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Case Report
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SHOCK with MODS: A Case that Changed Everything to Me

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Abstract

For me title of this article is such that as this case report deals with case of my father who deceased few months ago, and his final diagnosis was Shock with Mods. Firstly, it all started with right thoracic mid back pain and then radiated too anterior thoracic. These symptoms lasted for about 15 days and were on–off, while treatment was on with analgesics and muscle relaxants. Later as whole-body checkup was done where it was found the patient FBS and RBS Elevated thus was considered as *de novo* DM, in addition chest CT was done when abnormalities were observed in pulmonary function test, through CT-chest: Loculated collection with thin internal septations noted in right pleural space, thus cardiothoracic surgeon suspecting empyema and RT pleural effusion (~200CC) opted for VATS–decortication. Although one day after the surgery patient developed emphysema leading to panic attack causing hemodynamic instability. Consequently, leading to shock which steadily lead to multi organ dysfunction in the end to death. On the whole through this case report, I wanted to give detailed analysis preoperative treatment initially followed by postoperative till death.

Keywords: Shock; VATS (Video Assisted Thoracoscopic Surgery); MODS (Multiorgan Dysfunction Syndrome); Cardiogenic shock; Emphysema; Empyema

Introduction

A lack of blood flow to the body can result in shock, a potentially fatal condition. The cells and organs need blood flow in order to receive enough oxygen and nutrients to function properly. As a result, many organs may sustain harm. Shock must be treated right away since it might quickly develop worse. One in 5 people who are in shock will pass away as a result [1].

Inadequate organ and peripheral tissue perfusion are the definition of shock, and it is classified according to its origin as either Cardiogenic shock (due to heart problems), Hypovolemic shock (caused by too little blood volume), Anaphylactic shock (caused by allergic reaction), Septic shock (due to infections), Neurogenic shock (caused by damage to the nervous system) [2].

The most frequent type of shock among patients hospitalized to the critical care unit is septic shock, a type of distributive shock; it is followed by cardiogenic and hypovolemic shock; obstructive shock is uncommon. A study of 1,600 patients with undifferentiated shock, for instance, revealed that septic shock occurred in 62% of cases, cardiogenic shock in 16% of cases, hypovolemic shock in 16% of cases, other types of distributive shock in 4% of cases (such as neurogenic shock, anaphylaxis), and obstructive shock in 2% of cases [3].

A sudden, widespread decrease in effective tissue perfusion is known as shock. Shock causes an imbalance between the supply and demand for oxygen, anaerobic metabolism, lactic acidosis, cellular and organ dysfunction, metabolic abnormalities and, if it lasts for a long time, irreparable harm and death. With changes in hemodynamics, oxygenation, fluid compartment composition, and a number of mediators, the pathophysiologic events in the many forms of shock are unique and complex [4].

Blood pressure is incredibly low in someone who is shocked. The following signs may be present depending on the precise origin and kind of shock.

Agitation or restlessness caused by anxiety bluish lips, fingernails, and skin a chest ache confusion. faintness, light-headedness, or vertigo pale, chilly, or clammy skin lack of urine production, excessive perspiration, wet skin, rapid yet feeble heartbeat sluggish breathing, being unresponsive (unresponsive) [5].

A common complication of trauma is Multiple Organ Dysfunction Syndrome (MODS).

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Hypovolemic shock, huge volume replacement, length of resuscitation prior to admission to the hospital, systemic inflammatory response syndrome, infection, and sepsis are risk factors for MODS following trauma. The priority in MODS prevention is tissue hypoxia prevention [6].

The enhancement of tissue oxygenation and perfusion by hemodynamic optimization is crucial. Patients can survive trauma and lower their risk of systemic problems with the use of nutritional assistance, antimicrobial prophylaxis, pain management, sedation, and other treatment modalities [7].

Case Report

Subjective evidence

A male patient of age 54 years was admitted to the hospital for the procedure of VATS-decortication as suggested by cardio thoracic surgeon with chief complaints of Rt side infra-axillary/hypochondriac pain, initially the patient was having Rt Mid back pain in the last 15 days later developed radiating pain near Rt hypochondriac region. Symptoms were on–off and were on treatment with muscle relaxants and analgesics.

On examination patient was conscious coherent and obeying commands, Cvs-S1s2+; all peripheral pulses felt, Rs-Bae: No added sounds, P/A- Soft; Bs+; no tenderness, CNS-NFND: GCS- 15/15, BP 120/60 mmHg, SPO_2 -85 LI min, Respirate 22 min, Heart rate 70 bpm, Temp- 98.6 (F). Past medical history: *De novo* diabetes mellitus, no significant family Hx and social Hx, Blood group 'B' positive. Initial diagnosis: S/O Rt loculated empyema.

Objective evidence

Preoperative lab reports: Through Figures 1.2(A) & 1.2(B): CT chest plain with contrast had provided evidence of loculated collection with thin internal septations noted in right pleural space. Minimal free fluid collection in left pleural space. Thus S/O Rt loculated pleural effusion with internal septations (~200cc), Lt minimal effusion.

