per mm)	14000	2800	9600
Platelet count (per mm)	133000	80000	204000
Fibrinogen (g/l)	2,75	3,93	3,2
Total bilirubin (mg/dl)	1,32	1,34	0,98

scan, which revealed minimal bilateral pleural effusion with multiple parenchymal infiltrates compatible with post-contusive consolidations and hematoma of thoracic wall.

A serious anemia and thrombocytopenia have subsequently there occurred due to excessive platelet consumption, thus the patient received transfusion with two packed red cell units.

The ultrasognographic findings described above underwent to close follow up and correlation with clinical condition and therapy's adjustment:

- After first three days of fluid therapy at rate of 60-80 ml /h un-

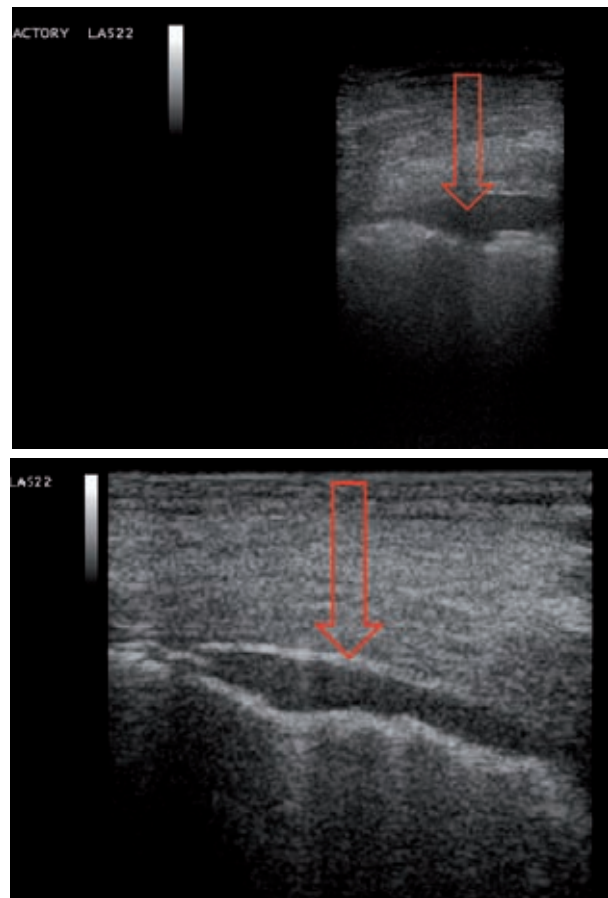


Fig. 5, 6 - Ultrasonographic pattern of parenchymal lung consolidation; hypoechoic subpleural focal images with or without pleural line gap, either isolated or multiple, appear as hypoechoic pleural-based focal images allowing ultrasound transmission, from which B-line-like artifacts arise, with confluent consolidations (“hepatization”).

Table 2

Aetiological classification of main causes of shock.

Low cardiac output	High cardiac output
Hypovolemic	Septic
Cardiogenic	Anaphylactic
Extra-cardiac obstructive	Neurogenic

Table 3

Diagnostic tools with comparison in rapidity of execution and availability referring to most common clinical abnormalities.

Condition	Test	Rapid	Available
<i>Hypovolemic</i>			
• Gastrointestinal bleeding	Upper: NGT Lower: DRE	+++	+++
	Endoscopic	+	+
• Peritoneal bleeding/ AAA	Focused-US	+++	+ / ++
	CE-CT	+ / ++	++
• CVP low	Focused-US	+++	+ / ++
<i>Cardiogenic</i>			
• ACS	ECG	+++	+++
	I-troponin	+	+++
	Copeptin*	+++	?
• Structural hearth disease	Focused-US	+++	+ / ++
• Rhythm disturbance	ECG	+++	+++
• Aortic dissection/ rupture	CE-CT	+ / ++	++
<i>Extra-cardiac obstructive</i>			
• Pneumothorax	RX	++	+++
	Focused-US	+++	+ / ++
• Pleural effusion	RX	++	+++
	Focused-US	+++	+ / ++
• Cardiac tamponade	Focused-US	+++	+ / ++
• Pulmonary embolism	CE-CT	+ / ++	++
	Focused-US**	+++	+ / ++
*Not validated yet. **Can exclude pulmonary embolism with emodinamical instability – useful for DVT.			

