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RHABDOMYOLYSIS-ASSOCIATED WITH PNEUMONIA AND ACUTE RENAL FAILURE:BIOPSYCHOSOCIAL INTERVENTION

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ABSTRACT

Objective

The overall aim of this report is to highlight and share information and experience with my colleagues: researchers and health clinicians about a rare disease with an explanation of signs and symptoms, appropriate diagnosis, treatment and the outcomes in this case.

KEYWORDS: Rhabdomyolysis, Pneumonia, Acute Renal Failure, Biopsychosocial Intervention.

Introductionbackground

Rhabdomyolysis is a rare condition often self-limiting condition that involves the rapid destruction of muscle tissue that affects the kidneys as muscle protein passes through to the urine. It is a syndrome that responds well to supportive care, but rhabdomyolysis can cause serious complications such as acute kidney injury and compartment syndrome, as well as death. The etiologies, pathophysiology, and complications of rhabdomyolysis are complex and the incidence high enough that health clinicians at all levels of experience would benefit from ongoing review of the research and case studies supporting safe and appropriate care.

Definition

Rhabdomyolysis is a condition characterized by breakdown of skeletal muscles. This leads to the release of toxic intracellular contents into the systemic circulation. It is diagnosed by the presence of significantly elevated levels of creatine kinase (CK), and the excretion of myoglobin in the urine (myoglobinuria), which may impart a cola color to the urine. Rhabdomyolysis was initially described in the victims of war and natural disasters with crush injuries. However, the entity is frequently encountered in patients with a non-traumatic etiology of muscle breakdown.

Chemical agents, especially the use of alcohol and illicit drugs have frequently beenimplicated in triggering rhabdomyolysis.

Rhabdomyolysis is often encountered in the intensive care setting and may have amultifactorial etiology.

A high index of clinical suspicion and prompt initiation of treatment plays a vital rolein the management of these cases.

There are many traumatic and non-traumatic causes of rhabdomyolysis

Traumatic causes include

A crush injury such as from an auto accident, fall, or building collapse

- Long-lasting muscle compression such as that caused by prolonged immobilization after a fall or lying unconscious on a hard surface during illness or while under the influence of alcohol or medication
- Electrical shock injury, lightning strike, or third-degree burn
- Venom from a snake or insect bite
- Non-traumatic causes of rhabdomyolysis include:
- The use of alcohol or illegal drugs such as heroin, cocaine or amphetamines
- Extreme muscle strain, especially in someone who is an untrained athlete; this can happen in elite athletes, too, and it can be more dangerous if there is more muscle mass to break down.
- The use of medications such as antipsychotics or statins, especially when given in high doses
- A very high body temperature (hyperthermia) or heat stroke
- Seizures or delirium tremens
- A metabolic disorder such as diabetic ketoacidosis
- Diseases of the muscles (myopathy) such as congenital muscle enzyme deficiency or Duchene's muscular dystrophy
- Viral infections such as the flu, HIV, or herpes simplex virus
- Bacterial infections leading to toxins in tissues or the

bloodstream (sepsis)

- A previous history of rhabdomyolysis also increases the risk of havingrhabdomyolysis again.

The terms listed below are helpful for understanding rhabdomyolysis

- **Acute kidney injury:** is a sudden decrease in renal function characterized by an increase in serum creatinine and decreased urine output. The prefix rhabdo means rod-shaped and when examined closely, skeletal muscle has a rod-shaped appearance. Myo means of or relating to muscles and lysis is defined as destruction, decomposition.
- **Compartment syndrome:** Increased tissue pressure in a confined anatomical spacethat causes decreased blood flow and ischemia.
- **Creatine kinase (CK):** An intracellular enzyme that helps form adenosine diphosphate and phosphocreatine. There are three isoenzymes of CK; CK1 (BB) isfound primarily in the brain, CK2 (MB) is found primarily in the heart, and CK3 (MM) is found primarily in the muscle.
- **Myoglobin:** The primary intracellular oxygen-transporting molecule.
- **Syndrome:** A set of signs and symptoms that occur together.

Pathophysiology

Rhabdomyolysis occurs due to injury whether it is mechanical, chemical, toxins, poisons, or burns, these injuries have a detrimental effect to the cell membranes throughout the body. When a cell membrane is damaged, the breakdown or lysis releases organic and inorganic intracellular components such as potassium, myoglobin, lactic acid, purines, and phosphate, which enter the circulation. Exhaustive work of cells and stretching can increase sarcoplasmic influx of sodium, chloride, and water, which can result in swelling and auto destruction. After the restoration of blood flow after the injury these components become toxic to the body and in most cases are life threatening, making rhabdomyolysis a medical emergency. "Myoglobin levels rise within hours of muscle damage, but can return to normal in 1-6 hours if continuous muscle injuryis not present.

Myoglobin is usually filtered through glomeruli and reabsorbed in the proximal tubules by endocytosis, however when rhabdomyolysis occurs there is an excess of myoglobin, which overloads the proximal tubule cells ability to convert iron to ferritin, which then results in intracellular ferrihemate accumulation. Since iron can donate and except electrons as well as having the ability to generate free radicals the urine's pH can lead to metabolic acidosis. This process puts oxidative stress and injury to the renal cells, which if untreated can lead to renalcell failure.

When there is an excess of myoglobin the tubules are unable to reabsorb it. Systemic vasoconstriction sets in which results in water reabsorption in renal tubules, which then increases myoglobin concentration in urine. This in turn causes formation of casts that obstruct renal tubules. Another contributing factor of cast formation is apoptosis that occurs in epithelial cells. This obstruction causes formation of free radicals from iron, which can lead to renal failure.

Potassium is another byproduct of muscle lysis. If there is too much potassium in the circulation, then hyperkalemia can occur which is life threatening, because of its cardio toxicity effects, this is a medical emergency. Cardiac arrhythmias can occur due to increased levels of potassium in the blood. In some cases, early death occurs due to ventricular fibrillation.

Calcium accumulation in the muscles occurs in the early stages of rhabdomyolysis. Massive calcification of necrotic muscles can occur which can lead to hypercalcemia. If hyperkalemia is present, hypercalcemia can lead to cardiac arrhythmias, muscular contraction, or seizures.