Table 1: Surgical profile test-1 (Provisional diagnosis: Right loculated empyema plan: Vats procedure)

TEST	Value	Impression
Blood Glucose Test		
HBA1c	11.6	Diabetic (>6.5)
Fasting Blood Glucose	296 mg/dl	Diabetic (>126 mg/dl)
Infectious Disease Tests		
HBsAG	0.19	Non-Reactive (<1)
HIV	0.13	Non-Reactive (<1)
HCV	0.08	Non-Reactive (<1)
Serum Electrolytes		
Sodium	129	Below Normal (136-145 mmol)
Potassium	4.5	Normal (3.5-5.1)
Chloride	90	Below Normal (98-107 mmol)
CBP & Peripheral smear Test		
RBC	4.1 million	Normocytic (4.5-5.5 mill)
WBC	7.8thousand	Normocytic (4500-10000)
Platelet	4.7 Lakhs	Thrombocytosis (1.5-4.5 Lakhs)
Hb	10 gm%	Below Normal (1-17 gm%)
PCV	33 Vol%	Below Normal (40-50 Vol%)

Table 1.2: Surgical profile test-2 (where other test such as LFT, RFT were performed to observe abnormalities, values.)

TEST	Value	Impression
Renal Function Test		
Sr. Creatinine	0.64 mg/dl	Normal (0.7-1.2)
Blood Urea Nitrogen	28 mg/dl	Normal (16.6-48.5)
Liver Function Test		
Total Bilirubin	0.7 mg	Normal (<1.2)
Direct Bilirubin	0.48 mg	Above Normal (<0.2)
ALT	25 U/L	Normal (< 41)
AST	14 U/L	Normal (< 40)
ALP	133 U/L	Above Normal (4.5-5.5)
Total Protein	6.6g	Normal (6-8)
Albumin	3.3g	Normal (3-5)
Globulin	3.2g	Normal (2-4)
Lipid Profile		
Total Cholesterol	159 mg/dl	Desirable (<200)
HDL	26 mg/dl	Low(<40 mg)
LDL	112 mg/dl	Desirable (100-129)
VLDL	24 mg/dl	Desirable (2-30)
Triglycerides	120 mg/dl	Low (<150)

Table 1.3: Surgical profile test 3 (Clotting factors and 2D echo was done in order to get cardio clearance before surgery.)

TEST	Value	Impression
Clotting Factors		
Prothrombin Time	17.1 sec	Above Normal (9.5-13.5)
Fibrinogen	678 mg/dl	Above Normal (220-496)
Activated Partial Thromboplastin	28.7 sec	Normal (25-35)
2DEcho		
No RWMA Trivial MR/TR, Mild PAH Grade –I LV Diastolic dysfunction No Clots/PE		

Post operative lab report: Consists of Table-2.1, Table-2.2, Table-2.3, Table-2.4, Figure 2.1 Chest X –Ray, Figure-2.2: Histo Pathology Report and Figure-2.3: ECG

Management: The patient was admitted as mentioned in the subjective evidence with certain symptoms and was considered for VATS after cardio thoracic surgeon opinion suspecting empyema.

On Day-1 (26th August) the patient's vital were to be monitored and as his FBS level were elevated as shown in Table-1.1 as a result insulin had been prescribed to reduce the glucose levels as surgery was supposed to take place next day. Twenty units was given twice once before food and after food in an interval of 3h to 4 h that night. Injection Piptaz 4.5 gm IV TID was given as prophylactic medication as there was slight elevation in body temperature.

And then Day-2 (27th August) vitals stable, provisional diagnosis: Right loculated empyema.

Plan: Vats procedure today, Hba1c- 11.6 Sodium 129, Hb- 10 gm% as seen in Table 1.1, whereas other test such as LFT, RFT were performed to observe abnormalities, values in Table 1.2, clotting factors and 2D echo was done in order to get cardio clearance before surgery impression can be noticed in Table 1.3.

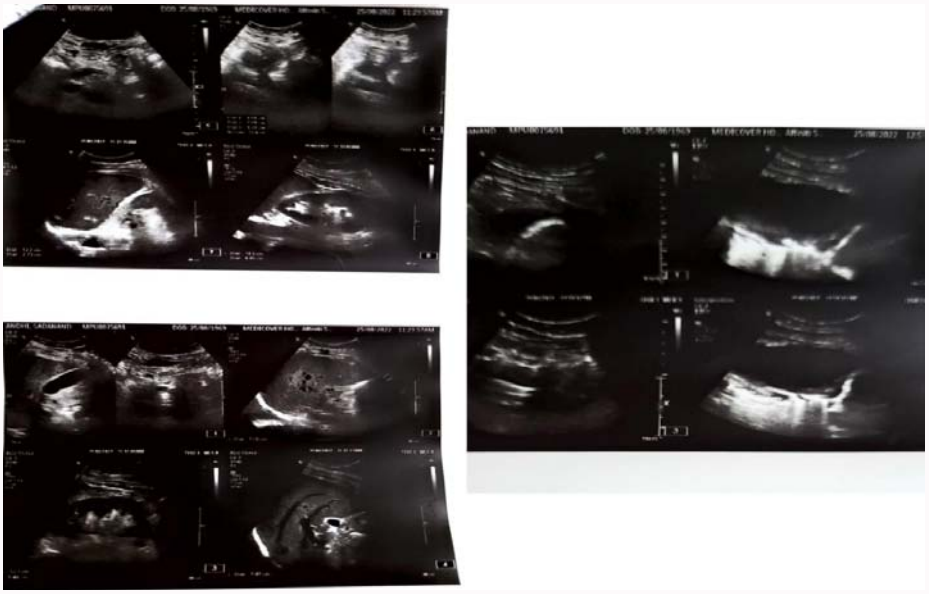

Figure 1.1: USG Abdomen: Shows impression of grade I fatty liver, mild splenomegaly (12.7 cm × 7.7 cm). There is no evidence of obvious retroperitoneal adenopathy /ascites.

