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Management Of Septic Shock Et Causa Secondary Peritonitis With Complications Acute Kidney Injury And Hospital Acquired Pneumonia In Treated Patients In The Intensive Care Unit

Sitti Ziarah[1], Faisal[2], Haizah Nurdin[3], Ari Santri Palinrungi[4]
[1234]*Intensive Therapy Fellowship Of Anesthesiology And Intensive Therapy Subspecialthy Program Anesthesia, Medical Faculty Of Hasanuddin University*
Dr. Wahidin Sudirohusodo Central Public Hospital, Makassar, Indonesia

Abstract
Bacground: *Sepsis and Septic shock are reported to be the most common causes of death in the Intensive Care Unit (ICU). The reported incidence of sepsis in the world varies between 100-300 per 100,000 world population per year. Data in Indonesia itself is still limited. Peritonitis is a case of sepsis that often occurs. The mortality rate increases when it develops into septic shock. It can occur together with nosocomal infections. More than 40% of patients with septic shock also suffer from acute kidney injury (AKI). One case of sepsis which developed into septic shock due to secondary peritonitis, which was also complicated by acute kidney injury and hospital acquired pneumonia (HAP) was reported.*
Case Report: *Male, 51 years old, admitted to the Intensive Care Unit with sepsis due to secondary peritonitis, underwent laparotomy. In the course of treatment, experienced septic shock, human acquired pneumonia, acute kidney injury, hyperkalemia, hypoalbuminemia. Treated for 6 days with mechanical ventilation, given antibiotics according to culture and vasopressor support. Improvement occurred on day 6, with minimal vasopressor support as well as resolution and improvement.*
Conclusion: *Early management of critical patients regarding fluid resuscitation, mechanical ventilation management, antibiotic management, AKI management and management of HAP according to culture, is expected to reduce mortality rates and provide good outcomes.*
Keywords: *Septic Shock, Secondary Peritonitis, Acute Kidney Injury, Hospital Acquired Pneumonia, Intensive Care Unit.*

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I. Introduction

Sepsis is defined as life-threatening organ dysfunction caused by dysregulation of the host response to infection. Organ dysfunction can be assessed through the SOFA score. Septic shock is defined as sepsis accompanied by a disturbance in the circulatory and cellular metabolism that is large enough to cause death.[1] Sepsis and septic shock are major problems in health services. Millions of people worldwide die every year due to sepsis, with a ratio of 1 to 3, and affecting 1 in 6 people.[2]

In Intensive Care Unit (ICU) treatment, intra-abdominal infection is the most common cause of sepsis with a mortality rate of 10.5% worldwide. [3]Peritonitis is one of the most common causes of infection. [4]Peritonitis is localized or generalized inflammation in the peritoneal cavity which is generally caused by non-infectious substances such as gastric contents or bile contents. Peritonitis due to infection is classified as primary, secondary or tertiary. Secondary peritonitis is a peritoneal cavity infection originating from the intestine or pelvis and includes peritonitis following perforation of the hollow viscus, anastomotic leak, necrosis or other injury to the gastrointestinal tract. This peritonitis often occurs in critical surgical patients. [5]Secondary peritonitis occurs in 1% and is the second leading cause of sepsis in ICU patients globally. Overall mortality was 6%, but mortality increased to 35% in patients with severe sepsis. [6]Acute Kidney Injury (AKI) is the most frequent complication of sepsis which endangers 30-40% of patients in the ICU, more than 45% of patients with septic shock suffer from AKI3. The mortality rate for patients with AKI in the ICU exceeds 50%.[7]

In addition, sepsis can arise due to infections acquired from the community (community acquired), hospitals (hospital acquired) and the health care system (hospital acquired), where pneumonia is the main cause of more than 50% of the incidence of sepsis in hospitalized patients. in the ICU. [8]Hospital acquired pneumonia (HAP) is defined as a lung infection that develops during hospitalization, 48 hours or more after admission and

is absent or not within the incubation period at the time of admission. The incidence of HAP is 0.5–2.0% among all hospitalized patients and is the second most common nosocomial infection, but first in terms of mortality at 30–70%.[9]

II. Case Report

The patient is male, 51 years old, with a body weight (BW) 60 kg, height (TB) 160 cm, body mass index (BMI) 22.2 kg/m2 (normoweight), predicted body weight (PBW) 57 kg, came to the Emergency Room (ER) for surgery on 25 October 2022, with complaints of pain all over the stomach accompanied by a stomach that was getting bigger and bigger which had been experienced since 3 days before entering the hospital. Other complaints with fever come and go. History of last bowel movement 3 days previously, with a watery consistency and yellow color. History of defecating like goat feces. Urinating through a catheter since 1 day before. History of treatment at another hospital before being referred with a diagnosis of generalized peritonitis suspected of gastric perforation.

The patient was diagnosed with generalized peritonitis et causa suspected hollow viscus. Exploratory laparotomy + gastric perforation repair was performed. The operation lasted for 2 hours under general anesthesia with a bleeding output of 300 cc, ascites 1000 cc, urine 20 cc with an input of 1400 cc. During intraoperative surgery, it was found that the gastric organ had a perforation of <1 cm in the pyloric antrum, and the stomach was intact. The entire intestine appears intact. Refreshing the network and primary hecting all layers, omentum patch. After surgery the patient was consulted for Intensive Care Unit (ICU) treatment while maintaining mechanical ventilation.

Initial information obtained before surgery from vital signs with blood pressure (BP) 110/70 mmHg, pulse 118 x/minute, respiratory rate (RR) 33 x/minute, room air saturation 89-90%, Glasgow Coma Scale (GCS) 15, qSOFA gets the value.[1]

Routine blood examination with white blood cells (WBC) 2.6 x 103/µL (suggest leukopenia), albumin (alb) 3.1 g/dl, urea (ur) 213 mg/dl (impression increased 5.4 times), creatinine (kr) 4.70 mg/dl (increased impression 3.6 times), creatinine clearance 14.9 (decreased impression < 15), potassium (K) 5.1 millimoles/liter (high normal impression). The hypertension kidney department has been consulted, with the answer showing the impression of acute on chronic kidney disease (CKD) accompanied by hyperkalemia and hypoalbumin with plans for diagnostic urinalysis and abdominal ultrasound, with proposed protein diet therapy 1 g/kgbb, nephrosteril 250 cc/24 hours/intravenous (IV), correction of hyperkalemia with insulin novorapid in dextrose 10% 50 cc finished in 30 minutes.

