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Case Report

Fat Embolism Syndrome (FES) In Patient With Multiple Injury: Case Report

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Abstract

Background: Fat embolism syndrome (FES) is defined as a symptom that arises due to the presence of fat clots in the pulmonary or peripheral circulation. Although it is very rare but FES has a mortality rate of 10%-20%.

Aim: We report FES in patient with multiple injury who experience close fracture femur dextra, open fracture cruris sinistra, and open degloving antebrachii dextra.

Case Report: Women aged 20 years came to IGD after being struck by a wall. The patient complained of pain in the femur dextra, cruris sinistra, and ramus antebrachii dextra. Deformities were found on the femur dextra and cruris sinistra. Then the patient got debridement for antebrachii dextra and ORIF for tibia sinistra and femur dextra. In the second day of hospitalization (two days after the trauma or one day after orthopedic action) the patient experienced restlessness followed by increasing heart rate. On the third day of hospitalization, patient got altered in consciousness followed by increasing in respiratory rate, heart rate, and temperature, and decreased SpO2 to 89%-91%.

Conclusion: Fat embolism syndrome is a critical condition that can improve on its own when it gets proper treatment.

Keywords: Fat Embolism Syndrome; Fracture Femur; Orthopedic Action

Introduction

Fat embolism syndrome (FES) is defined as a symptom that arises due to the presence of fat clots in the pulmonary or peripheral circulation. Generally, FES occurs after trauma and orthopedic procedures within 24-72 hours. FES occurs very rarely, the percentage of FES occurrence in trauma or orthopedic procedures by 30% with a mortality rate of 10%-20%. FES's risk factors include male gender, young age (10-40 years), obesity, multiple fractures, and long bones fractures.

FES is diagnosed based on a combination of Gurd Wilson criteria. According to Gurd and Wilson, FES symptoms are divided into major and minor criteria. Major criteria include respiratory disorders, cerebral manifestations, and petekie rash. Minor criteria include pyrexia, tachycardia, retinal changes, jaundice, and renal changes. For diagnosing FES, the patient must have two major criteria or one major criterion followed by four minor criteria. The criteria of Gurd and Wilson were enhanced by Lindeque through the assessment of PaO2 > 8 kPa (60 mmHg); PaCO2 > 7.3 kPa (55 mmHg) or pH < 7.3; respiratory rate > 35 x/min Even after receiving adequate sedation; and increased breathing work assessed through additional muscle respiration use, tachycardia, and anxiety.

Case Report

A 20-year-old woman came to IGD after being crushed by a collapsed wall. Primary surveys show patients come with stable hemodynamics, GCS 456, and no active bleeding. From the secondary survey, patients have deformities in the femur dextra and cruris sinistra. In the antebrachii dextra found the presence of crush injury. Patients then received a clinical diagnosis close fracture femur dextra, open fracture cruris sinistra grade II, and open degloving antebrachii dextra. The patient then underwent an X-ray photograph in a wound to strengthen the diagnosis (Figure 1). Then the patient was consuled to orthopedic companions and received advice for debridement and the Open Reduction Internal Fixation (ORIF) action in the tibia and femur.

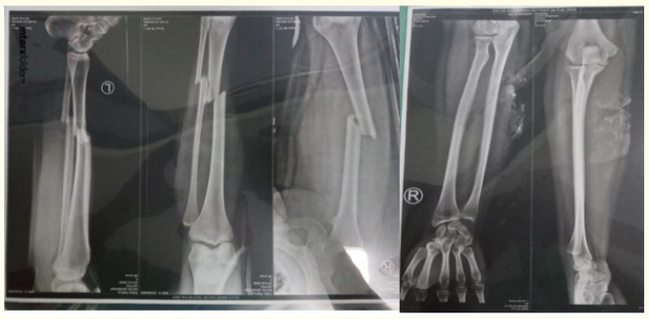

Figure 1: X-Ray photo's patients when beginning to come to IGD.

Second day hospitalized or one day after the orthopedic action, the patient begins to show a sign of restlessness. Vital signs are still within normal limits, so is hemoglobin. Then, the patient was injected in half the ampoule diazepam and consuled to the neurosurgeon's colleague. From neurosurgery doctor, he gave advice to check electrolyte serum and head CT-Scan. The results of the electrolyte serum and CT-Scan within normal limits, so the patient only got conservative treatment.