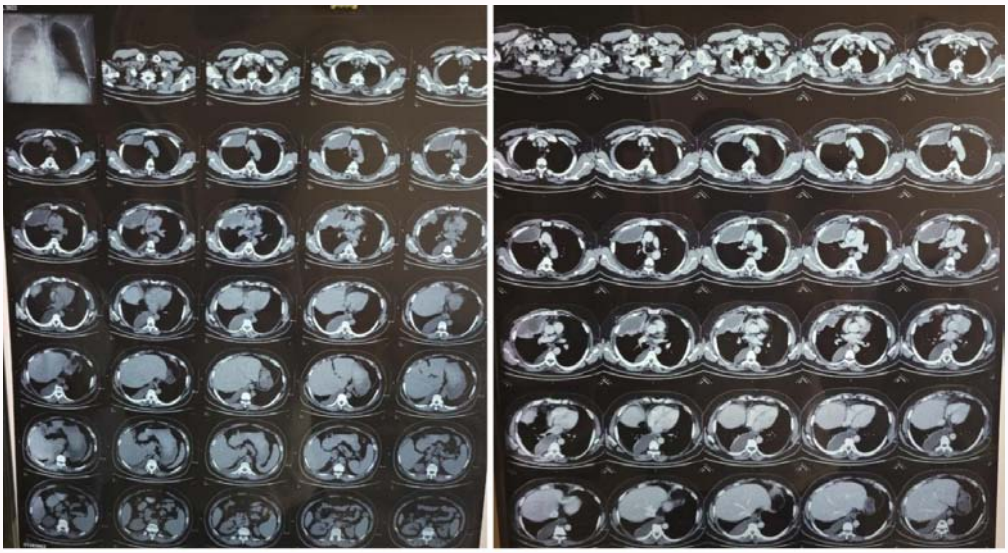

Figure 1.2 (A): CT Chest plain with contrast.

Patient can be taken up for surgery with high risk of procedure and post procedure. Cardiac clearance given by cardiologist case can be taken for procedure with mild risk.

Procedure/Surgery Details

Two ports inserted 7th ICS in posterior mid axillary line and 5th ICS in anterior axillary line, fluid drained, adhesions dissected, visceral pleura peeled off and send for histopathology examination, biopsy taken from suspicious Rt upper lobe lesion, hemostasis achieved, drain placed -two, wound closed in layers.

Surgery Findings

Adhesion present, loculated pleural collection blood stained suspicious of malignancy, thick visceral pleura.

Status of the patient after surgery was stable. Inj Piptaz 4.5 gm IV BD; Inj Tramadol 50 mg IV BD; Inj Pcm 1 gm IV TID; Inj Zofer 2 mg IV BD; Inj Pantocid 40 mg IV BD, Tab Plumoclear PO BD, Tab Azee 500 mg PO BD. Neb Duolin and Budecort 1 Resp TID, 1 PRBC @ 5 pm were planned for post operative care.

Next Day Day-3 (28th August) morning vitals were stable, while patient on 02 support with C/O Cough+, Edema+. Then after everything changed drastically as patient developed "Surgical Emphysema" even we can see those changes in report such as RFT before and after of surgical emphysema, in Table 2.1. As matter of fact there were variation in LFT, Sr electrolytes and CBP etc. when compared Table 2.1 and Table 2.2 with Table 1.1 and Table 1.2. In addition, PAL (Persistent Air Leak) in lungs was observed through chest X-ray Figure 2.1.

Above all surgical emphysema leads to "Panic attack "with certain symptoms like irregular heartbeats, dry mouth, breathlessness, sweating etc. Patient had become sudden unresponsiveness, around

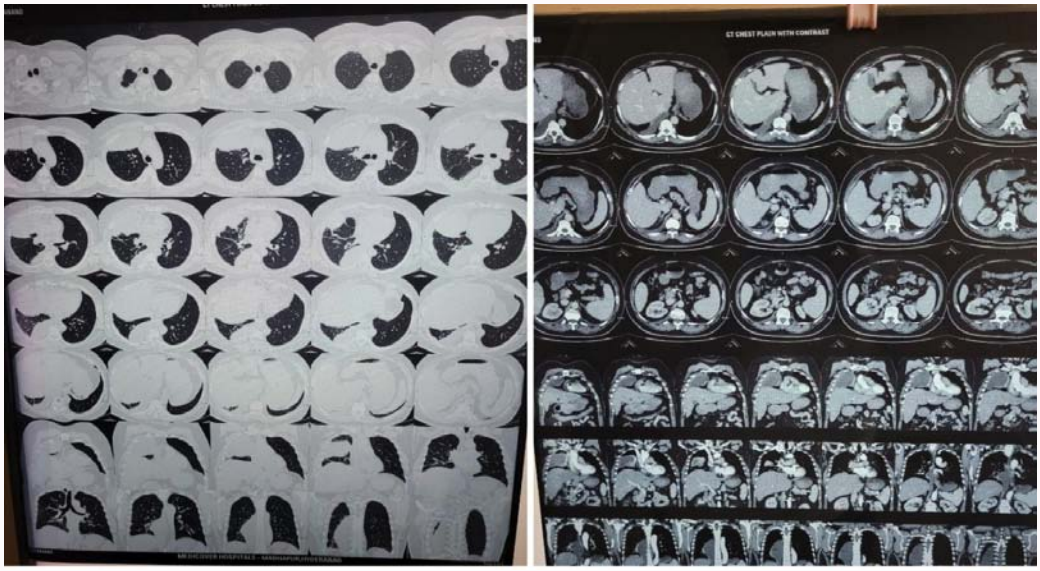

Figure 1.2 (B): CT chest plain with contrast.