Day 0 (26/10/2022 at 03.29 am) when admitted to the ICU, the data was obtained:

S	ETT installed
O:	
B1	Installed ETT 7.5 ID via *Jackson Reese* 8 lpm, vesicular, Rh-/-, Wh -/-
B2	BP 113/57 mmHg, MAP 69 mmHg, RR 119 x/i, regular, strong lift, warm acral, capillary refill time < 3 seconds
B3	GCS sedated, right pupil isochore 2.5 mm left 2.5 mm, light reflex +/+, normal impression, body temperature (SB) 36.6ºC
B4	Urine per catheter 100 ml/hour (sufficient impression), fluid balance -100 cc/4 hours, cumulative balance -100 cc
B5	no extremity edema, no fractures

Initial Assessment Day 0

1. Respiratory failure type 3 ec Sepsis ec Secondary peritonitis
2. AKI KDIGO 3
3. Hyperkalemia
4. Hypoalbumin

With the initial resuscitation management plan, the mechanical ventilator uses a lung protection strategy with a ventilator bundle protocol, hemodynamic monitoring, blood and sputum culture, administration of antibiotics and fluid and nutritional management.

Initial management in the *Intensive Care Unit* (ICU):

Ventilasi Mekanik dan evaluasi kondisi paru

In ICU day 0, mechanical ventilation was administered using a lung protection strategy via oropharyngeal ETT with SIMV-PC mode settings, Pins 10, frequency 16 x/minute (i), Tins 1.2, Psupp 10 cmH2O, PEEP 5, FiO2 70%, The TV produced is 400-500 ml, RR 19 x/i, vesicular, Rh-/-, Wh -/-. To evaluate the lungs, lung ultrasonography (USG), chest x-ray and AGD examination are carried out. With the results of the *bedside lung ultrasound* (LUS), no *b lines* were found in all right and left lung fields. The results of the chest x-ray at the beginning of admission to the ICU showed the impression of left pneumonia. The following are the results of a chest x-ray examination upon initial admission to the ICU:

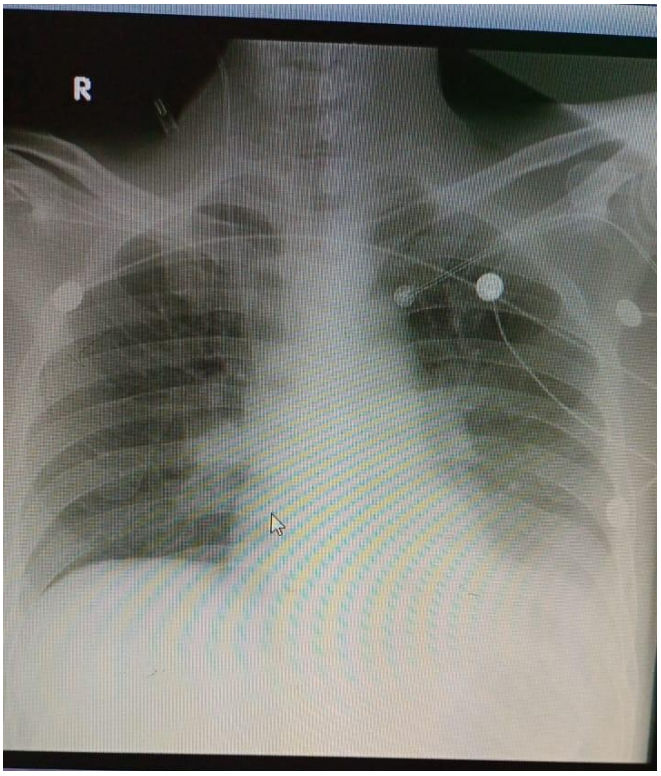


Figure 1. Chest photo upon initial admission to the ICU

A repeat chest x-ray was carried out the next day, but it still showed the impression of left pneumonia (st.Qa). The results of the BGA (Blood Gas Analysis) examination upon initial admission are:

Table 1. BGA at initial ICU admission

Jenis Pemeriksaan	Tanggal (26/10/2022) (FiO_2 100%, PEEP5)
pH	7,305
pO_2	318,8
pCO_2	38,8
HCO_3	19,5
Laktat	2,7
pf rasio	318,8
A-a DO_2	343,9

Sedation and Analgesia

The patient was deeply sedated using fentanyl at a dose of 0.5 mcg/kgbb/hour/iv/*syrine pump* (sp), and dexmedetomidine 0.5 mg/kgbb/hour/iv/sp also with atracurium 5 mg/hour/iv/sp

Installation of *Caphnograph*

A *caphnograph* was installed to measure ETCO2 levels, where $ETCO_2$ levels were targeted in the *normocaphnia* range (35-45 mmHg).

Installation of invasive access and monitoring

The patient has had central venous access installed in the right brachiocephalic vein, and the target in the ICU is to perform a central line bundle. In the ICU, an arterial line access is placed in the right radial artery with the aim of carrying out hemodynamic evaluation because patients with sepsis have the potential for septic shock, if there is a possibility of requiring the use of vasopressor support so that immediate intervention can be carried out, also to assess cardiovascular function, guidelines resuscitation and for access to carry out regular blood gas analysis checks.

Resuscitation

The patient was resuscitated before entering the ICU. When initially admitted to the ICU, BP was found to be 113/57 mmHg with MAP 69 mmHg (above 65 mmHg), with urine 100 cc/hour (above 0.5 ml/kg/hour with a sufficient impression), an initial *point of care ultra sound* examination was also carried out.