til normalization of blood pressure, we could assist to a progressive lost of inspiratory inferior vena cava collapse (CVP exstimated 15-20 mmHg) with appearance of right lung B-lines; so diuretic therapy was started and anemia (also due to emodilution) was corrected.

- In entire course of follow up no signs of left ventricular failure or pericardial effusion or free abdominal fluid were evident.
- Lung B-lines became less evident after four days from admis-

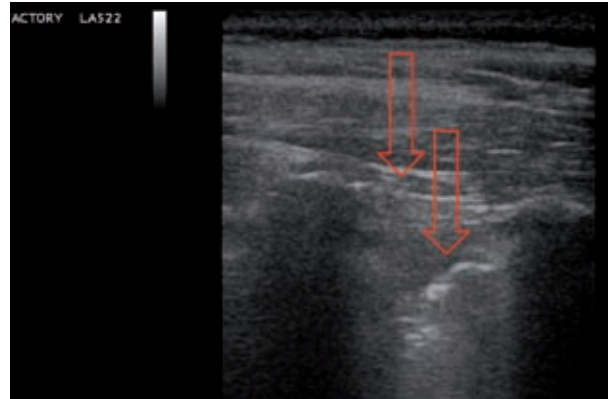


Fig. 7 - The presence of parenchymal disruption with localized pleural effusion.

sion and extinguish at right lung after six days and at left lung after eight days.

- Signs of alveolar consolidation begun to decrease after five days and were not relievable after eleven days, while pleural effusion was not reliable after seventeen days (outward follow up visit).

Our patient was treated furthermore with oxygen, broad-spectrum antibiotics, low-molecular weight heparin (after suspension of dicumarolic therapy) and diuretics, obtaining a clinical and radiological remission in two weeks.

With close ultrasonography monitoring it has been possible to “titrate” fluid and diuretic therapy according to haemodynamic conditions, in particular after three-five days of admission. Also use of anticoagulant therapy (low molecular weight heparin) was safer because of the faculty of exclude free blood loss at any time. Signs of alveolar consolidation became progressively less evident with course of antibiotic therapy(Figure 6), while resolution of “reactive” pleural effusion was slower.

Discussion

Rapid diagnosis and management of critical patients is strategic in the Emergency Department but hard in cases of unusual presentation or clinical abnormalities common to different pathologic conditions²⁻⁵. In our case report, careful anamnestic record, accurate physical examination and a few diagnostic tools – with the support of bedside-focused ultrasonography – allowed rapid diagnosis and effective treatment.

The reliability of bedside-focused ultrasonographic evaluation of the chest in the critical patient is demonstrated by the study in BLUE⁶.

Dyspnea with chest pain and hypotension generally indicates

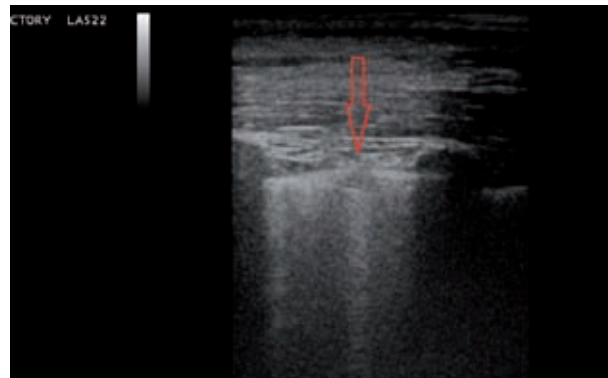


Fig. 8 - Ultrasound control shows size reduction of lesion.

shock due to low cardiac output (Table 2) and suggestive for hypovolemic (bleeding), cardiogenic (acute coronary syndrome or acute vascular injury), or extra-cardiac obstructive (occupation of pleural space, acute cardiac tamponade, massive pulmonary embolism) conditions.