4.40 pm, thus was intubated in view of unresponsiveness, and moreover had bradycardia which improved on dose of Inj Atropine.

Post-intubation, patient was hemodynamically unstable (Bp<100/60 mmHg; HR<60 bpm and MAP<60 mmHg), Rt femoral line inserted, ABG taken S/O of metabolic and respiratory acidosis. Patient connected to ventilator on PCV. Ion-tropic agents started with infusions Dobu 10 ml/h; Noradrenaline 25 ml; Adrenaline 13 ml/h; NAHCO3 25 ml/h; fentanyl 100 mcg/kg/h urine output decreased Lasix infusion started. Vitals, urine production, and hemodialysis, if necessary, were all scheduled for monitoring.

Same treatment was continued as of previous day with addition of Neb Mucomix 1 Amp TID, Inj Mucomix 1.2 gm IV BD, Inj Meropenem 1 gm IV TID, Inj MGSO4 2 gm IV BD, Inj Emeset 4 mg IV BD, Inj Dexa 8 mg IV BD. Stats Inj Lasix 20 mg @ 2 pm, Inj Adre 4 mg IV @ 5 pm, Inj Dexa 2 mg @ 7 pm, Inj Lasix 40 mg 7 pm, Inj cort 100 mg 6:30 pm, Inj Calcium 500 mg @ 6:20 pm, Inj KCL 20 mcg @ 6 pm.

On Day-4 (29th August), Vitals PR: 105, BP: 117/52 mmHg MAP: 70 mmHg, SPO_2; 96, Urine output; NIL.; Myocardial infarction @ 5:30 am, arrhythmia stabilized by cardio version done (150J) as noticed in Figure 2.3. **Diagnosis:** AKI, Sepsis; K/C/O DM. Patient is on mechanical ventilator on PCV, mode sedated and paralyzed, CRRT 12 h done extended 6 h, Procal raised, Creat 1.5 mg/dl; Urine output NIL. Persistently low PO_2 with raised PCO_2, high on inotropes, Echo-No RWMA; poor Echo window, Dilated RA/RV etc.

Infusions, Inj Dobu 500 mg 4 ml/h, Inj Norad 16 mg 18 ml/h, Inj Adre 8 mg 6 ml/h, Inj Vaso 80 u 1.2 ml/h, Inj Hydrocort 10 mg/h, IVF 75 ml/h, drains minimal, air leak present, continued hemodialysis. Continued same treatment with additions Inj Polymixin B 7.5 Lac unit IV BD, Inj Heparin 2500 SC TID, Stats Tab Digoxin 500 mcg @ 6 am; 12 pm; 6pm, Inj Vancomycin IV @ 6 am. Medication stopped. Inj Piptaz 4.5 gm IV BD, Inj Tramadol 50 mg IV BD, Inj Dexa 8 mg IV BD, Tab Plumoclear PO BD and Tab Azee 500 mg PO BD. Ryles tube feeds to give kabipro 2 scoops in 100 ml water and ensure dm 3 scoops in 100 ml water.

Meanwhile on Day-5 (30th August) O/E vitals BP-116/68, RR-23/min, Temp-98.3 F*, HR-99 bpm, SPO_2 – 98%. Sensorium sedated, Severe hemodynamic instability S/O septic shock? Obstructive? Patient is on mechanical ventilator on PCV, anuric metabolic acidosis- CRRT 12 h done extended 6 h. 2D echo showed RA/RV dilated, Grade-1 LV dysfunction as observed in Table 2.3, where PIF RATIO- 160, MAP 60 mmHg caused requirement for very high vasopressor support. Hypoglycemia requiring 25D infusion. Drains minimal and persistent air leak present.

Infusions given Inj Dobu 5 mcg/kg, Inj Norad 16 mg 30 ml/h, Inj Adre 8 mg 6 ml/h, Inj Vaso 40 IU 2.4 ml/h, Inj Hydrocort 10 mg/h, Inj Fentanyl 70 mcg/h. Same treatment was continued with additions Inj Targocid 40 mg IV BD and Inj Digoxin 0.25 mg IV OD. Medications stopped none Inj Heparin 2500 u SC TID altered instead with dosage increased to 5000 u. Stats Inj Dobu 0.2 mg/h till 2 ml, Inj Calcium 500 mg IV, Inj Heparin 100 u/4 h infusion and 2 PRBC transfusion done.

However, on Day-6 (31st August) there was "guarded prognosis" and was similar to previous day treatment continued similar with addition of Inj optineuron IV OD, Inj Thaimine 100 mg IV TID and Inj Vit-C 500 mg IV BD.

Patient was receiving multiple vasopressors for support, and despite ongoing RRT, metabolic acidosis persisted. Patient also had sudden bradycardia followed by asystole.

The patient's son was informed of ongoing CPR efforts, and CPR was administered for 30 min in accordance with ACLS protocol. Despite prolonged CPR, the patient's return of spontaneous circulation was unsuccessful, and at 6:38 PM on August 31st, 2022, death was pronounced.

Assessment

Day-1

No therapeutic duplication, drug-drug interaction or drugs-disease interaction.

Day-2

No therapeutic duplication, found 8 possible drugs–drug interactions, no drugs disease interaction.

Table 2.1: Day wise lab report-1 (even we can see those changes in report such as RFT before and after of "surgical emphysema".)