(POCUS) *inferior vena cava* (IVC) obtained a distensibility index of 17.2% (*distensibility index* value ≥ 12-18% fluid responsiveness) to predict fluid response. The following is a picture of POCUS IVC when initially admitted to the ICU:

Figure 2. Initial POCUS IVC entry (*distensibility index* 17.2%)

Echo examination was also carried out by cardiology (bedside) with interpretation of LV systolic function, good impression, EF 68%, RV systolic function, good impression, TAPSE 2.0, normal cardiac chamber, normal cardiac valves, global normokinetics, ERAP 8 mg (1.95/ 1.45). After day 1 of the patient being treated in the ICU, the patient was found to be hypotensive, BP 86/43 mmHg, MAP 58 mmHg, then norepinephrine support was installed with a dose titration of 0.05-0.01 mcg/kgbb/hour/sp/iv (before using the support, this was done POCUS IVC measurements, with a *distensibility index* of 15%), showed BP 97/66 mmHg, with MAP 75 mmHg. The patient used support for 6 days of treatment, and on the 6th day began to titrate again to the minimum dose. On day 1 of treatment, a cumulative balance of +1522 cc was also obtained. It was planned to remove the fluid using the diuretic furosemide titrated to a dose of 5-10 mg/hour/sp/iv with a negative balance target. Before using diuretics, a POCUSIVC examination was carried out again, showing a distensibility index of 15%. The following is a picture of POCUS IVC the following day:

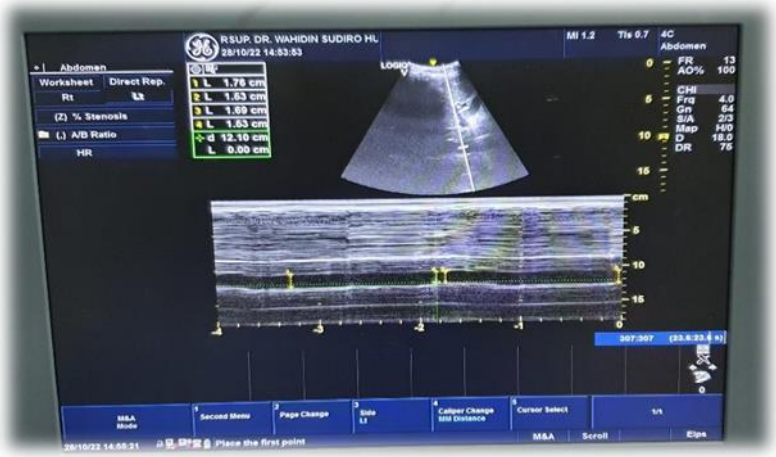
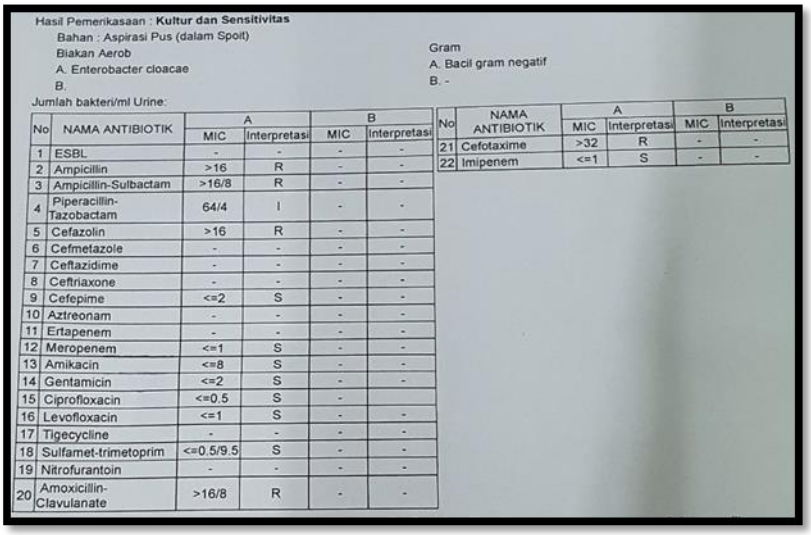


Figure 3. POCUS IVC the next day (*distensibility index* 15%)

Culture and administration of anti-microbials

When initially admitted, the patient received the antibiotic ceftriaxone 1 gram/12 hours/iv. On October 27 2022, pus aspiration was taken (in a syringe), culture and sensitivity examination was carried out, aerobic culture was obtained, *entero bacter cloacae*, gram negative bacilli. The following are the results of the culture examination and sensitivity of the pus aspiration.



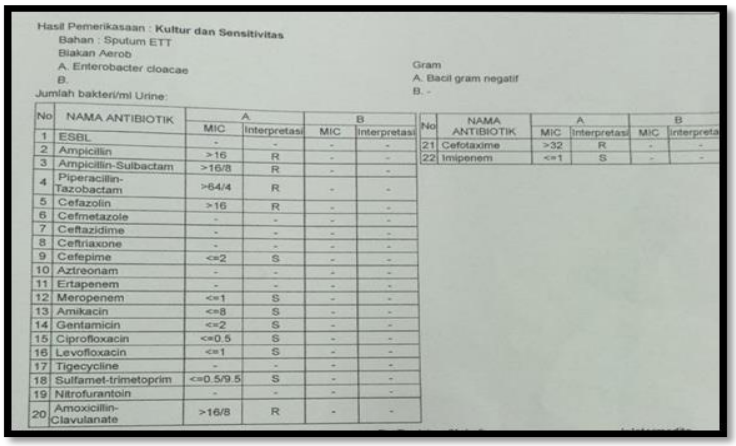
Hasil Pemeriksaan : **Kultur dan Sensitivitas**
Bahan : Aspirasi Pus (dalam Spoit)
Biakan Aerob
A. Enterobacter cloacae
B.
Jumlah bakteri/ml Urine:

Gram
A. Bacil gram negatif
B. -

No	NAMA ANTIBIOTIK	A		B	
		MIC	Interpretasi	MIC	Interpretasi
1	ESBL	-	-	-	-
2	Ampicillin	>16	R	-	-
3	Ampicillin-Sulbactam	>16/8	R	-	-
4	Piperacillin-Tazobactam	64/4	I	-	-
5	Cefazolin	>16	R	-	-
6	Cefmetazole	-	-	-	-
7	Ceftazidime	-	-	-	-
8	Ceftriaxone	-	-	-	-
9	Cefepime	<=2	S	-	-
10	Aztreonam	-	-	-	-
11	Ertapenem	-	-	-	-
12	Meropenem	<=1	S	-	-
13	Amikacin	<=8	S	-	-
14	Gentamicin	<=2	S	-	-
15	Ciprofloxacin	<=0.5	S	-	-
16	Levofloxacin	<=1	S	-	-
17	Tigecycline	-	-	-	-
18	Sulfamet-trimetoprim	<=0.5/9.5	S	-	-
19	Nitrofurantoin	-	-	-	-
20	Amoxicillin-Clavulanate	>16/8	R	-	-
21	Cefotaxime	>32	R	-	-
22	Imipenem	<=1	S	-	-

Figure 4. Culture results and sensitivity of pus aspiration

From the results of culture and sensitivity of pus aspiration, it was found to be sensitive to cefepime, meropenem, amikacin, gentamicin, levofloxacin, and imipenem. Furthermore, an examination of the culture and sensitivity of ETT sputum was also carried out on October 27 2022, the results of an aerobic culture of *entero bacter cloacae*, a gram-negative bacillus, were obtained. The following are the results of the culture and sensitivity examination of ett sputum.