On the fourth day hospitalized or three day after orthopedi action, the patient suffered a decline in consciousness with the GCS 215. Patients also experienced takipneu with a respiratory rate of 28 x/min, pulse rate of 120 x/minute, followed by a decrease in SpO_2 to 89%-91%. Then, the patient was consuled to a nervous doctor. Advice obtained is the diagnosis of encephalopathy followed by the recommendation for the x-ray thorax (Figure 2), liver examination and renal function test, installing the nasogastric tube, and the installation of the O_2 simple mask of 6 liter per minute.

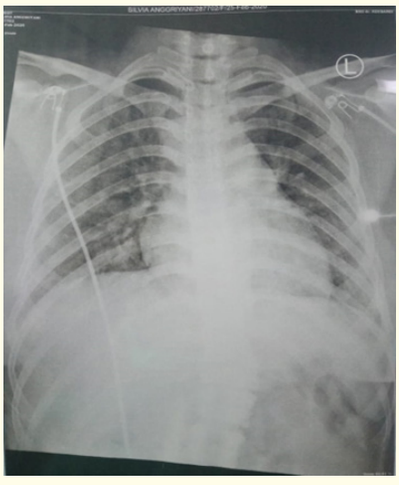

Figure 2: Photos of Thorax X-ray patients on the fourth day of treatment shows bilateral flufy sign.

The fifth day hospitalized, the patient was still experiencing a decline in consciousness followed by hemodynamic instability. Blood pressure 100/70 mmHg, pulse rate of 137 x/min, axial temperature 40 °C, respiratory rate 40 x/min, and followed by a decrease of SpO_2 to 67%-77% with simple mask. Then, the simple mask was replaced by the O_2 reservoar mask which followed by the rise of SpO_2 to 96%-97%. The patient was re-consuled to a nervous specialist and received a normal saline 0,9% 20 mL as loading therapy with 1000 mL as maintenance and vascon at a speed of 50 mL/h. The five-hour interval, the patient suffered a decrease in conditions with blood pressure 80/40 mmHg, pulse rate of 140 x/min, respiratory rate of 40 x/min, and GCS 224. Then, the patient was consuled to an anesthetic doctor and received advice to move the patient to an intensive care unit (ICU) with a diagnosis fat embolism syndrome. Patients have been conducted resuscitation with the loading of Ringer's lactate as much as 1000 mL and increased blood pressure to 120/70 mmHg, for other vital signs still remain the same.

While in ICU, the patient was used ventilator with BIPAP mode FiO_2 50%, P. inspiration 12, ASB 12, and PEEP 5. Patient was in the condition of sedated by midazolam 5 mg. Then PEEP ventilator is increased to 6 and the others remain. The ventilator arrangement was retained until the eighth day treatment. On the ninth day of treatment, patients were able to breathe spontaneously with GCS 4x6 so that PEEP was lowered to 5.

The 10^{th} day hospitalized, the patient had already been in stable condition. Then conducted evaluation for 2x24 hours and no problems hemodynamics. Finally on the day of the 12^{th} treatment the patient was performed an extubations of the ventilator and transferred to the previous inpatient room. The patient was returned home on the 13^{th} day of treatment with the GCS 456 and stabilize hemodynamic.

The table below is the patient's laboratory examination during hospitalized.

Discussion

Fat embolism syndrome (FES) occurs after trauma and during the orthopedic procedure [1,2]. FES may appear within 24-72 hours after trauma or after the orthopedic procedure [3]. Risk factors of FES include male gender, young age (10-40 years), obesity, multiple fractures, and long bones fractures [4,5]. FES mortality rate reaches 10%-20% [3] with hinghest rate 36%[6] due to an inadequate ventilation exchange process. Therefore, patients with FES requires a ventilator for ventilation support.