TEST	Day-3		Day-4	Day-5	Day-6
CBP& Peripheral Smear					
Hb (13-17gm%)	9.8 gm%		8.7 gm%	10.2 gm%	10 gm%
RBC (4.5-5.5 million cell/cumm)	3.4 million Normocytic Normochromic		3.2 million Normocytic Normochromic	3.3 million Normocytic Normochromic	3.5 million Normocytic Normochromic
WBC (4000-10000 cells/cumm)	9000 Within Limits		8900 Within Limits	8600 Within Limits	8400 Within Limits
Platelet (1.5-4.5 Lakh/cumm)	5.9 lakhs Thrombocytosis		3.6 lakhs Adequate	2.2 lakhs Adequate	1.6 lakhs Adequate
PCV (40-50 Vol%)	32%		30%	34%	33%
RCW (11.6-14%)	12.9%		13%	14%	13.6%
Clotting Factors					
PT (9.5-13.5 sec)	----------		41.5 sec	----------	32.6 sec
APTP (25-30 sec)	----------		----------	----------	51.9 sec
Renal Function Test					
Sr. Creatinine (0.7-1.2 mg/dl)	0.75 mg/dl	1.52 mg/dl	2.18 mg/dl	----------	2.06 mg/dl
Blood Urea Nitrogen (16.6-48.5 mg/dl)	37 mg/dl	54 mg/dl	82 mg/dl	----------	60 mg/dl

Table 2.2: Day wise lab reports-2 (variation in LFT, and Sr electrolytes)

TEST	Day-4	Day-6
Liver Function Test		
Total Bilirubin (<1.2 mg/dl)	1.5 mg/dl	1.69 mg/dl
Direct Bilirubin (<0.28 mg/dl)	1.45 mg/dl	1.68 mg/dl
ALT (<40 U/L)	854 U/L	2681 U/L
AST (<40 U/L)	5 U/L	2696 U/L
ALP (40-129 U/L)	158 U/L	245 U/L
Total Protein (6-8 g)	4.8 g	4.3 g
Albumin (3.5-5.2 g)	2.4 g	2.1 g
Globulin (2.5-3.5 g)	2.3 g	2.2 g
Serum Electrolytes		
Sodium (136-145 mmol/L)	137 mmol/L	131 mmol/L
Potassium (3.5-5.1 mmol/L)	5.4 mmol/L	6 mmol/L
Chloride (98-107 mmol/L)	96 mmol/L	94mmol/L

Table 2.3: Day wise lab reports-3 (2D echo showed RA/RV dilated, grade-1 LV dysfunction as observed. Elevated levels of serum Mg,P, Procalcitonin and CRP)

TEST	Value	Day Performed
Sr. Magnesium (1.6-2.6 mg/dl)	2.3 mg/dl	Day-6
Sr. Calcium (1.15-1.32 mg/dl)	1.18 mg/dl	
Sr. Phosphorous (2.5-4.5 mg/dl)	7.5 mg/dl	
Procalcitonin (>5 high risk for sepsis)	26.9	Day-4
CRP (<5 mg/L)	287.3 mg/L	Day-3
2D echo		
No RWMA Trivial MR/TR, Mild PAH Grade –I LV Diastolic dysfunction No Clots/PE		Day-2
Poor Echo No RWMA Dilated RA&RV Mild TR, Mild PAH IVC Dilated Grade-I LV Diastolic dysfunction No Clots/PE		Day-6

Serious

- (Azithromycin + Ondansetron {Causing bradycardia})

Monitor closely

- Pantoprazole + Acebrophylline {Toxicity of Acebrophylline}
- Azithromycin + Pipercillin {Reduce efficacy of Pipercillin}
- Tramadol + Levo salbutamol {Reduced sedation}

Day-3

There are 5 therapeutic duplications, found 54 possible Drug-drug interaction, 3 drugs –disease interaction

Serious

- Fenatyl + Tramadol –synergistic effect {increase Sedation, Negative effects on cardio hemodynamics, Reduce Blood Pressure, Coma, etc.}
- Azithromycin + Ondansetron {Causing bradycardia}

Monitor closely

- Fentanyl + furosemide {Reduced efficacy of Furosemide}
- Dobutamine + furosemide {Reduce Sr. potassium levels}
- Adrenaline + Noradrenaline- Additive effect {increase BP}
- Noradrenaline + Furosemide {Increase Heart rate, Reduce Sr. potassium levels}
- Adrenaline + Azithromycin {Increase QT interval}

Day-4

There are 2 therapeutic duplications, found 30 possible Drug-drug interaction, 1 drug -disease interaction

Serious

- Pantoprazole + Digoxin {Increase Gastric pH, Increase, toxicity of digoxin}
- Hydrocortisone + Heparin {Reduced efficacy of Heparin}

Table 2.4: Day wise lab reports 4 (Biopsy-Pleural fluid and culture sensitivity tests).