Diagnosis

The term rhabdomyolysis is used to describe the breakdown or disintegration of striated muscle. Almost independent of the miscellaneous initial events, the pathogenesis follows a common final pathway with intracellular calcium accumulation and the depletion of ATP.

Many clinical features of rhabdomyolysis are nonspecific, and the course of the disease varies depending on the underlying condition. As some complications of rhabdomyolysis can be quite severe (such as hyperkalemia, cardiac arrest and acute renal failure), early recognition and prompt management of this condition are pivotal for a successful outcome.

Clinical presentation

Almost half of all patients with rhabdomyolysis present with the following triad of symptoms: myalgia's, weakness and typical brown-red colored urine due to the presence of myoglobin. A large number of patients presenting with calf pain or muscle swelling have their diagnosis confounded by other conditions, most notably deep venous thrombosis.

Non-specific systemic symptoms, such as fever, abdominal pain, malaise and nausea and may also be observed. Proper assessment of symptoms can be hampered in patients with altered mental status, electrolyte imbalance or uremic encephalopathy. The transaminases tend to be increased, which can lead to the condition being confused with acute liver injury.

Physical examination may reveal signs of dehydration, such as decreased skin turgor, delayed capillary refill and dry mucous membranes. If trauma has occurred, the overlying skin is often bruised or discolored. With the development of a compartment syndrome, the affected

area is painful with specific sensory and motor deficits.

The most common complication is acute renal failure due to acute tubular necrosis as a result of mechanical obstruction by myoglobin (in particular if serum creatine kinase levels are higher than 16.000 IU/l). Mortality rate is approximately 10% and even higher in patients with acute renal failure.

Laboratory findings

First screening of rhabdomyolysis can be performed with a urine dipstick test. The Orth toluidine portion of the dipstick turns blue in the presence of hemoglobin or myoglobin; therefore, it can be used as a surrogate marker for myoglobin if there are no red blood cells in freshly spun sediment of urine.

The most sensitive laboratory test for detecting rhabdomyolysis is serum creatine kinase level. As muscle cells disintegrate and release creatine kinase into plasma, the degree of its elevation shows a direct correlation with the degree of muscle necrosis.

Other muscle markers can also be employed. Aldolase is a glycolytic pathway enzyme that is found in high concentration in skeletal muscle (but also liver and the brain), and together with creatine kinase is highly suggestive of muscle injury. An increase in levels of carbonic anhydrase III is specific for skeletal muscle injury, as it is not present in myocardium.

On the other hand, determining levels of myoglobin (skeletal muscle protein involved in oxidative metabolism) appears to be less sensitive tests for establishing the correct diagnosis, as it rapidly and unpredictably eliminated by hepatic metabolism.

Finally, directed laboratory testing aimed at uncovering the underlying cause of rhabdomyolysis is crucial. Such diagnostic evaluations may include toxicologic testing, bacteriologic and viral tests, genetic analysis, muscle biopsy and forearm ischemic test

Rhabdomyolysis complications

- If not treated early, rhabdomyolysis can lead to complications, such as:
- Very high potassium levels in the blood (hyperkalemia); this can lead to an irregular heartbeat or cardiac arrest
- kidney failure or problems with the liver
- Compartment syndrome; this occurs when too much pressure builds up in the injured compartment groups of muscles (compartment). The extreme pressure hinders blood flow to and from the affected tissues and can cause further muscle damage and death.

Prevention

The easiest way to prevent rhabdomyolysis is to avoid prolonged periods of immobilization, stay hydrated, and exercise within healthy limits.

Other common tips to help prevent the condition include:

- Avoiding weight-loss, muscle gain, or performance-enhancing dietary supplements, especially those containing creatine, ephedrine, ephedra, or high levels of caffeine
- Listening to one's body when exercising and not going beyond what feels comfortable or natural to do seeking training advice if attempting a major physical event (such as running amarathon)
- Increasing workout intensity and frequency slowly
- Treating conditions or complications that are considered to be risk factors
- Seeking immediate medical attention as soon as symptoms occur or if thecondition is suspected

CONCLUSION

Rhabdomyolysis is a frequent condition in patients with acute traumatic SCI admitted to the ICU, and renal dysfunction occurs in half of the cases. Creatinine values should be requested starting at the admission while neither the peak CPK values nor the hemodynamic SOFA scores could be used to properly discriminate between patients with and without renal dysfunction.

Case study

Objective: To describe a case of severe rhabdomyolysis due to muscular dystrophy ina young girl.

Background: A 20 years old female with history of recurrent rhabdomyolysis. She hadfrequent complaints of generalized muscle weakness and dark urine color not related to any exercises.

Differential diagnosis: In the initial presentation, upon physical examination and review of blood work a diagnosis of rhabdomyolysis was made. In the subsequent presentations there was a suspicion for underlying genetic cause of the rhabdomyolysis.

Treatment: Patient receive IV fluid and IV bicarbonate and treated conservatively for the other problems.

Uniqueness: The rhabdomyolysis was due to genetic cause. Also, the massive elevation in muscles enzymes (ex: ck reach 68000) made the case unique.

Case presentation

20 years old, female,156 cm tall, 54 kg weight, she was engaged for only one week, and then she broke up with her fiancée for health reasons, live in north of Gaza strip, she does not have any chronic diseases, no history of hypertension, or diabetes mellitus, she is a product of normal vaginal delivery.

The neonatal period and infant passed smoothly without problem of medical importance, with normal developmental milestone, she was doing well till the age

of 3 years when she suffered from rapidly progressive general weakness associated with decreased oral intake, followed few hours later by impaired consciousness, family described that her child become flaccid at that time no fever chills, cough, cyanosis, diarrhea, vomiting, or abnormal movement or any neurological symptoms.