Hasil Pemeriksaan : **Kultur dan Sensitivitas**
Bahan : Sputum ETT
Biakan Aerob
A. Enterobacter cloacae
B.
Jumlah bakteri/ml Urine:

Gram
A. Bacil gram negatif
B. -

No	NAMA ANTIBIOTIK	A		B	
		MIC	Interpretasi	MIC	Interpretasi
1	ESBL	-	-	-	-
2	Ampicillin	>16	R	-	-
3	Ampicillin-Sulbactam	>16/8	R	-	-
4	Piperacillin-Tazobactam	>64/4	R	-	-
5	Cefazolin	>16	R	-	-
6	Cefmetazole	-	-	-	-
7	Ceftazidime	-	-	-	-
8	Ceftriaxone	-	-	-	-
9	Cefepime	<=2	S	-	-
10	Aztreonam	-	-	-	-
11	Ertapenem	-	-	-	-
12	Meropenem	<=1	S	-	-
13	Amikacin	<=8	S	-	-
14	Gentamicin	<=2	S	-	-
15	Ciprofloxacin	<=0.5	S	-	-
16	Levofloxacin	<=1	S	-	-
17	Tigecycline	-	-	-	-
18	Sulfamet-trimetoprim	<=0.5/9.5	S	-	-
19	Nitrofurantoin	-	-	-	-
20	Amoxicillin-Clavulanate	>16/8	R	-	-
21	Cefotaxime	>32	R	-	-
22	Imipenem	<=1	S	-	-

Figure 5. Culture results and sensitivity of ett sputum

From the results of culture and sensitivity of pus aspiration, it was found to be sensitive to cefepime, meropenem, amikacin, gentamicin, ciprofloxacin, levofloxacin, sulfameth-trimethoprim and imipenem. Apart from that, a gram staining examination of the ett sputum specimen was also carried out on October 27 2022. The following are the results of the gram staining examination of the ett sputum specimen.

PEMERIKSAAN	HASIL	NILAI RUJUKAN	SATUAN
MIKROBIOLOGI			
Mikrobiologi			
Pengecatan Gram			
Spesimen	Sputum ETT		
Afinitas Gram	Gram positif dan Gram negatif		tidak ditemukan
Bentuk dan Konfigurasi	Diplococcus dan Bacil satu-satu		tidak ditemukan
Kuantitas	Positif (4+) dan Positif (2+)		tidak ditemukan
Lokalisasi	-		tidak ditemukan
Sel Lain	Leukosit :2+ dan Epitel :4+		tidak ditemukan
Jamur	Yeast dan Hypa		tidak ditemukan
Biakan dan Resistensi (Medium Padat)			
Metode Pemeriksaan			
Jenis Spesimen			
Kultur & Sensitivitas			

Figure 6. Results of gram staining of ett sputum specimens

From the gram staining results, yeast and hypa fungi were obtained. Based on the results of culture examination and the sensitivity of pus aspiration, ETT sputum and gram staining of ETT sputum specimens, it was decided to use a combination of antibiotics meropenem 1 gram/8 hours/IV and levofloxacin 750 mg/24 hours/IV, as well as oral antifungal fluconazole 800 mg per NGT. on the first day divided into 2 doses per 12 hours, the next day 400 mg per day divided into 12 hours of administration.

Correction of Hyperkalemia

Correction of hyperkalemia with insulin novorapid 10 units in dextrose 40% 50 cc finished in 30 minutes, the first day found K 5.1 millimoles/liter, the second day 5.6 millimoles/liter and the third day decreased 4.9 millimoles/liter, with daily blood sugar monitoring between 140-180 mg/dl.

Other Checks

Results of abdominal ultrasound examination (bedsidera diology): for both kidneys, size, echo cortex and corticomedullary differentiation were within normal limits. PCS is not dilated, no echo stones/SOL are visible. Due to changes in hemodynamic conditions, BGA and chest x-ray, the assessment on the following day changed.

Table 2. BGA day 1

Type of Inspection	Date 10/27/2022 (PEEP 5, FiO2 50%)
pH	7,48
pO2	95,5
pCO2	39,1
HCO3	24,9
Laktat	1,2
pf rasio	191
Aa-DO2	210

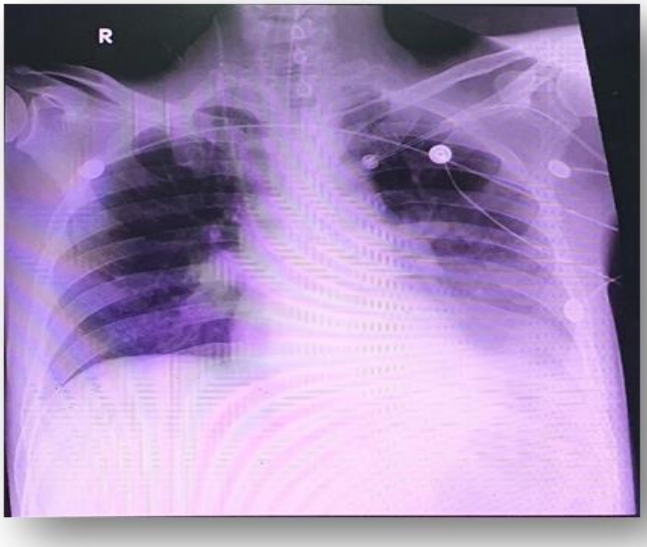


Figure 7. Control chest photo (impression: still pneumonia compared to previous st.Qa status)

Assessment on Day 1

1. Respiratory failure type 4ec Sepsis shock ec Secondary peritonitis dd HAP
2. Sepsis Shock
3. HAP
4. *Moderate Acute Respitratory Distress Syndrome*

Perawatanselama di ICU

From the results of the examination, it was found that the patient was critically ill with an APACHE II score of 21 with an estimated mortality rate of 40%, and a SOFA score of 9, with an estimated mortality rate of 15-20%. The patient's daily management is carried out using the FASTHUGBID protocol, where the therapy plan from the initial day of admission to the 6th day of ICU can be seen in the following table:

Table 3. Summary of subjective and objective examinations, assessments, plans and actions during the day of treatment in the ICU