Culture Sensitivity	
Parameter	**Result Values**
Pleural Fluid for Culture and Sensitivity (Method: Vitck Id & Ast Incubation At 37°C.)	Report: No Bacterial Growth Sen in Culture After 48 h of Aerobic
24 h Aerobic Blood Culture (Bact/Alert) (Method: Vitck 1d & Ast Incubation At 37°C.)	Site Blood: (Right femoral ar) Report: No bacterial growth seen in culture after 24 h of aerobic Note: 48 hours report to follow.
24 h Anaerobic Blood Culture Bact/Alert) No Growth (Method: Vitek 1d & Ast Incubation At 37°C.)	Site Blood: (Right Cvc) Report: No Bacterial Growth Seen in Culture After 24 h of Anaerobic Note: 48 h Report to Follow.
Modified Afb (Method: 2n Stain)	Result: Negative
48 h Aerobic Blood Culture (Bact/Alert) (Method: Vinck Id & Ast Incubation At 37°C.)	Site Blood :(Right femoral ar) Report: No bacterial growth seen in culture after 4 h of aerobe. Note: 5 days report to follow.
48 h Anaerobic Blood Culture (Bact/Alert) (Method: Vitek Id & Ast)	Site Blood: (Right femoral ar) Report: No bacterial growth seen in culture after 48 h of anaerobic Incubation at 37°C. 5 days report to follow.
Grams Stain	
Specimen Type: Pleural Fluid (Method: Vitek Id & Ast)	Report: Plenty of polymorphs and no microorganisms see.
Pleural Fluid for Afb	
Specimen Type: Pleural Fluid (Method Microscopy)	Report: No acid-fast bacilli seen.
Pleural Fluid for Malignant Cells	
Cytology No.	C367/2022
Specimen	Pleural fluid
Gross Appearance	Received 7 ml of reddish color turbid fluid 1-H &E 1-Leishman
Microscopic Examination	Smears show plenty of neutrophils in a hemorrhagic background. No atypical cells in the smears studied. Pleural fluid, cytology
Impression	Suppurative inflammation with haemorrhage No atypical cells seen in the smears studied.

Monitor closely

- Meropenem + Digioxin {Effects intestinal flora}
- Dobutamine + Adrenaline {Reduced sedation, Reduced Sr. potassium levels}
- Adrenaline + Noradrenaline- Additive effect {increase BP}

Day-5

There are 3 therapeutic duplications, found 37 possible Drug-drug interactions, 1 drug –disease interaction

Serious

- Pantoprazole + Digoxin {Increase Gastric pH, Increase, toxicity of digoxin}
- Digoxin + Calcium-gluconate-Synergistic effect {Increase efficacy of Digoxin}

Monitor closely

- Ticolplanin + Digoxin {Increase Digoxin Efficacy}
- Teicoplanin + Vancomycin {Increase Nephrotoxicity}
- Digoxin + Noradrenaline/Adrenaline {Reduces Sr. potassium}

Day-6

There are 3 therapeutic duplications, found 35 possible Drug-drug interactions, 1 drug –disease interaction

Serious

- Pantoprazole + Digoxin {Increase Gastric pH, Increase, toxicity of digoxin}
- Digoxin + Calcium-gluconate-Synergistic effect {Increase efficacy of Digoxin}

Monitor closely

- Meropenem + thiamine {Reduced thiamine absorption, Effects intestinal flora}
- Teicoplanin + Thiamine {Reduce thiamine efficacy}
- Heparin + Vasopressor {Reduced Efficacy of Vasopressor}

Ade (Adverse Events)

Surgical emphysema followed by Panic attack on Day-3 @ 4 pm Myocardial infraction @ 5:30 am, arrhythmia stabilized by Cardio version (150J) Death of Patient on Day-6 @ 6:38.

Discussion

Due to the numerous clinical manifestations of cardiogenic shock, septic shock, and hypovolemic shock as well as the limited range of available modern therapeutic options, shock is very difficult to treat. Endogenous catecholamines (epinephrine, norepinephrine, and dopamine) and other vasopressors that have shown effectiveness in the treatment of the different forms of shock are administered as part of the shock protocol as per article written by Kislitsina ON, Rich JD, et al. [8]. Thus, similar treatment was observed in our case too.

When three or more organ systems malfunction, a patient's prognosis is dire and fatality rates can be significant. This condition is known as Multiple Organ Dysfunction Syndrome (MODS), often referred to as organ dysfunction or organ failure [9]. As observed in this case where after Day-3 there was gradually failure of organs can