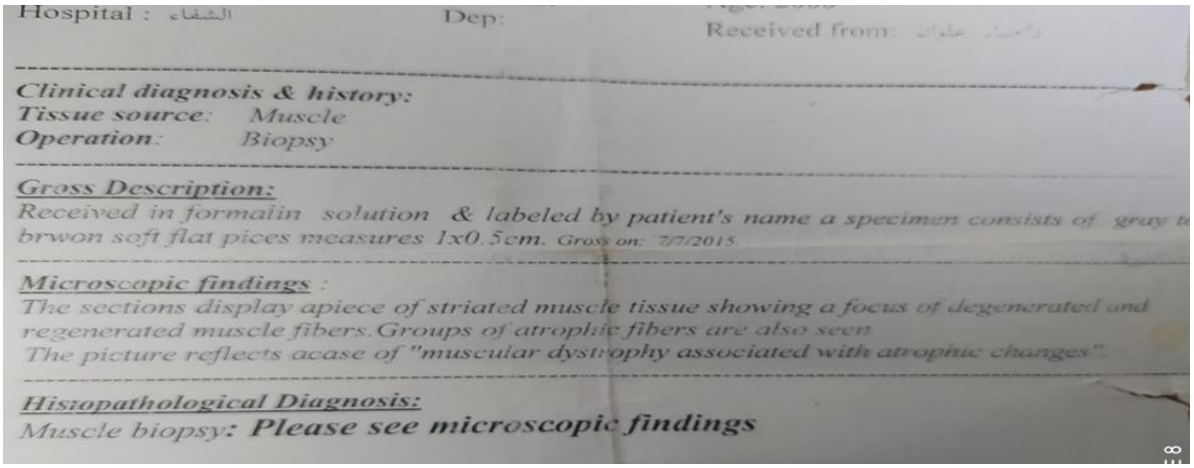
She sought medical help found to have hypoglycemia as the family said, treated for two days at hospital and she was discharged with mild general weakness as decreased usual activity from that time she started to complain of slowly progressive lower limbs weakness, started distal described as inability to control or maintain ankle position during walking, she used to lift her leg higher during walking with history of recurrent tripping but no change in sensation, urine or fecal incontinence.

Based on her strong family history for myopathy, her doctor prescribed ankle stabilizer feet orthosis to use during walking and after few years she underwent surgical intervention to stabilize the ankle join.

After few years mainly at the age of 8 she started to complain of progressive upper limbs weakness mainly the hands, especially after repetitive activity interfering with eating, washing hair and handwriting on paper for more than 3 min.

She also complains of ptosis of the eyes led after prolonged eyes opening ex: during TV watching. at that time her weakness got worsen and involving more proximal muscles as the knees joint move deteriorated, need using help and support on standing from sitting position.

In 2015 muscles biopsy done in AL Shifa pathology department showed focus of degenerated and focus of regenerated muscles, fibers Groups of atrophic fibers are seen. Labeled as muscles dystrophy associated with atrophic changes.


Hospital : الشفاء Dep: Received from:

Clinical diagnosis & history:
Tissue source: Muscle
Operation: Biopsy

Gross Description:
Received in formalin solution & labeled by patient's name a specimen consists of gray t
brwon soft flat pices measures 1x0.5cm. Gross on: 7/7/2015

Microscopic findings :
The sections display apiece of striated muscle tissue showing a focus of degenerated and regenerated muscle fibers.Groups of atrophic fibers are also seen
The picture reflects acase of "muscular dystrophy associated with atrophic changes".

Histopathological Diagnosis:
Muscle biopsy: **Please see microscopic findings**

In the last 4 years, pt. had recurrent admission to hospitals due to dark colored urine, in each time managed by IV fluid.

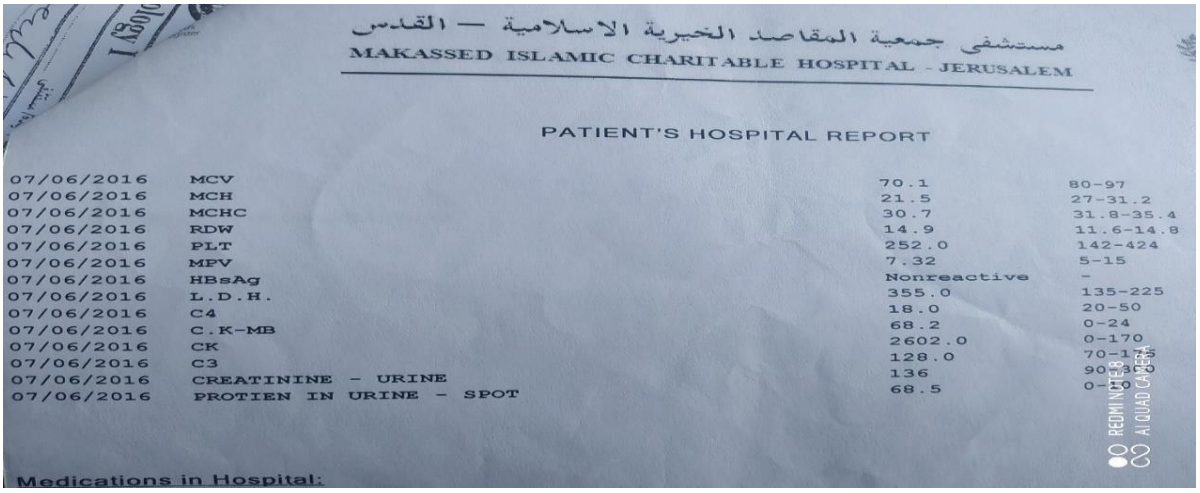
She was referred to Al Makassed Hospital- Jerusalem in 14/6/2016 for further evaluation and possible genetic study; she denied any changes in sensation, no changes in facial expression, vision, speaking, swallowing, and no changes in urine incontinence, fecal incontinence.

Physical examination in al makassed hospital
Conscious and oriented to x3
Upper limp normal tone 5/5 power in shoulder, elbow, hands hyporeflexia
Lower limp normal tone 5/5 power in the hip and knee 4/5, in the ankle dorsiflexion and planter flex, hyporeflexia, down planter.

Almakased hospital workup
Routine labs, chest X- Ray, Urine analysis, high CPK 2026, LDH 1068, abdominal ultrasound showed bilateral relatively small size kidneys.

As her sister died at age 10 due to respiratory depression as a result of carnitine palmitoyl transferase II deficiency (CPT-II). Genetic analysis was done which reveal absence of disease-causing mutations in the exons and exon-intron boundaries of the CPT2.

Note: Carnitine palmitoyl transferase II deficiency (CPT-II) is an autosomal reces+sively inherited genetic metabolic disorder characterized by an enzymatic defect that prevents long-chain fatty acid from being transported into the mitochondria for utilization as an energy sour.

