Posttraumatic Pleural Empyema (PTPE) and Multidetector CT (MDCT) Findings.

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Abstract

Background: PTPE is a significant complication and the main cause for 2–10% of victims. MDCT is increasingly used. Our study is an analysis focused on the anatomy of pleura, principles behind fluid formation/reabsorption and an imaging approach to assessing pleural effusion and PTPE under-CT evacuation.
Material and methods: The study is conducted on eight (8) patients with PTPE at University Hospital of Trauma, University Hospital Centre "Mother Theresa", University Hospital " Shefqet Ndroqi" in Tirana, during the period January 2015 – June 2018, by using a MDCT of 128 slice – 64 detector – dual source, SIEMENS, German machine.
Results and conclusions: The frequency of post-traumatic pleural injuries with presence of Hydrothorax is 75.6% in total; second after that of Chest wall injuries (94.2%). Among the variable forms are reported Hemothorax – 17.4 % and Pneumothorax – 7.3 %. Empyema is rare – 2 %. MDCT is the most sensitive, specific and accurate imaging modality in the assessment of PTPE and management of patients:
- demonstrates the significant disorder in patients with normal initial radiographs,
- indicates changing of management in up to 20% of cases with abnormal initial radiographs,
- assists several micro-invasive procedures in order to prevents development of empyema,
- enables early prediction of respiratory compromise and limits the severe invasive interventions.

Keywords: Posttraumatic Pleural Injuries, Empyema, Multidetector CT

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Full Text

Introduction

Thoracic empyema, defined as collection of pus in the pleural space, has been recognized since the time of Hippocrates and historically has been associated with high mortality. The mortality rate from empyema thoracic remains high and it ranges between 6%–24%. A significant proportion of pleural space infection complicates community- or hospital-acquired pneumonia. However, a proportion of pleural space infection results from iatrogenic causes; it is also known that pleural infection may develop without pneumonia—so called primary empyema.

Physiology of pleural space

Simple PPE have characteristic biochemical and microbiological features namely: pH > 7.2, LDH < 1000 iu/l, Glucose > 2.2 mmol/l and no organisms in culture or gram stain. Pleural fluid amount is 0.1-0.2ml/Kg; <10ml in a 70 kg man. Normal rate of production 17ml/day

Maximal absorptive capacity into lymphatics 0.2-0.3ml/kg/hr

Content: Protein 1.5 gm/dl; rare macrophages, mesothelial cells and lymphocytes.

Main causes	**↑hydrostatic pressure ↓colloid osmotic pressure**	**Inflammation ↓ ↑vascular permeability**
Appearance	Clear	Cloudy
Specific gravity	< 1.012	> 1.012
Protein Content	< 2.5 g/dL	> 2.9 g/dL
Fluid Protein/ProteinSerum	> 0.5	< 0.5
SAGA=[Serum-Effusion]alb	> 1.2 g/dL	< 1.2 g/dL
Fluid LDH max. for serum	< 0.6 or < $^{2}/_{3}$	> 0.6 or > $^{2}/_{3}$
Cholesterol content	< 45 mg/dL	> 45 mg/dL

***Table 1** : biochemical and microbiological features*

"Empyema thoracis" causes and risk factors

Ventilated and immobilized patients, pulmonary contusion and onset of pneumonia, direct infection from penetration, secondary infection from abdominal injuries and iatrogenic infection during chest tube insertion. Thoracic empyema is most common in male then in female, in age 50 years.
Alcoholism, HIV, drug usage and pre-existing lung disease are giant risk for empyema.

Pathophysiology

In most series of patients with community acquired empyema, aerobic bacteria predominate. These include Streptococcus pneumoniae and Staphylococcus aureus. Aerobic organisms also include Gram negative bacteria such as Escherichia coli, Hemophilus influenza and Klebsiella pneumoniae. Organization of empyema is a matter of time.

Stage	Pathology	Evolution
1. Exudative/Acute	Protein-rich sterile fluid Normal glucose & pH Low cellular count	0 - 2 weeks
2. Fibrinopurulent	Bacterial invasion Polymorphs ,Activated coagulation & fibroblastic activity ,↓ glucose & pH	1 - 6 weeks
3.Organized phase	Thick pus Thick inelastic peel over pleura	5 weeks

Table 2: *pathophysiology*

Diagnosis

Most patient with aerobic bacterial pneumonia and PPT have an acute onset of chest pain (60%), cough (70 %), fever (80%) and sputum production. Symptoms are often more indolent in patients with anaerobic infection. Dyspnea, Tachypnea, Leukocytosis, increase of CRP.