Treatment Day Date	Subjectivity and Objectivity	Evaluation	Plan	Action
Day 0 10/26/ 2022	S : Ventilator O : B1 = O2 via ETT on mechanical ventilator SIM V-PC mode, Pins 10 fr 16x/i, Tins 1.2, Psupp 10 cmH2O, PEEP 5, FiO2 70%, TV 400-500, RR 19 x/i, vesicular, Rh-/- ,Wh -/-, SpO2 100%. B2: BP 113/57 mmHg, MAP 69 mmHg without support, N 119 x/irregular, lifting strength, warm acral, capillary refill time (CRT) <3 seconds. B3 = sedated GCS, isocorricular pupil size 2.5mm ka/ 2.5 mm ki, light reflex +/+ normal impression, S 36.6 ºC, BPS 3 B4 = Urine per catheter, 100 ml/hour, bc -100 ml/4 hours, bk -100 ml B5 = Supel, defansmuscular (-), peristalsis 5-6 x/i, normal impression, NGT (+), residue - ml B6 = Edema (-), Fracture (-) WBC 2.6 103/µL Ur 213 mg/dL Kr 4.70 mg/dL Alb 3.1 gr/dL lactate 2.7 K 5.1 millimoles/liter AGD: pH 7.305 HCO3 19.5 pCO2 38.8 pO2 318.8 pf ratio 318.8 Chest x-ray: pneumonia S/ POCUS IVC 17.2% qSOFA 1 SIRS 2	- Respiratory failure type 3 ec sepsis ec secondary peritonitis -AKI KDIGO 3 - Hyperkalemia - Hypoalbuminemia	- Resuscitation -Mechanical Ventilation - Hemodynamic monitoring -Antibiotics - Culture -Fluid and nutritional management -The arterial line/CVC has been installed - Check AGD, chest photo	F = Clear fluid dextrose 5% 10 cc/hour/NGT A = Fentanyl 30 mcg/hour/sp/iv, metamizole 1gr /8 hours/iv S = Dexmedetomidine 0.5 mg/kgbb/hour/sp/iv T = - H = Heads up 15-30 U = Omeprazole 40 mg/12 hours/iv G = Check GDS/24 hours, target GDS 120-180 mg/dl S = O2 via ETT B:- I = IVFD 1500 ml/24 hours D = Ceftriaxon 1gr/12 hours/intravenous (H1), Tranexamic acid 500mg/8 hours/iv (H1), Atracurium 5 mg/hour/iv/sp
Day 1 10/27/ 2022	S : Ventilator O : B1 = O2 via ETT on mechanical ventilator SIMV-PC mode, Pins 8 fr 16x/i, Tins 1.2, Psupp 8 cmH2O, PEEP 5, FiO2 50%, TV 350-400, RR 19 x/i, vesicular, Rh -/- ,Wh -/-, SpO2 100% B2: BP 86/43 mmHg, supported by norepinephrine 0.05-0.1 µg/kgbb/minute/sp/iv, obtained BP 97/66 mmHg, MAP 75 mmHg, N 94 x/i B4 = Urine per catheter, 50 ml/hour, bc +1622/4 hours, bk +1522 ml K : 5.6 millimoles/liter AGD : pH 7.408 HCO3 24.9 pCO2 35.3 pO2 95.5 pf ratio 191 lactate 1.2 SOFA Score 9 APACHE II 21 PSI 130 Chest x-ray: pneumonia S/ (st.Qa compared to previous) POCUS IVC 15% *Echo (bedside)*: LV *systolic function* good impression EF	-Respiratory failure type 4 ecSeptic shock ec Secondary peritonitis dd HAP -Septic shock -HAP -ARDS Moderate -Hyperkalemia	-Antibiotics according to culture + antifungal -Diuretic -Correction of hyperkalemia -Vasopressors -Check inflammatory biomarkers	D = Meropenem 1 gr/8 hours/iv + Levofloxacin 750 mg/24 hours/iv, atracurium stop, furosemide 5-10 mg/hour/sp/iv, oral fluconazole 400 mg/12 hours/NGT, insulin novorapid 10 units in dextrose 40% 50 cc/sp runs out in 30 minutes

Treatment Day Date	Subjectivity and Objectivity	Evaluation	Plan	Action
	68%, *RV systolic function* good impression, TAPSE 2.0, normal *cardiac chamber*, normal *cardiac valves*, global normo kinetic, ERAP 8 mg (1.95/1.45) Abdominal ultrasound (*bedside*): for both kidneys, size, *echo cortex* and *corticomedullary differentiation* are within normal limits. PCS is not dilated, no *echo* stones/SOL are visible Results of culture of pus aspiration and ett sputum: sensitive to meropenem, levofloxacin Gram staining results: found *yeast* and *hypa* fungi			
Day 2 10/28/ 2022	S : Ventilator O : B1 = O2 via ETT on mechanical ventilator SIMV-PC mode, Pins 14 fr 25x/i, Tins 1.2, Psupp 10 cmH2O, PEEP 10, FiO2 60%, TV 400-500, RR 28 x/i, vesicular, Rh-/- ,Wh -/-, SpO2 100% B2: BP 86/63 mmHg, supported by norepinephrine 0.05-0.1 µg/kgbb/minute/sp/iv, obtained BP 122/82 mmHg, MAP 92 mmHg, N 59 x/i B4 = Urine per catheter, 38 ml/hour, bk +706 ml K : 4.9 PLT : 116,000 Neut 91.2 Ur 155 Kr 1.55 GOT 104 Alb 2.7 CRP quantitative< 130 Procalcitonin 37.00 AGD: pH 7.523 HCO3 29.3 pCO2 35.3 pO2 102.5 pf ratio 128.2 lactate 1.9	-Hyperkalemia (improvement) -Hypoalbumin		D = fluconazole 200 mg/12 hours/NGT
Day 3 10/29/ 2022	B2: BP 86/63 mmHg, supported by norepinephrine 0.05-0.1 µg/kgbb/minute/sp/iv, obtained BP 122/82 mmHg, MAP 92 mmHg, N 59 x/i B4 = Urine per catheter, 38 ml/hour, bk +706 ml AGD: pH 7.332 HCO3 40 pCO2 74.9 pO2 85.5 pf ratio 106.9 lactate 1.3			D = Tranexamic acid stop
Day 4 10/30/ 2022	S : Ventilator O : B1 = O2 via ETT on mechanical ventilator SIMV-PC mode, Pins 18, f 27 x/i, Tins 1.2, Psupp 10 cmH2O, PEEP 12, FiO2 80%, TV 400-450, RR 30 x/i, vesicular , Rh-/- ,Wh -/-, SpO2 100%, B2: BP 149/90 mmHg, MAP 102 mmHg support NE 0.1 µg/kgbb/minute/iv/sp, N 78 x/i B4 = Urine per catheter, 38 ml/hour, bc +249 cc/24			