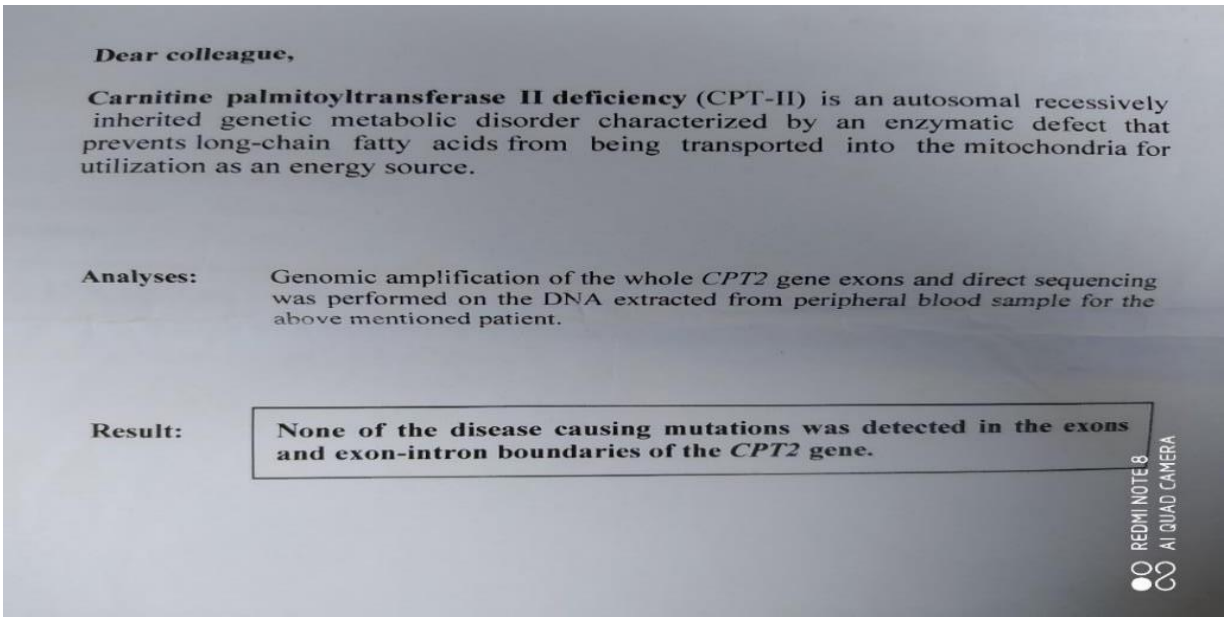


مستشفى جمعية المقاصد الخيرية الاسلامية — القدس
MAKASSED ISLAMIC CHARITABLE HOSPITAL - JERUSALEM

PATIENT'S HOSPITAL REPORT

07/06/2016	MCV	70.1	80-97
07/06/2016	MCH	21.5	27-31.2
07/06/2016	MCHC	30.7	31.8-35.4
07/06/2016	RDW	14.9	11.6-14.8
07/06/2016	PLT	252.0	142-424
07/06/2016	MPV	7.32	5-15
07/06/2016	HBsAg	Nonreactive	-
07/06/2016	L.D.H.	355.0	135-225
07/06/2016	C4	18.0	20-50
07/06/2016	C.K-MB	68.2	0-24
07/06/2016	CK	2602.0	0-170
07/06/2016	C3	128.0	70-175
07/06/2016	CREATININE - URINE	136	90-130
07/06/2016	PROTIEN IN URINE - SPOT	68.5	0-150

Medications in Hospital:

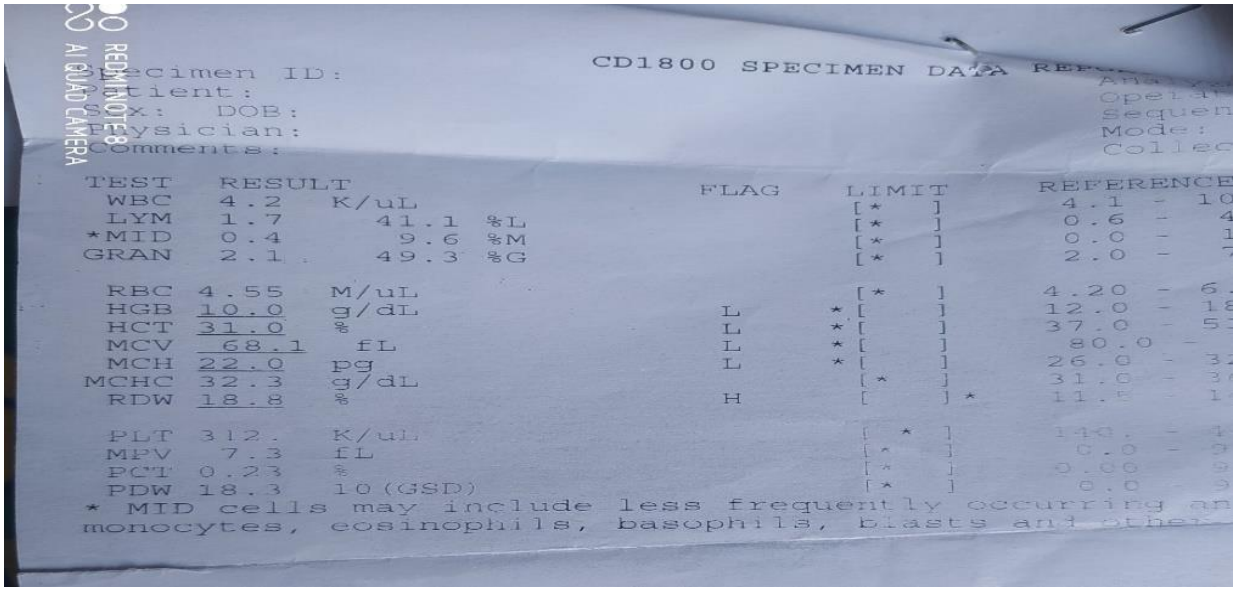


Dear colleague,

Carnitine palmitoyltransferase II deficiency (CPT-II) is an autosomal recessively inherited genetic metabolic disorder characterized by an enzymatic defect that prevents long-chain fatty acids from being transported into the mitochondria for utilization as an energy source.

Analyses: Genomic amplification of the whole *CPT2* gene exons and direct sequencing was performed on the DNA extracted from peripheral blood sample for the above mentioned patient.