Radiography

Pleural-based opacity in non-dependent regions if loculation occurs. "D-sign" - loculated pleural fluid bulging out from the chest wall. Elliptical shape with well-delimited borders and conforms to the chest wall. Loculated collection of pleural fluid with or without gas pockets ***(Figures 1,2).***

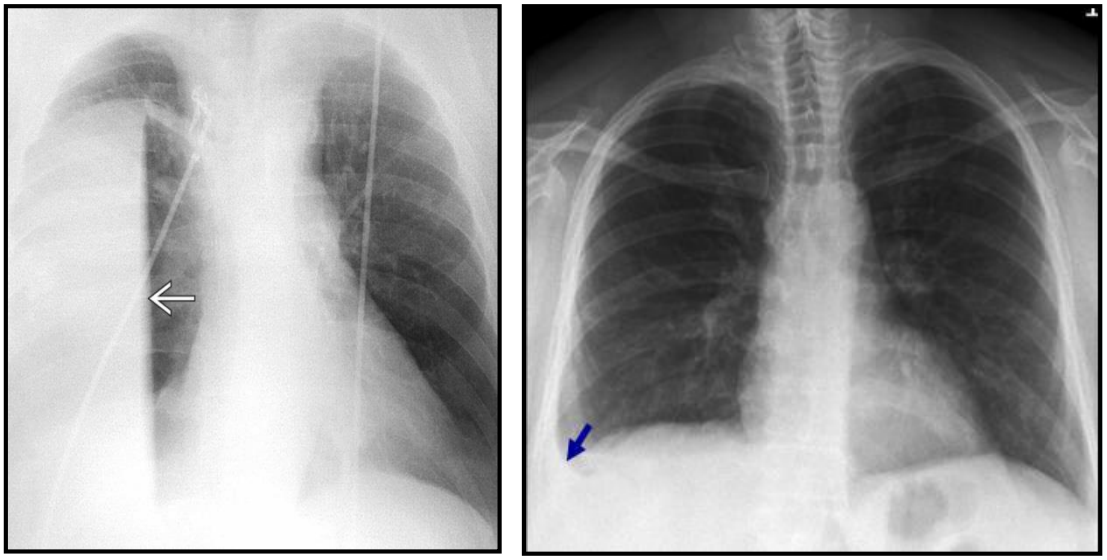


Figure 1, 2

MRI & Ultrasonography

Simple fluid, nonseptated, Complex collections, - internal echoes & septations, Shadow – gas pocket ***(Figures 3, 4).***