Treatment Day Date	Subjectivity and Objectivity	Evaluation	Plan	Action
	hours AGD: pH 7.461 HCO3 41.9 pCO2 58.3 pO2 115 pf ratio 143.7 lactate 1.3			
Day 5 10/31/ 2022	S : Ventilator O : B1 = O2 via ETT on mechanical ventilator SIMV-PC mode, Pins 12, f 18x/i, Tins 1.2, Psupp 10 cmH2O, PEEP 7, FiO2 60%, TV 350-400, RR 20 x/i, vesicular, Rh-/- ,Wh -/-, SpO2 100%, B2: BP 143/63 mmHg, MAP 90 mmHg support NE 0.1 µg/kgbb/minute/iv/sp, N 66 x/i B4 = Urine per catheter, 64 ml/hour, bc +323 cc/24 hours, bk -681 cc AGD: pH 7.491 HCO3 39.4 pCO2 51.2 pO2 111.1 pf ratio 185.2 lactate 1.5 Ur 66 mg/dL Kr 1.10 mg/dL PCT 12.90 CRP 16.4			
Day 6 11/1/2022	S : Ventilator O : B1 = O2 via ETT on mechanical ventilator spontaneous mode, Pins 8, Tins 1.2, Psupp 10 cmH2O, PEEP 5, FiO2 45%, TV 350-400, RR 30 x/i, vesicular, Rh-/- ,Wh - /-, SpO2 98%, RSBI 75 breath/min/L, RSBI interpretation < 105 (positive), prediction of successful extubation, then the patient is extubated, then non-rebreathing mask (NRM) 8 lpm, RR 20-30 x/i, SpO2 98% B2: BP 135/58 mmHg, MAP 84 mmHg support NE minimum 0.05 µg/kgbb/minute/iv/sp, N 65 x/i B3 = 10x (E4M6Vx), after GCS 15 extubation B4 = Urine per catheter, 72 ml/hour, bc -506 cc/24 hours AGD: pH 7.515 HCO3 38.5 pCO2 45.9 pO2 77.5 pf ratio 155.1 lactate 1.4 Chest x-ray: pneumonia S/ improvement from before Ur 49 mg/dL Kr 0.7 mg/Dl	-Septic shock (improvement -HAP (repair) -AKI (repair)	*-Weaning* *-Extubation* *-Moving to high care (HCU)*	F = Soft diet S = O2 via NRM 8 lpm B:- I = IVFD 1000 ml/24 hours D = Meropenem 1 gr/8 hours/iv (H6), Levofloxacin 750 mg/24 hours/iv (H6), fluconazole 200 mg/12 hours/oral, furosemide stop

III. Discussion

Sepsis and septic shock

It was explained earlier regarding the definitions of sepsis and septic shock. Clinically, septic shock is said to be sepsis accompanied by tissue hypoperfusion, with hypotension using vasopressors accompanied by increased lactate. In patients with sepsis, initial scoring can be assessed using qSOFA which includes 3 things, namely consciousness, systolic blood pressure and respiratory rate.[1] This initial assessment is expected to help early resuscitation in reducing morbidity and mortality rates. In this case, qSOFA was obtained with a score of 1, namely a respiratory rate of 33 x/minute. The patient still has a Glasgow Coma Scale (GCS) of 15, and a systolic blood pressure above 100 mmHg, namely 110 mmHg. Apart from qSOFA, other parameters are used, namely the SIRS criteria. In this case, the SIRS score was 2, namely tachypnea > 30 x/minute, and leukopenia was found (2.6 x 103/µL).[2]

As for organ dysfunction, the SOFA score was calculated and the score was 9 with a mortality rate of 20%, and the APACHE II score was calculated as 21, with a mortality rate of 40%.[10] Apart from that, the patient was also calculated for the pneumonia severity index (PSI) and obtained a score of 130 with a mortality rate of 8.2%.[8] Sepsis can have an effect on all organs, especially the cardiovascular and respiratory systems, manifestations can be hypotension and ARDS. The manifestations of AKI can include oliguria and an increase in serum creatinine.[1]

Resuscitation management

The patient was resuscitated before entering the ICU. Regarding resuscitation, there are 4 phases: rescue (resuscitation), optimization, stabilization and de-escalation.[11] In the salvage phase, where resuscitation begins on day 0. In this case, the patient has been resuscitated previously. When initially admitted to the ICU, BP was found to be 113/57 mmHg with MAP 69 mmHg (above 65 mmHg), with urine 100 cc/hour (above 0.5 ml/kg/hour with a sufficient impression), and an initial POCUS IVC examination was also carried out and it was found to be 17. 2% (distensibility index value ≥ 12-18% fluid responsive) to predict fluid response.[12 13] Echo examination was also carried out by cardiology (bedside) with interpretation of LV systolic function, good impression, EF 68%, RV systolic function, good impression, TAPSE 2.0, normal cardiac chamber, normal cardiac valves, global normo kinetic, ERAP 8 mg (1 .95/1.45).

In phase 2 of optimization, reassess resuscitation. After confirming that the patient has been resuscitated, the impression volume status is sufficient, then stabilize with fluids and active vaso therapy until the patient is stable. POKUS IVC measurements were carried out, a distensibility index of 15% was obtained (with a fluid responsiveness between ≥ 12-18%). Next in phase 3, stabilization, continuing monitoring and titration of fluids and active vaso therapy. On the first day to the 6th day, monitoring and stabilization is carried out. At the beginning of admission, the lactate examination was found to be 2.7 millimoles/liter, and on the first day of treatment the BP was 86/43 mmHg (POCUS IVC 15%), this patient experienced hypotension and was then given norepinephrine support at a dose of 0.05-0.1 µg/kgbb /minute/iv/sp, BP was 97/66 mmHg with MAP 75 mmHg. The vasopressor used is norepinephrine at a dose of 0.05-0.1 µg/kgbb/minute/iv/sp with dose titration with a target MAP ≥ 65 mmHg. On the 6th day with a minimum dose and then stopped, the urine output during treatment seems sufficient (0.5-1 cc/kgbb/hour).