Result: **None of the disease causing mutations was detected in the exons and exon-intron boundaries of the *CPT2* gene.**

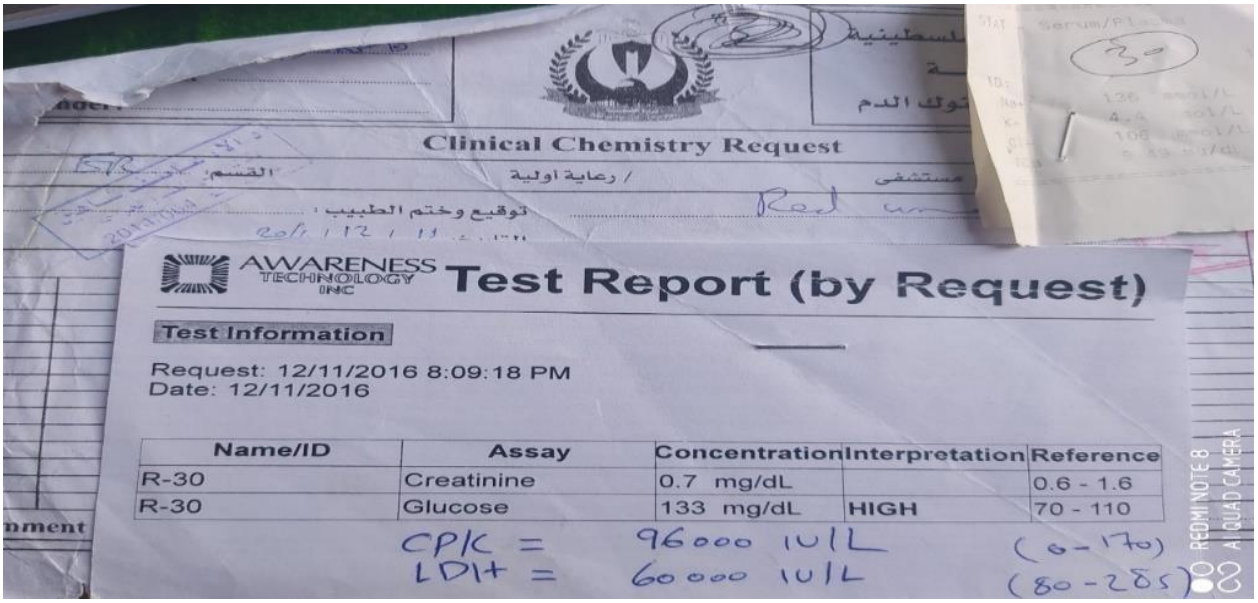


CD1800 SPECIMEN DATA

Specimen ID:
Patient:
Sex: DOB:
Physician:
Comments:

TEST	RESULT		FLAG	LIMIT	REFERENCE
WBC	4.2	K/uL		[*]	4.1 - 10
LYM	1.7	41.1 %L		[*]	0.6 - 4
*MID	0.4	9.6 %M		[*]	0.0 - 1
GRAN	2.1	49.3 %G		[*]	2.0 - 7
RBC	4.55	M/uL		[*]	4.20 - 6.
HGB	10.0	g/dL	L	* [ ]	12.0 - 18
HCT	31.0	%	L	* [ ]	37.0 - 5
MCV	68.1	fL	L	* [ ]	80.0 -
MCH	22.0	pg	L	* [ ]	26.0 - 3
MCHC	32.3	g/dL		[*]	31.0 - 3
RDW	18.8	%	H	[ ] *	11.5 - 1
PLT	312.	K/uL		[*]	140. - 4
MPV	7.3	fL		[*]	0.0 - 9
PCT	0.23	%		[*]	0.00 - 9
PDW	18.3	10(GSD)		[*]	0.0 - 9

* MID cells may include less frequently occurring an
monocytes, eosinophils, basophils, blasts and other



Clinical Chemistry Request

AWARENESS TECHNOLOGY INC

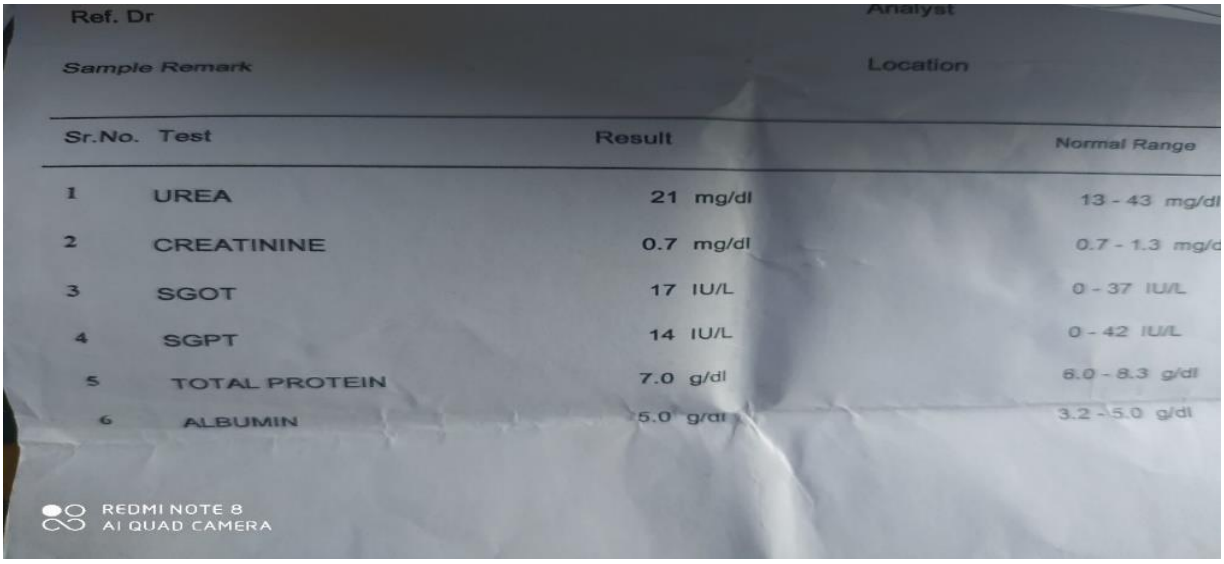
Test Report (by Request)