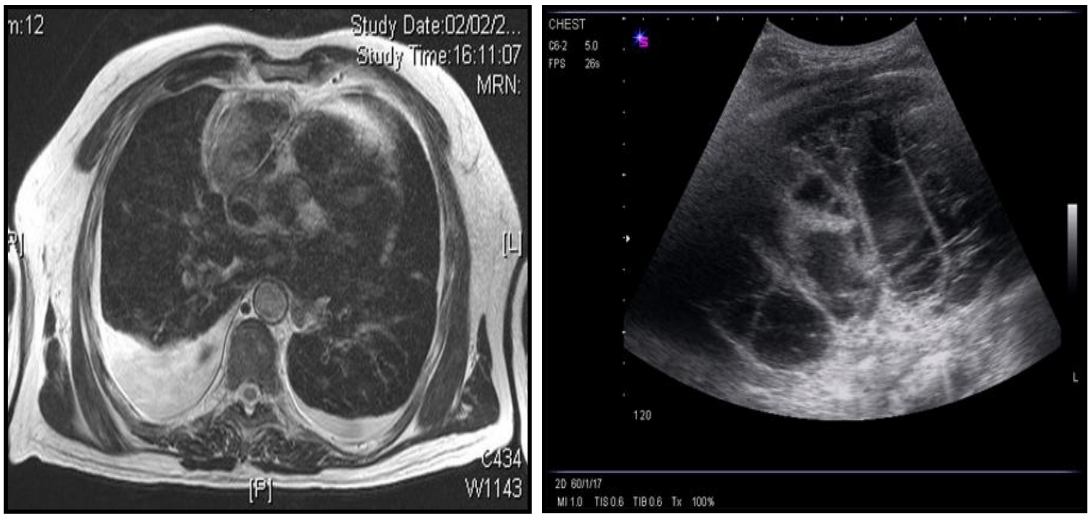


Figure 3, 4

Imaging – CT

Stage I

Air-space disease associates with pleural effusion. Lenticular fluid collection ***(Figure 5, 6).***

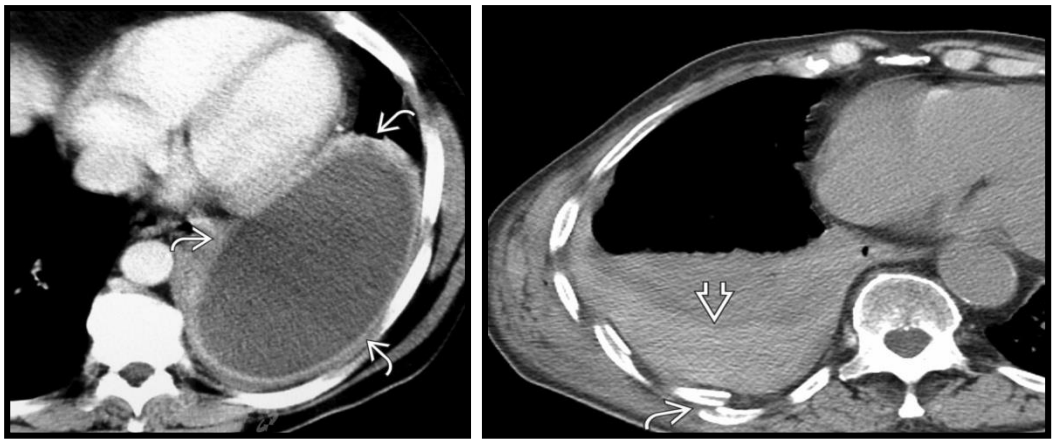


Figure 5, 6

Stage II

Fixed non mobile pleural *"mass"*.

Pleural fluid in atypical location.

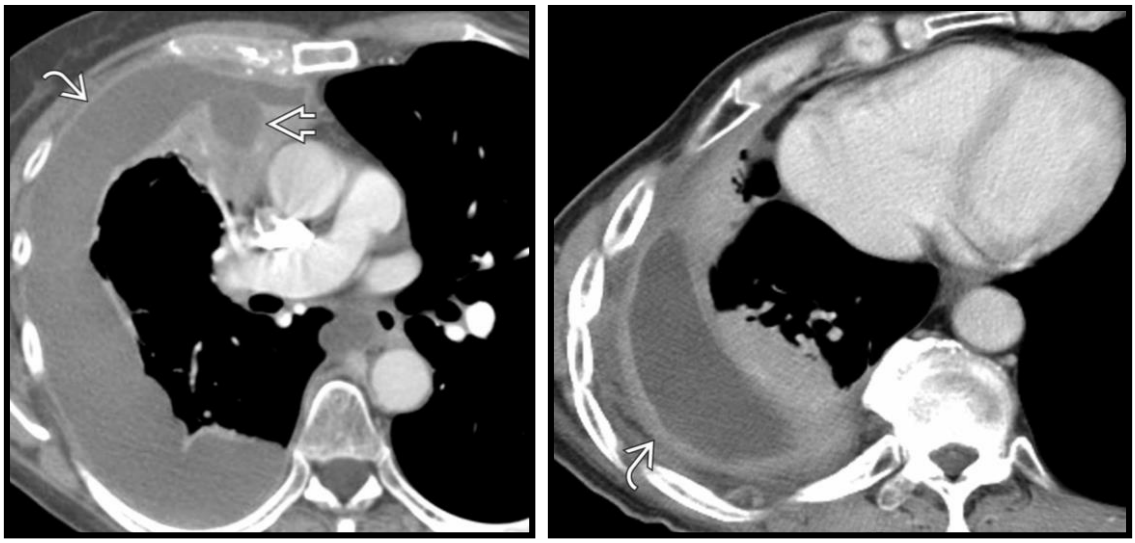
Thickening and/or enhancement of parietal pleura.