Indicators	Value	Normal Value
22 February 2020		
Hemoglobine	10,9	12-16 g/dL
Leukocyte	9,4	4,5-11 x 10^9/L
Hematocrite	31,7	36-46%
Trombosite	154	150-450 x 10^9/L
Albumine	3,7	3,4-4,8 g/dL
Natrium	137,5	135-155 mmol/L
Kalium	3,50	3,5-5 mmol/L
Chlorida	104,1	90-110 mmol/L
Calsium	2,39	2,15-2,57 mmol/L
Kreatinin serum	0,6	0,5-1,1 mg/dL
BUN	11	6-20 mg/dL
Urea	23	12-43 mg/dL
Uric acid	3,2	2,0-5,7 mg/dL
24 February 2020		
Hemoglobine	8,7	12-16 g/dL
Leukocyte	13,5	4,5-11 x 10^9/L
Hematocrite	25,4	36-46%
Trombosite	217	150-450 x 10^9/L
SGOT	69	10-31 U/L (37°C)
SGPT	62	9-36 U/L (37°C)
Albumine	3,6	3,4-4,8 g/dL
Randomise glucose	98	<200 mg/dL
Natrium	138,5	135-155 mmol/L
Kalium	4,69	3,5-5 mmol/L
Chlorida	106, 3	90-110 mmol/L
Calsium	2,26	2,15-2,57 mmol/L
Kreatinin serum	1,1	0,5-1,1 mg/dL
BUN	24	6-20 mg/dL
Urea	51	12-43 mg/dL
25 February 2020		
Hemoglobine	8,6	12-16 g/dL
Leukocyte	33,2	4,5-11 x 10^9/L
Hematocrite	25,8	36-46%
Trombosite	303	150-450 x 10^9/L
Albumine	3,7	3,4-4,8 g/dL
Natrium	139	135-155 mmol/L
Kalium	4,83	3,5-5 mmol/L
Chlorida	107,6	90-110 mmol/L
Calsium	2,43	2,15-2,57 mmol/L
BGA		
Temperature	40,5	37°C
FiO_2	0,61	0,21
Ca	1,17	1,15-1,35 mmol/L
pH	7,28	7,35-7,45
pCO_2	49	35-48 mmHg
pO_2	51	80-100 mmHg
BE	-3,5	-2-3,0
tCO_2	23,4	
HCO_3	22,1	18-23 mmol/L
tHb	5,9	11,7-17,4 g/dL
SO_2	71	95-98%
$AaDO_2$	323	
Na	140	135-145 mmol/L
K	5,2	3,5-5 mmol/L
Laboratory analysis	Possible mixture with venous blood, SO2 < 90%	
26 Februari 2020		
Hemoglobine	10,7	12-16 g/dL
Leukocyte	15,9	4,5-11 x 10^9/L
Hematocrite	31,9	36-46 %
Trombosite	217	150-450 x 10^9/L
BGA		
Temperature	36,6	37°C
FiO_2	0,6 (ventilator)	0,21
Ca	1,07	1,15-1,35 mmol/L
pH	7,36	7,35-7,45
pCO_2	41	35-48 mmHg
pO_2	53	80-100 mmHg
BE	-2,3	-2-3,0
tCO_2	24,5	
HCO_3	23,2	18-23 mmol/L
tHb	7,4	11,7-17,4 g/dL
SO_2	86	95-98%
$AaDO_2$	324	
Na	134	135-145 mmol/L
K	4,7	3,5-5 mmol/L
Laboratory analysis	Possible mixture with venous blood, SO2 < 90%	
27 Februari 2020		
BGA		
Temperature	37,1	37°C
FiO_2	0,50 (ventilator)	0,21
Ca	1,1	1,15-1,35 mmol/L
pH	7,47	7,35-7,45
pCO_2	36	35-48 mmHg
pO_2	138	80-100 mmHg
BE	2,5	-2-3,0
tCO_2	27,3	
HCO_3	26,2	18-23 mmol/L
tHb	9,3	11,7-17,4 g/dL
SO_2	99	95-98%
$AaDO_2$	174	
Na	129	135-145 mmol/L
K	4,1	3,5-5 mmol/L
Laboratory analysis	Alkalosis metabolic with hiponatremi	
SGOT	240	10-31 U/L (37°C)
SGPT	132	9-36 U/L (37°C)
28 Februari 2020		
Kreatinin serum	0,5	0,5-1,1 mg/dL
BUN	18	6-20 mg/dL
Urea	39	12-43 mg/dL
02 Maret 2020		
Hemoglobine	10,9	12-16 g/dL
Leukocyte	16,8	4,5-11 x 10^9/L
Hematocrite	31,4	36-46 %
Trombosite	425	150-450 x 10^9/L
Glucose	118	<200 mg/dL
03 Maret 2020		
Direct bilirubin	3,61	0,2-0,4 mg/dL
Total bilirubin	5,6	<1,2 mg/dL

Table 1: Patient laboratory results during hospitalization.