Test Information

Request: 12/11/2016 8:09:18 PM
Date: 12/11/2016

Name/ID	Assay	Concentration	Interpretation	Reference
R-30	Creatinine	0.7 mg/dL		0.6 - 1.6
R-30	Glucose	133 mg/dL	HIGH	70 - 110

CPK = 96000 IU/L (0-170)
LDH = 60000 IU/L (80-285)



Ref. Dr

Analyst

Sample Remark

Location

Sr.No.	Test	Result	Normal Range
1	UREA	21 mg/dl	13 - 43 mg/dl
2	CREATININE	0.7 mg/dl	0.7 - 1.3 mg/dl
3	SGOT	17 IU/L	0 - 37 IU/L
4	SGPT	14 IU/L	0 - 42 IU/L
5	TOTAL PROTEIN	7.0 g/dl	6.0 - 8.3 g/dl
6	ALBUMIN	5.0 g/dl	3.2 - 5.0 g/dl

REDMI NOTE 8
AI QUAD CAMERA

The last presentation

Patient presented in 05/06/2020 to the emergency department after 2 days of cough and mild shortness of breathing, vomiting, her oxygen saturation 92%, arterial blood gases normal, p 112 bpm, BP128/90 mmhg, temp 36.5 C, and RR 27 bpm. Routine laboratory tests, x-ray done, IV cannula inserted and the patient was admitted to medical department with a diagnosis of pneumonia and treated with a course of antibiotic and oxygen therapy. Doctor know that patient had rhabdomyolysis disease.

After 1 day in 07/06/2020 patient, the patient condition gets worse and she started to complain of dyspnea so she was admitted to the intensive care unit in Indonesia Hospital. At admission physical examination reveal the following: patient was looking ill, severely distressed, conscious, oriented to time person and place, nocyanosis. Vital signs: HR: 115, BP: 90/50, RR: 28, T: 37, SO2: 98% on oxygen mask. chest: normal bilateral vesicular breath sounds, no crepitation, no wheezes. Abdomen: Soft and lax, heart: normal s1, s2 no murmurs. No leg swelling, no signs of DVT. normal neurological examination G.C.S 15, pupils' equal round and reactive to light. Urine was dark in color, UOP 500cc in 24hr.

In 07/06/2020 at morning investigation done as

- CBC: HGB 9.1g /dl, Platelets count183k/ul, Wbc 9.3 k/ul.
- ABG: PH 7.02, PCO2 61, SO2 87
- Chemistry: Na 145 mEq/L, K 3.9 mEq/L, CI 115 mEq/L, Ca 8mg/dl, total protine5.3g/dl, LDH3449u/l, Albumin2.3g/dl, AST552u/l, ALT390u/l, Glucose 147, CK68149u/l, creatine 1.3mg/dl
- INR 1.5
- X- Ray and CT: right lower lobe consolidation
- Urine analysis done (positive ketone, RBS8-10 and epithelial cell).
- Echocardiogram showed normal functioning heart
- ECG was normal

- Ultrasound reveal normal study

Differential diagnosis

- Drugs & Toxins (drug abuse)
- Direct Muscle Damage (Trauma/crush injury, burns, extreme exercise)
- Muscle Hypoxia (Prolonged immobilization)
- Infections (Influenza)
- Malignant hyperthermia, Temperature (Heat stroke, malignant neurolepticsyndrome)
- Inflammatory Muscle Disease (myositis)
- Metabolic Disorders (Diabetic ketoacidosis, hypothyroidism)
- Genetic Defects (Deficiencies in glycolytic enzymes or lipid metabolism,mitochondrial)
- Guillain- Barrie syndrome

Treatment

- O2mask on 7 L/min.
- Nacl 0.9% at rate of 120ml/hrs
- Rocephin1g/12hrs IV
- Ca-gluconat 10ml/12hrs IV
- Heparin5000u/12hrs SC
- Randin50mg/8hrs PO
- Lazix10mg/8hrs IV
- Acamol 1/2g/8hrs po
- Aerovent+ventoline/6hrs Inh
- In addition to other medications up to pt. needs

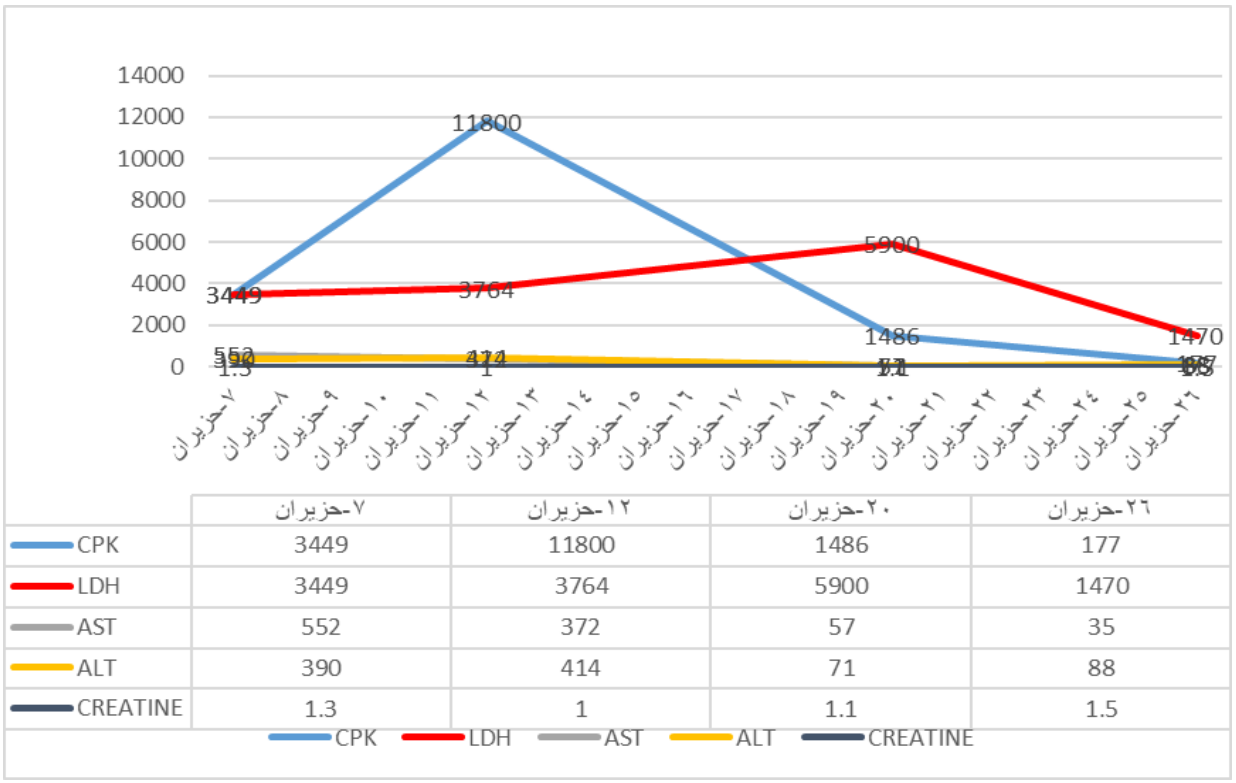
PT became complain of lower, upper limp and abdominal pain but still sensation inleg and arm and I gave her 10mg morphine SC as dr. order.