Increased thickness and attenuation of costovertebral subpleural fat.

"Split pleural sign" – enhancing, thickened visceral and parietal pleura layers separated by an intervening layer of low attenuation fluid ***(Figures 7, 8).***

Loculated gas bubbles with complicating bronchopleural fistula ***(Figure 9.)***

Empyema necessitates with infected pleural fluid collection extend into chest wall. ***(Figure 10).***



Figures 7, 8*: Effusion; lobular borders, Pleural surfaces enhancement, Hemothorax, Atelectasis)*

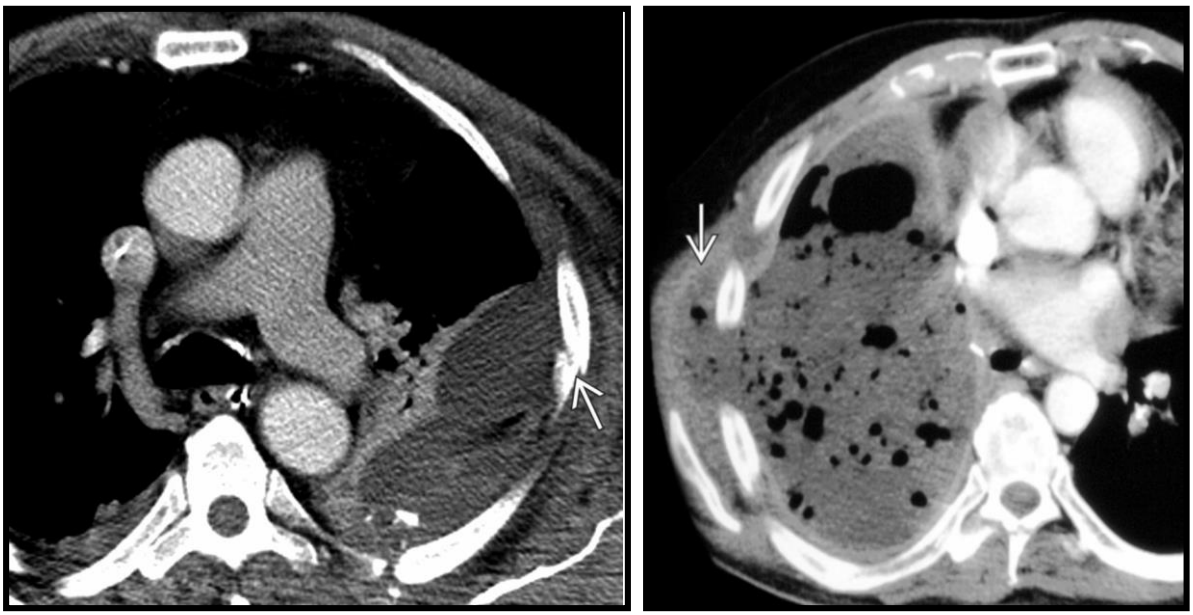


Figure 9*: Multiple rib fractures - pleural collection (40 HU) Gas within pleural space;*
Figure 10: *Consider empyema necessitates in patients with empyema & adjacent chest wall abnormality*

Stage III

Pleural rind or peel with calcific pleuritic.

Entrapment of the lung.

Chest wall invasion or violation.

Management

- Pus Elimination
- Lung re-expansion
- Chest wall and diaphragm mobility restoration
- Pulmonary function improvement
- Complications & chronicity Elimination ***(Figure 11).***