There are two suggested FES pathophysiology, namely mechanical and biochemical theories. The mechanical theory was first introduced by the Gauss in 1924 which called that trauma and a long bone fracture destroy the fats in the bone marrow and tear down the interosseous veins [1]. This condition causes the fat flakes and bone marrow to enter vein circulation. Debris is later stuck in a pulmo microvasculature causing a mechanical obstruction of the pulmo circulation. The reduction of free fatty acids from the hydrolysis of fat triggers the systemic inflammatory response and induces endothelial pulmo damage. The damage to pulmonary endothelial occurs due to increased capillary leakage and increased adhesion of platelet forming clot in small blood vessels. The presence of intra-cardiac shunts (patent the foramen ovale) or lung pushing fat enters the systemic circulation leading to the manifestation of the brain and skin [2].

Biochemical theories explain that fatty embolism in the lungs leads to the release of localized lipase that breaks down fat into free fatty acids and glycerol. Free fatty acids are toxic to endothelial cells and cause vasogenic edema as well as bleeding. This condition triggers the release of proinflammatory cytokines such as TNF-α, IL-1, and IL-6 which may cause Acute Respiratory Distress Syndrome (ARDS) [3]. In addition, fats in the bone marrow are prothrombotic. The fats are rapidly shrouded by platelet and fibrin and initiate the coagulation cascade to cause thrombocytopenia. In extreme cases can occur Disseminated Intravascular Coagulation (DIC) [1].

FES is diagnosed based on a combination of Gurd Wilson criteria. According to Gurd and Wilson, FES symptoms are divided into major and minor criteria. Major criteria include respiratory disorders, cerebral manifestations, and petekie rash. Minor criteria include pyrexia, tachycardia, retinal changes, jaundice, and renal changes. For diagnosing FES, the patient must have two major criteria or one major criterion followed by four minor criteria [7,8]. The criteria of Gurd and Wilson were enhanced by Lindeque through the assessment of PaO2 > 8 kPa (60 mmHg); PaCO2 > 7.3 kPa (55 mmHg) or pH < 7.3; respiratory rate > 35 x/min [6]. Even after receiving adequate sedation; and increased breathing work assessed through additional muscle respiration use, tachycardia, and anxiety.

Examination that can be done to support clinical diagnosis or to monitor the therapy include (1) Blood Gas Analysis (BGA), (2) hematology and biochemistry, and (3) a plain photo of thorax. Blood Gas Analysis (BGA) shows low O_2 partial pressure (< 60 mmHg) and low CO_2 partial pressure with alkalosis respiratoric [9].

Haematological and biochemical examinations in the acute phase exhibit unclear anemia (70% of cases) and thrombocytopenia (platelet count of < 150,000 in 50% of cases). It can also occur hypocalcemia (because calcium binds to free fatty acids), hypofibrinogenemia, increased ESR and prothrombin time indicating the coagulation abnormality [9].

Some FES patients show a bilateral depiction of fluffy shadows to snow storm appearance on the chest X-ray photo. Some photo sightings have a consolidation of air space due to edema or alveolar hemorrhage. This radiological picture can last for three minggu [8,9].

Management therapy in FES is supportive with airway evaluation, breathing, circulation, disability, and fracture stabilization. A protected, unobstructed, and adequate ventilation airway is crucial to preventing hypoxia. For example the installation of oropharyngeal tube to prevent airway obstruction due to the tongue which blocking oropharyng [10].

Correction of hypoxia can be performed by maintaining normal oxygen pressure through oxygen supplementation or mechanical ventilation and positive end expiratory pressure (PEEP)[4]. Approximately 44% of patients require mechanical ventilation although respiratory function can improve by itself in 3-7 days [11].

Shock condition in FES patients can exacerbate lung lesions so that the recovery of intravascular volume is required with normal saline or albumin solution. Albumin administration not only extends the intravascular volume but also can reduce the level of lung injury as a result of bonding with fatty acid [4].

Vasopresor can be used to maintain haemodinamic [4]. The use of vasoactive drugs can maintain cardiac output and reduce preload. Cardiac dysfunction occurs due to increased pulmonary resistance and shock due to the fatty embolism syndrome which needs to be administered inotropic [12,13].

Conclusion

Fat embolism syndrome is a critical condition that can improve on its own when it gets proper treatment.

Conflict of Interest

There is no conflict of interest related to the materials and methods used in this study.

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Author's Contributions

The authors take part in research design, contributing to data collection, scriptwriting, and all agree to accept the same responsibilities against the accuracy of the content of this article.

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