At 9 PM in same day she was complaining of severe shortness of breathing and usage of accessory muscle of respiration mainly due to infection and progressive weakness of her respiratory muscles, ABG was PH 7.01, PCO2 78, and SO2 87.

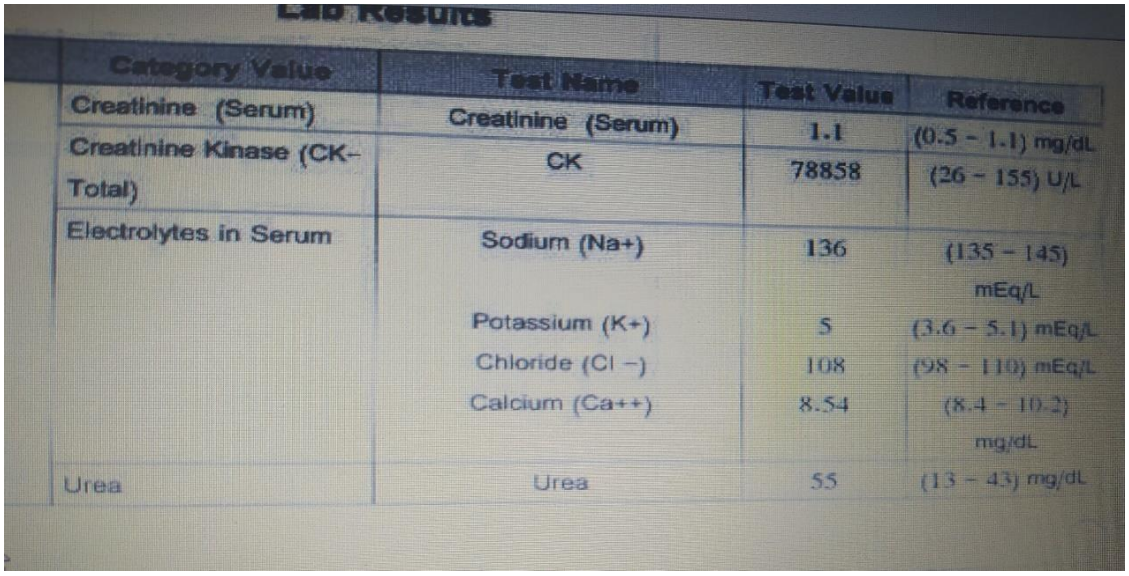
Doctor ordered to put the patient on mechanical ventilator, SIMV mode with full sedation. (0.3mg fentanyl and 30mg dormicom).

Fixed central line in the RT subclavian vein without any complication and measured central venous pressure and it was 3 cmh2o give 1000cc Nacl 0.9%, then measured again and it was 6 cmh2o, the we fixed NGT to give medication and feeding.

In 12/06/2020 PT conscious and oriented, lying flat on MV, CPAP mode, good equal bilateral air entry, abdomen soft and lax, mild bilateral lower limb edema, good UOP, chest X-ray become peter, V/S p 106 bpm, BP 112/70 mmhg, temp 37.7 C still fever, and RR 29 bpm.HGB 9.6g /dl, Platelets count180k/ul, Wbc 6.2 k/ul. ABG: PH 7.32, PCO2 44, SO2 89.
Chemistry: Na 141 mEq/L, K 4.2 mEq/L, CI 113 mEq/L, Ca 7.2mg/dl, total protine5.3g/dl,LDH3764u/l, Albumin2.1g/dl,AST372u/l,ALT414u/l, Glucose 134, CK11800u/l, creatine 1mg/dl, amylase 43u/l,Urea46mg/dl.

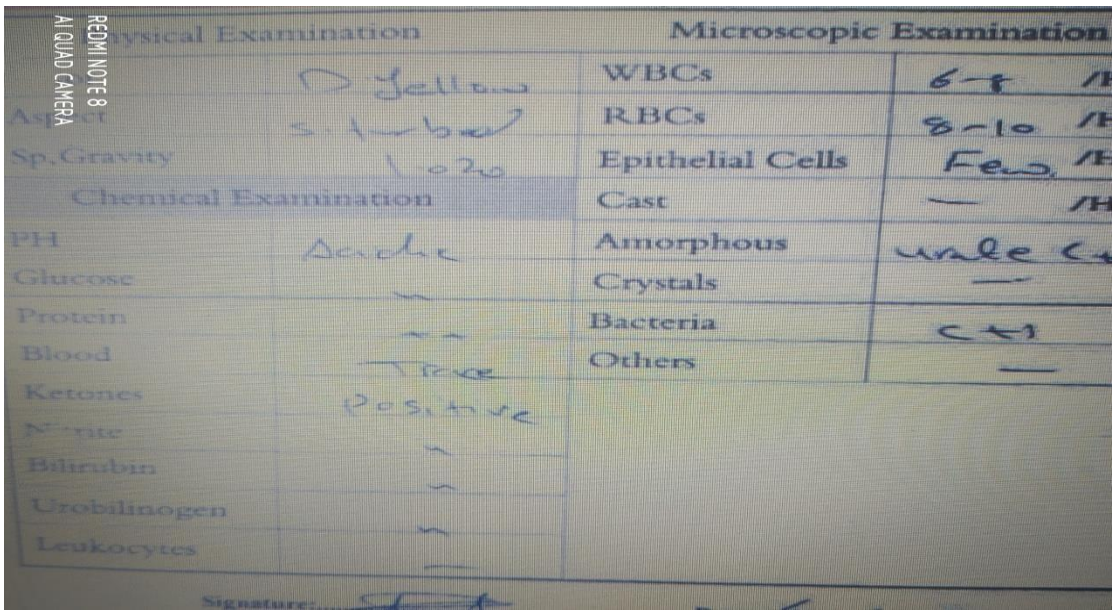


	٧-حزيران	١٢-حزيران	٢٠-حزيران	٢٦-حزيران
CPK	3449	11800	1486	177
LDH	3449	3764	5900	1470
AST	552	372	57	35
ALT	390	414	71	88
CREATINE	1.3	1	1.1	1.5