The next phase is de-resuscitation. On the 1st day of treatment, the fluid balance and cumulative balance were positive, and on the 2nd day, the cumulative balance was still positive, and on the 3rd day, the fluid and cumulative balance was negative. To obtain a negative balance, this patient underwent drug intervention in the form of the diuretic furosemide which was carried out at a titrated dose of 5-10 mg/hour/sp/iv given from days 1-6.

Regarding the concept of removing fluid, it is said that it can be done using pharmacological and non-pharmacological methods. Pharmacology is done using drugs, and non-pharmacology can be done using ultra filtration (UF). Regarding when this process is stopped if a target has been achieved, however there has never been a single marker of euvolemia that has been achieved. The goal to be achieved is a neutral balance, increasing oxygenation until extubation. In other references it is said that by looking at the capillary leak index (CLI) > 60 and the pf ratio < 150.[1114] In this case, when the balance was positive, the CLI calculation was carried out and the figure was 59.0 and the pf ratio at that time was 128. For daily fluid maintenance doses, use a dose of 25-30 ml/kgbb/day, which is around 1500-1800 cc/24 hours, using Ringer's lactate.

Mechanical ventilation management

Patients are maintained on mechanical ventilation from the operating room as initial control for patients with sepsis and septic shock for breathing in order to reduce oxygen consumption due to the work of the respiratory muscles and can provide good perfusion to vital organs. This patient was ventilated from day 0 in the ICU until day 6. When initially admitted he had type 3 respiratory failure ec sepsis ec secondary peritonitis. Type 3 respiratory failure (peri-operative) is a post-surgical disorder, the mechanism of which is atelectasis. While using mechanical ventilation, BGD is monitored periodically. The results of the BGD pf ratio on the initial day were 318.8 and the following day pf ratio was 191, which indicates a suggestive V-Q mismatch or diffusion defect.[15]

Monitoring BGD during treatment with PaO2 77.5-318.8 (target above 55-80 mmHg), SaO2 98-100% (target 88-95%), pplat 16-30 cmH2O (target < 30 cmH2O) and meeting the target pH 7.20-7.45. The PEEP variation during treatment is 5-12, with the tidal volume target achieved at 6-8 cc/kgbb PBW, TV between 300-500. The patient was extubated on day 6, after weaning in spontaneous mode for 120 minutes, with an RSBI of 75 breaths/min/L, with an RSBI interpretation of <105 (positive) predicting successful extubation. Furthermore, the patient was using NRM 8 lpm, hemodynamically stable, and was planned to move to high care treatment.

As for the criteria for carrying out weaning, some evidence suggests, namely adequate ventilation (PaO2/FiO2> 200, other references say> 150, receiving PEEP < 5-8 cmH2O, FiO2 < 0.4-0.5, and pH > 7.25),

hemodynamically stable, no myocardial ischemia, clinically no hypotension or on vasopressors (or on low doses of vasopressors), there is the ability to initiate inspiratory efforts. The ventilation goals in acute respiratory distress syndrome (ARDS) are mentioned, including oxygenation goals where PaO2 is 55-80 mmHg, or SpO2 88-95%, plateau pressure (Pplat) goal ≤ 30 cmH2O, driving pressure < 14 cmH2O, and pH goal 7 .2-7.45.[161718]

Antibiotic management

Regarding the administration of antibiotics, when initially admitted he received ceftriaxone 1 gr/12 hours/iv. On the first day, when septic shock occurred, it was decided to replace the antibiotic with meropenem 1 gr/8 hours/iv. Along with the culture and sensitivity results obtained from the pus aspiration and ett sputum which were still sensitive to meropenem and levofloxacin. So it was decided to give meropenem 1 gr/8 hours/iv and Levofloxacin 750 mg/24 hours/iv. From inflammatory biomarkers, Tawal leukocytes were 2.6 x 103/µL, CRP < 130 mg/L and PCT 37.00 ng/mL with the impression of increasing and decreasing on the 5th day of treatment with leukocytes 4.4 x 103/µL, CRP 16.4 mg/L and PCT 12.90 ng/mL. Patients are also given anti-fungals taking into account risk factors: long stay in the ICU, using broad spectrum antibiotics, using central veins, undergoing abdominal surgery. Gram staining also found yeast and hypa. So it was decided to give the anti-fungal fluconazole 800 mg/12 hours/NGT (H1), the second treatment day and then fluconazole 400 mg/12 hours/NGT.

A combination of beta-lactams and macrolides can be used in patients with pneumococcal bacteremia. 19 In addition, in this case, with risk factors for hospitalization > 48 hours, also accompanied by renal failure at the time of initial admission, the choice is to use a carbapenem (meropenem), as well as a pseudomonal fluoroquinolone (levofloxacin), also because it was found to be sensitive in cultures of sputum and surgical wound pus. Regarding the duration of antibiotic use, it can be given for 5 days. In general, it can be given in 7-10 days, and for slow responders it can be given up to 14 days.[20]

When associated with HAP, the pathogens most often involved are aerobic gram-negative bacilli *(Pseudomonas aeruginosa, Escherichia coli, Klebsiella pneumoniae, Acinetobacter spp etc*.) and *Staphylococcus aureus*. The most accurate clinical criteria for starting empiric antibiotic therapy: the presence of progressive new radiographic infiltrates, 2-3 clinical symptoms (fever, leukocytosis/leukopenia and purulent discharge). There are recommendations for 4 major factors and recommendations for the diagnosis of HAP, which include medical history and physical examination, chest photo (the results of this patient's chest photo show pneumonia), AGD, blood culture, thoracic osynthesis (if there is pleural effusion), endotracheal aspiration, broncho alveolar lavage, extra pulmonary infection site for examination.[9]