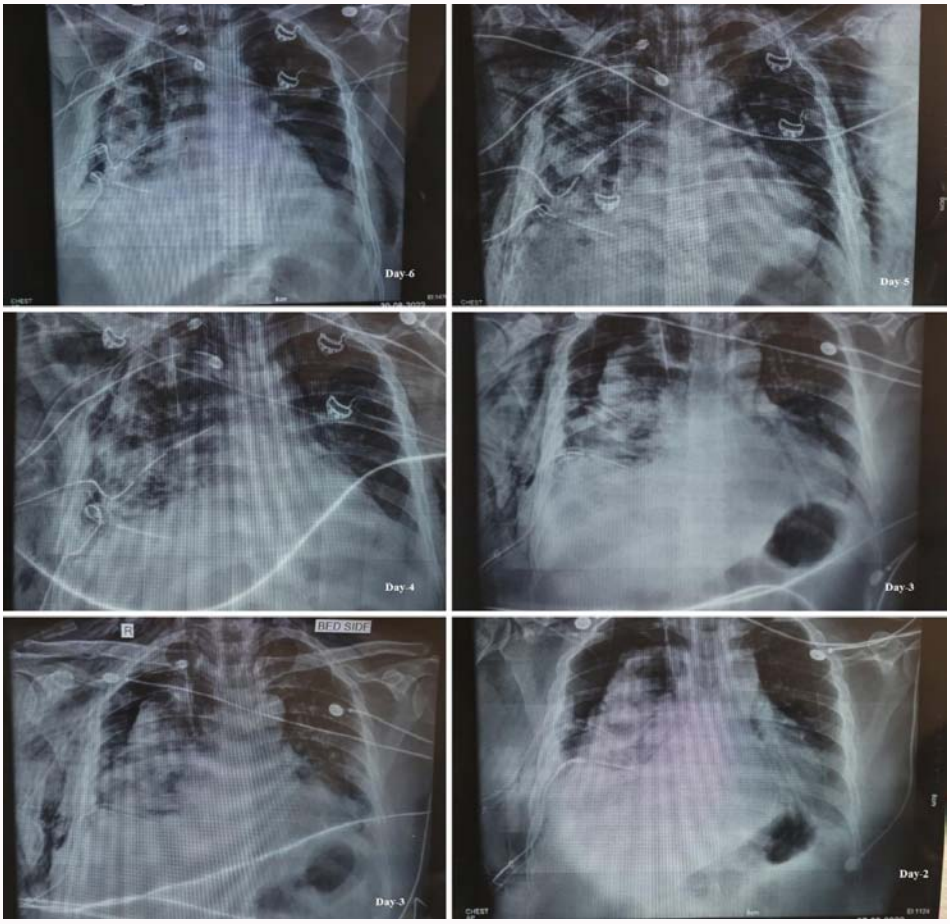

Figure 2.1: Chest X –ray (shows the Variation in Pleural effusion, persistent air leak in Rt pleural space from Day 2 to 6, Rt mild chest wall deformity and scoliosis and S/o Pneumothorax).

HISTOPATHOLOGY REPORT

BIOPSY NO.
HP- 2896/2022

SPECIMEN
A) Parietal pleura
B) Visceral pleura.

CLINICAL DETAILS
? mesothelioma
? T.B.

MACROSCOPY
Received two containers labelled as 1 & 2
A) Received multiple grey white to grey brown soft tissue bits altogether measuring 4 x 4 x 1 cm. All embedded A1 - A4
B) Received multiple grey white to grey brown soft tissue bits altogether measuring 3.0 x 3 x 1 cm. All embedded B1 - B3, B2 - lung

MICROSCOPY
Sections from parietal and visceral pleural tissue shows abundant fibrinous exudate, neutrophilic infiltrate, bacterial colonies, edema and fibroblastic proliferation with reactive atypia.
Sections from lung shows presence of clusters of macrophages in few alveolar space. Lymphomononuclear infiltrate noted at subpleural region. No evidence of granuloma / malignancy in the sections studied.

IMPRESSION
A) Parietal pleura - Acute inflammatory pleuritis
B) Visceral pleura - Acute inflammatory pleuritis
Lung - mild inflammation
No evidence of malignancy in the sections studied.

NOTE
Suggest clinical and radiological correlation.

*** End Of Report ***

Figure 2.2: Histopathology Report (Provides the information of about the histopathological Report of Pleura been sent to lab for examination).