While the use of a “pre-test probability” scoring system could rule out some pathological conditions (such as pulmonary embolism), the anamnestic recording of chest trauma and anticoagulant therapy associated with a normal ECG and the findings of moderate anemia and acute respiratory failure oriented our diagnostic pathway to a hypovolemic-post traumatic or pleuro-pulmonary acute condition (such as pneumothorax or emothorax or acute cardio-vascular rupture), despite a clinical cardiopulmonary examination without relevant pathologic findings (such as pleural effusion).

Rapidly performed bedside ultrasonography could easily exclude all pathological hypotheses listed above, thus confirming our diagnosis of ARDS secondary to pulmonary contusion⁷⁻⁹.

In the Table 3 we list the most appropriate diagnostic tools with comparison in rapidity of execution and availability according to our diagnostic hypothesis.

Nowadays, largely standardized step-by-step protocols and guidelines assist every Emergency Departments team, in order to “focus on” only some clinical and anatomic points of interest, and to make it simple to perform and very reproducible.

FASTCRASH procedure (Focused Assessment Sonography for Trauma, Cardiac failure, Respiratory failure, Acute abdomen and Shock), introduced a few years back, provides rapid clinical and ultrasonographic evaluation of critically ill patients and delivers rapid and reliable results in order to identify the main problems and quickly (less than ten minutes) refer the patient to the most appropriate diagnostic/therapeutic procedure^{2,9-11}.

Ultrasonographic examination of chest is very useful in an emergency approach of dyspnea/respiratory failure and often provides a more accurate diagnosis of acute cardio-pulmonary disorders compared to standard chest X-Rays; it may be performed at the patient's bed and repeated frequently during follow up, thus avoiding useless patient transport and radiological exposition.

With a “targeted approach”, recognition of pneumothorax, pleural effusion, parenchymal lung consolidation, diffuse interstitial infiltrates, pulmonary edema and other common conditions becomes very easy and accurate¹¹⁻¹³.

Conclusion

Lung contusion is a frequent clinical entity but often remains undiagnosed – especially upon first evaluation – because a diagnosis is unlikely with a visit and chest radiography^{5,7,8}. Although TC is the gold standard for lung contusion diagnosis, it is expensive and subjects the patient to the risks of transport, contrast agent side effects and radiation and is therefore non-viable for the serial monitoring of the patient. Various studies have demonstrated that chest ultrasonography can accurately detect lung contusion in blunt trauma victims: sensibility is 94.6%, specificity is 96.1%, positive and negative predictive values are 94.6% and 96.1% respectively and accuracy is 95%⁸.

In an emergency situation, a traumatized patient showing signs of shock and acute respiratory deficiency poses a challenge for an ER doctor whose quick and precise diagnosis is needed in order to ensure an effective treatment. Pneumothorax and pleural effusion are accessible to ultrasound; information obtained from lung, cardiac, venous and abdominal analysis provides a bedside visual approach to the critically ill^{9,14-16}; according to FASTCRASH method (Focused Abdominal Sonography for Trauma, Cardiac arrest / failure, Respiratory arrest / failure, Acute abdomen and Shock) which constitutes an important tool and

leads the intensivist to a more confident management and follow up of critically ill patients³.

In our case report, in compresence of thoracic traumatism and possible structural heart disease, it is difficult to distinguish cardiogenic responsibility of haemodynamic alteration and pulmonary direct injury from the responsibility of pain defense in the respiratory distress. Furthermore, in case of arterial hypotension associated, a distinction between hypovolemic form (iatrogenic or due to internal haemorrhage, especially in patient in anticoagulant therapy) and cardiogenic cause of hypotension itself became crucial.

Disclosures: all authors declare no conflict of interest.

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