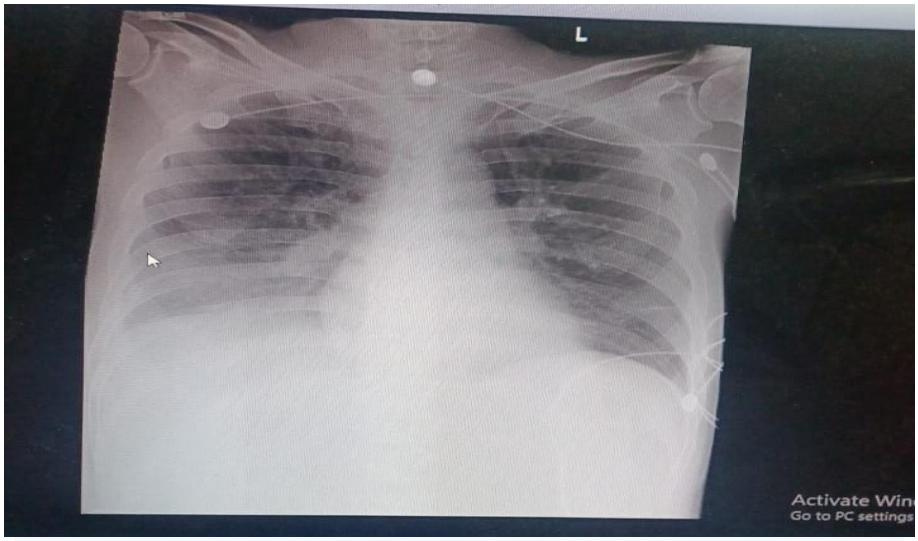


Figure 8. Control chest photo on day 6 (impression: improved left pneumonia)

Acute Kidney Injury

At the beginning of admission, the urea and creatinine were found to have increased, namely kr 4.7 mg/dl with ur 213 mg/dl, during the treatment day the kr and ur decreased, it was found kr 0.7 mg/dl and ur 49 mg/dl. AKI occurred in this case where the results of urea and creatinine at the time of initial admission were 243 mg/dl and 4.7 mg/dl with strong suspicion that it was a prerenal process because the patient had a previous history of loose stools, as well as the incidence of sepsis and septic shock in the patient. This patient may have hypovolemia, and is supported by ultrasound results with good renal interpretation, there is no corticomedullary differentiation (this indicates it is not a chronic process). The patient's urea and creatinine gradually recovered

with the final results obtained at 49 mg/dl and 0.7 mg/dl. The causes of AKI can be prerenal, renal and postrenal.[7] Prerenal causes include hypovolemia, gastrointestinal fluid loss (vomiting, diarrhea), renal fluid loss due to diuretic use, diabetes, hypoadrenalism, burns, bleeding, extravascular sequestration such as pancreatitis, trauma and hypoalbumin, low cardiac output, myocardial disease, valve , pericardial tamponade, massive pulmonary embolism, low renal perfusion pressure, shock (such as sepsis), abdominal compartment syndrome, systemic vascular resistance ratio, systemic vasodilation, sepsis, antihypertensives, anesthesia, renal vasoconstriction, hypercalcemia, use of norepinephrine, vasopressor agents , CyA, tacrolimus, hepatorenal syndrome with cirrhosis, hyperviscosity, multiple myeloma, macroglobulinemia. Renal causes include acute tubular necrosis, ischemic, toxins, exogenous (Hb, myoglobin, uric acid, oxalate, myeloma), acute interstitial nephritis, idiopathic, infectious (CMV, fungal, bacterial, pyelonephritis), infiltrative (lymphoma, leukemia, sarcoid), drug allergies (antibiotics, NSAIDs, diuretics), glomerular diseases, acute glomerulonephritis, vasculitis, hemolytic uremic syndrome (thrombotic thrombopenic purpura, disseminated intravascular coagulation, apreeclamptic toxemia, systemic lupus erymatosus, scleroderma, radiation), renovascular, renal vein thrombosis, renal artery (stenosis, plaque, embolism, thrombus, aneurysm), intratubular deposition and obstruction, myeloma protein, uric acid, oxalate, crystallacyclofir, methotrexate, sulfonamides, renal allograft rejection. The postrenal causes include ureteric, obstruction, external compression (retroperitoneal fibrosis), calculi, sloughed papilla, cancer, blood clot, bladder neck obstruction, neurogenic, prostatic hypertrophy, urethral obstruction, stricture, congenital valve, phimosis.[7]

Regarding Central Venous Pressure (CVP) is a local hemodynamic parameter that reflects intravascular volume and shows the interaction between venous return and cardiac function. CVP is generally used for bedside assessment of volume status and response in critically ill patients. However, the validity of CVP has recently been debated. In critical patients with multiple comorbidities including sepsis, heart failure, arrhythmia, hypertension, diabetes or others, the relationship between increased CVP and AKI is still unclear. Current research shows inconsistent conclusions about the relationship between CVP and AKI in critically ill patients. In this patient, CVP measurements were not carried out. In a study by Sun R et al, it was concluded that the average CVP value was associated with an increased risk of AKI in critical patients with several comorbidities.[21]

Nutrition

Regarding this patient's nutrition, a nutritional score of 5 was obtained, with the interpretation of a high score, it is said that he will probably benefit from nutritional therapy. But this patient's nutrition started on the 6th day after the patient was extubated. During the 5 days of treatment, patients with enteral feeding via NGT used 5% dextrose. In line with Aspen's guidelines of tolerating nutrition for less than 7 days. The Surviving Sepsis Campaign 2021, for adult patients with sepsis and septic shock who can be fed enterally, recommends early (within 72 hours) enteral nutrition (weak quality of evidence).[2] Nutritional evidence-based ICU guidelines recommend early nutrition progressively implemented within 48 hours. hours, however it is said that failure or inability to utilize enteral nutrition, in combination with inflammation and interstitial edema leads to bacterial translocation. On the one hand, prolonged fasting depletes the luminal brush border enzymes needed for digestion.[22] Regarding the link between nutrition and AKI, it does not provide recommendations regarding better early or late nutrition, it only says to be careful about excess water, hyponatremia (electrolyte gain). More importantly enteral nutrition is to maintain intestinal integrity.[23] In this case, early enteral consideration is not immediately given based on SSC 2021 that early nutrition can be given within 72 hours, also the latest Espen guideline can be given within 48 hours (24), also discuss with the doctor surgical treatment to delay up to 5 days. Apart from that, we consider that the patient can still be hypocaloric (within 7 days), Aspen can still tolerate up to 7 days, another option that can also be given is peptide-based nutrition. Vital 1.5 kcal (Abbot nutrition) is a recommendation if there are concerns about gastrointestinal malabsorption/intolerance. (abdominal surgery) which is a concern for the surgeon.

IV. Conclusion

It has been reported that the treatment of patients with sepsis and septic shock accompanied by complications of AKI and HAP, where management has been carried out including fluid resuscitation management, mechanical ventilation management, culture appropriate antibiotic management. Sepsis and septic shock with complications are complex problems and have a high mortality rate in the ICU. With good management, it is hoped that it will provide better outcomes and life expectancy.

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