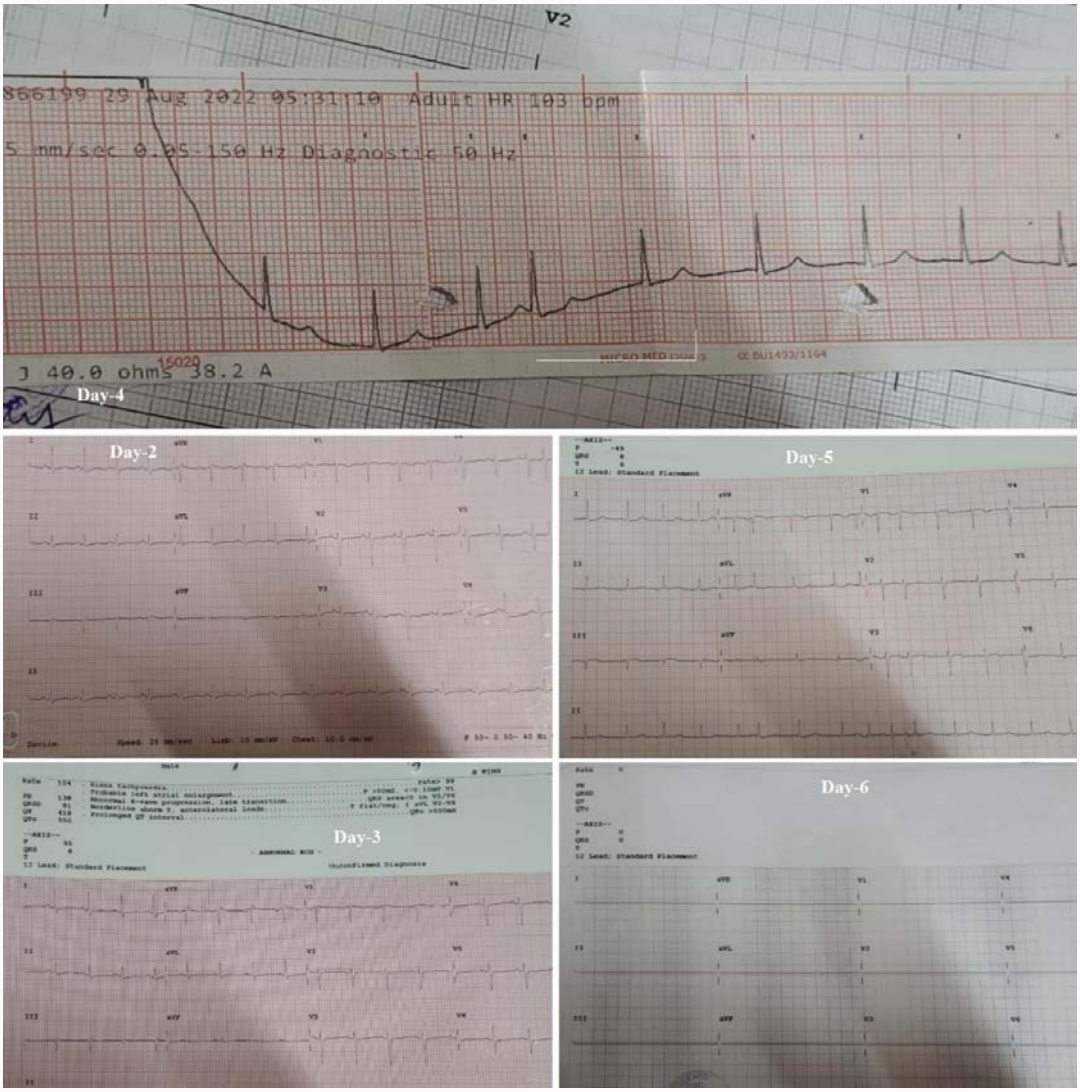

Figure 2.3: ECG (Shows variation in ECG on various days with day - 4 Cardioversion done).

be seen in Post operative reports in Tables 2.1-2.3.

Surgical emphysema in another term for subcutaneous emphysema. It happens when air or gas leaks into the subcutaneous tissue, the skin's lowest layer. Surgical emphysema can be brought on by an infection or a trauma. Bloating and a cracking sound when applying pressure to the swelling are typical indicators of surgical emphysema. Trusted Source. Here in this case Emphysema developed post-surgery due to persistent air leak as observed on Day-3 and also seen in reports i.e., Chest X-ray Figure 2.1. Similar reports observed in article [10].

In this case patient developed emphysema after performing VATS decortication which had risks like hemorrhage, persistent air-leak and bronchopleural fistula, persistent lung collapse, injury to vital structures, retained infective focus and sepsis, chest wall deformity and scoliosis and severe postoperative pain [11]. As the patient developed emphysema had a panic attack unknown of risks mentioned above like persistent air leak, etc. lead to hemodynamic instability.

There were several cases as such where patient developed postoperative anxiety like this one, therefore patients should be well counseled of pros and cons of procedure been considered to be operated on them [12].

Furthermore, VATS was considered by suspecting empyema but there was no pus accumulation as per the reports done after surgery and was just pleural effusion. The fluid and viscera collected was sent for biopsy can be in Figure 2.2 and 2.3 (Histopathological report) even Table 2.4 states there is no malignancy or bacterial growth in cultures. For this reason, we can consider this as a diagnostic surgery with suspected initial diagnosis of empyema as per pre operative reports chest CT Figure 1.2(A) and Figure 1.2(B). Such diagnostic surgeries consider in case of lung cancer as per article written by Naoki Ozeki et al. [13].

It is crucial for the treating physician to accurately record how a patient is being managed while in his care. The maintenance of medical records has developed into a distinctive expertise. The doctor will only be able to demonstrate that the treatment was administered correctly in this way. Additionally, it will be a huge assistance in the analysis and scientific examination of patient management difficulties. On the other hand, these days in the name international standards progress notes had been digitized and most the information been hide

from patient care provider/attainder and can be manipulative incase errors from the side of health care professionals [14]. As similar in our case were at the time of discharge the info provide in summary and progress notes were incomplete and partial. Beside manipulative in this case where the given record doesn't match the photo copy of the record previously taken for example cycles off CRRT done units of blood transfused, sedative given and dosage considered, other parameters etc.

Irrational usage of antibiotics was observed in this case where suspecting sepsis where report of culture sensitivity, gram strain and acid fast etc. shown no bacterial presence as noticed in Table 2.4. Even if considered as prophylactic usage therapy wouldn't go for three days or more days of broad-spectrum antibiotics in combination when 24 h report and 48 h show no growth. Kollef MH article states that before the pathogen causing the problem is discovered, treatment must begin. However, initial empirical therapy that is inefficient or improper is linked to higher rates of death, morbidity, and length of hospital stay [15].

According to article written by Prabhakaran ACJ, it is well known that the sedatives and analgesics used during restorative procedures have negative effects on heart hemodynamics. In the postoperative phase, this hemodynamic decline might occasionally result in multiple organ failure [16]. Similarly in our case fentanyl was given when tramadol or PCM was on causing negative effects on heart hemodynamics.

Nevertheless, there were therapeutic duplications, drug-drug interactions (Possible/monitored) and drug-disease interactions observed in this case and also conforms of study done by Hanlon JT, et al. [17].

Overall, despite these advancements, short-term death rates for shock are still reported to range from 20% to 50% [18].

Conclusion

To conclude shock is potentially lethal condition with a mortality-incidence ratio of 20% to 50%. But as per this case study, I would like end by saying there is need for proper initial diagnosis than going for diagnostic surgery, even though E-prescription or E-progression notes have their pros and cons in health care system, there is need to standardize it further for future purpose and information should be provide to patient care provider too without incomplete info/manipulated info. Patient should be counseled of procedure related risks in order to avoid Pre/post-operative anxiety/ panic attack. Rational Usage of Medications (Antibiotics, Ionotrops, sedatives etc) to be monitored. Therapeutic duplication, Drug-drug interactions, drug disease interactions to be monitored. In order to achieve desired therapeutic outcome.

Acknowledgement

Although it isn't an onerous or protracted case, I had tears in my eyes and sometimes felt anguish in my heart as I wrote about it. Because I had always been on the opposite side of the line handling a case while I was a student or an assistant professor. I delved into it and wrote this report to figure out what went wrong during those times when I had nothing in my hands and had to fend for myself while looking bewildered and worrying about the future.

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