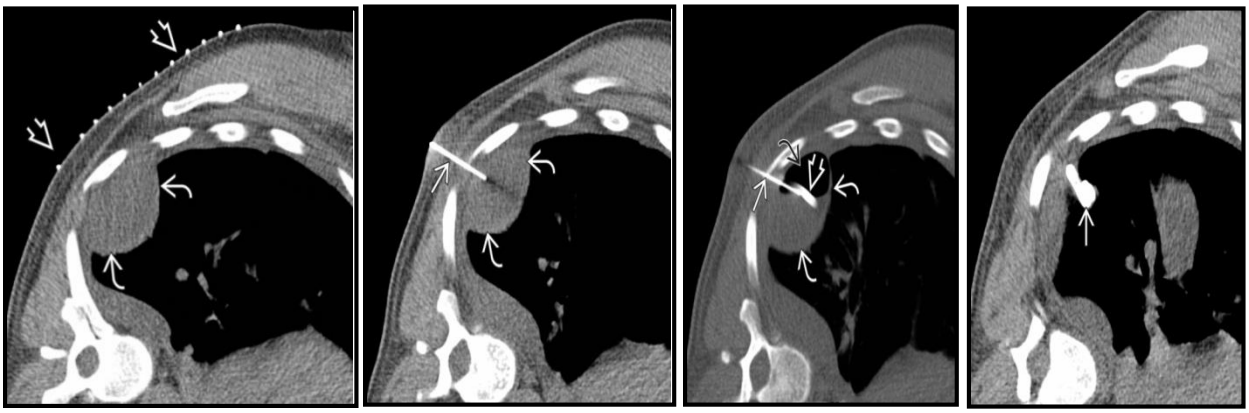


Figure 11

Antibiotic Therapy

Penicillin-Clindamycin-Ceftriazone-Vancomycin-Metronidazole-Ciprofloxacin

Fibrinolytics

Streptokinase & Urokinase activate plasmin through cleavage of plasminogen which initiate degradation of fibrin, reduce viscosity of fluid, dissolve loculations, dissolve peel.

Chronic open drainage *(Figure 12)*

Pulmonary decortications

After 6 months, pleura remains thickened and the patient's PFTs is significantly reduced to limit activities, decortication should be considered ***(Figure 13).***

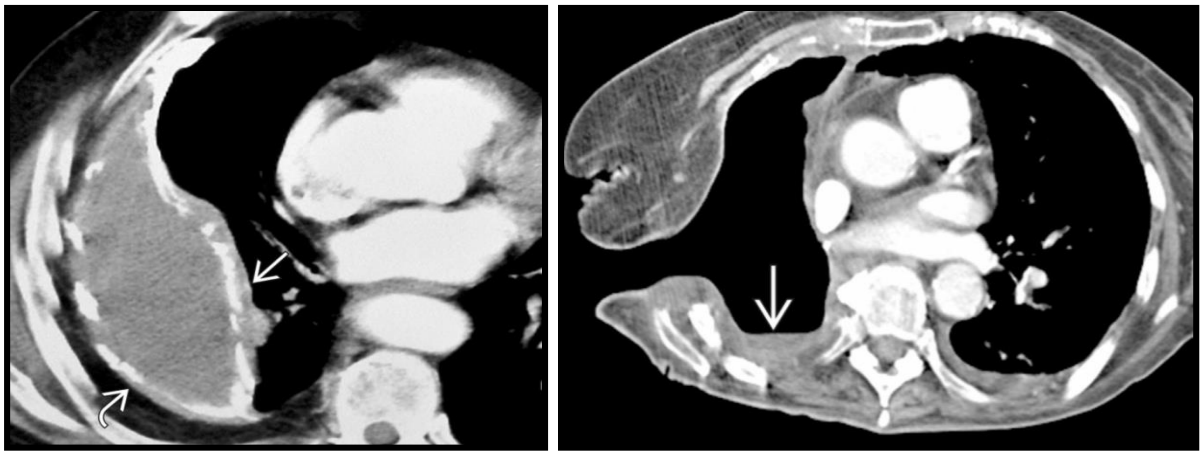


***Figure 12** (on the left), **13** (on the right)*

Conclusion

PTET remains a significant clinical problem that accounts for 2 up to 10% of trauma victims. Factors of ET are mostly preventable. Primary feature is a retained hematoma which needs to be evacuated (ICD/VATS) within 5 days of trauma**.**

ET management is prolonged with high risk of morbidity and mortality.

Best imaging tool: Chest radiography is best initial study, CT often needed to plan intervention

Pleural injuries MDCT protocol:

CT Scanning with intravenous contrast material at optimum arterial opacification (sometime not essential)

Routine coronal and sagittal reformations

Volumetric MIP/CTA reformations when indicated

5. doi10.1016/j chest. 2016.08.653.

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