Lab Results

Category Value	Test Name	Test Value	Reference
Creatinine (Serum)	Creatinine (Serum)	1.1	(0.5 – 1.1) mg/dL
Creatinine Kinase (CK–Total)	CK	78858	(26 – 155) U/L
Electrolytes in Serum	Sodium (Na+)	136	(135 – 145) mEq/L
	Potassium (K+)	5	(3.6 – 5.1) mEq/L
	Chloride (Cl –)	108	(98 – 110) mEq/L
	Calcium (Ca++)	8.54	(8.4 – 10.2) mg/dL
Urea	Urea	55	(13 – 43) mg/dL



REDMI NOTE 8
AI QUAD CAMERA

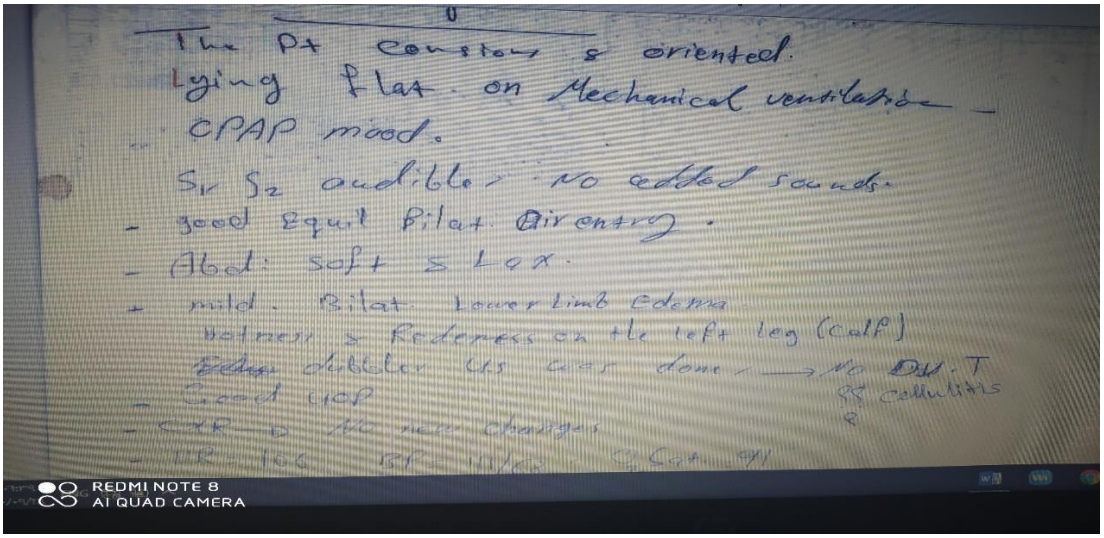
Physical Examination		Microscopic Examination	
Appearance	Yellow Slightly	WBCs	6-8 /H
Sp.Gravity	1.020	RBCs	8-10 /H
Chemical Examination		Epithelial Cells	Few /H
PH	Acidic	Cast	— /H
Glucose	—	Amorphous	urate Cr
Protein	—	Crystals	—
Blood	Trace	Bacteria	Ctl
Ketones	Positive	Others	—
Nitrite	—		
Bilirubin	—		
Urobilinogen	—		
Leukocytes	—		

Signature

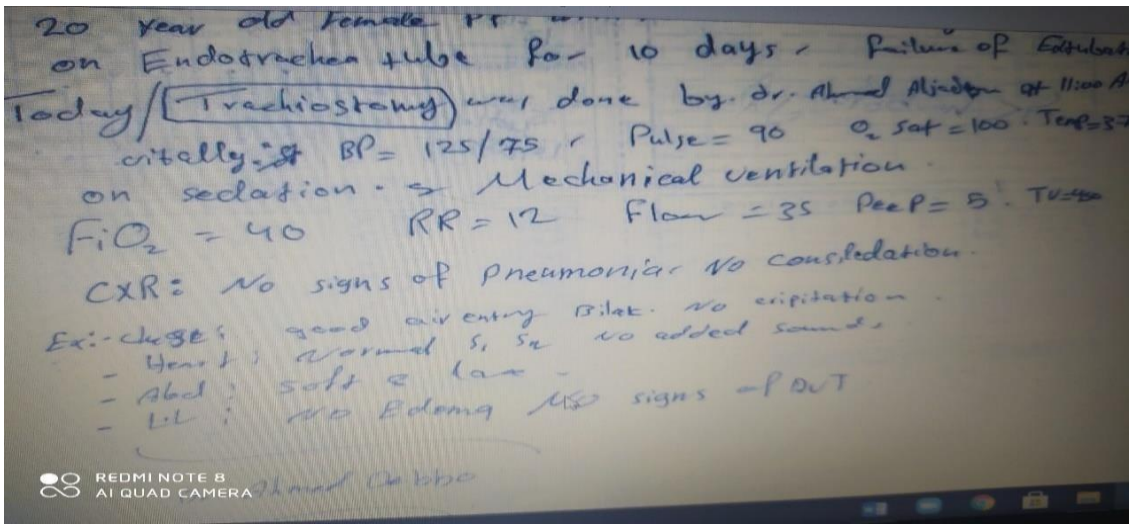
After full examination, extubating done at 2pm but she became tired and in more respiratory distress that led the team to reintubate her due to weakened muscles.

She developed lower limb edema, H Albumin 1.8g/dl, we give albumin 20% /8hrs for 3days then measured 2.7g/dl, edema decreased.

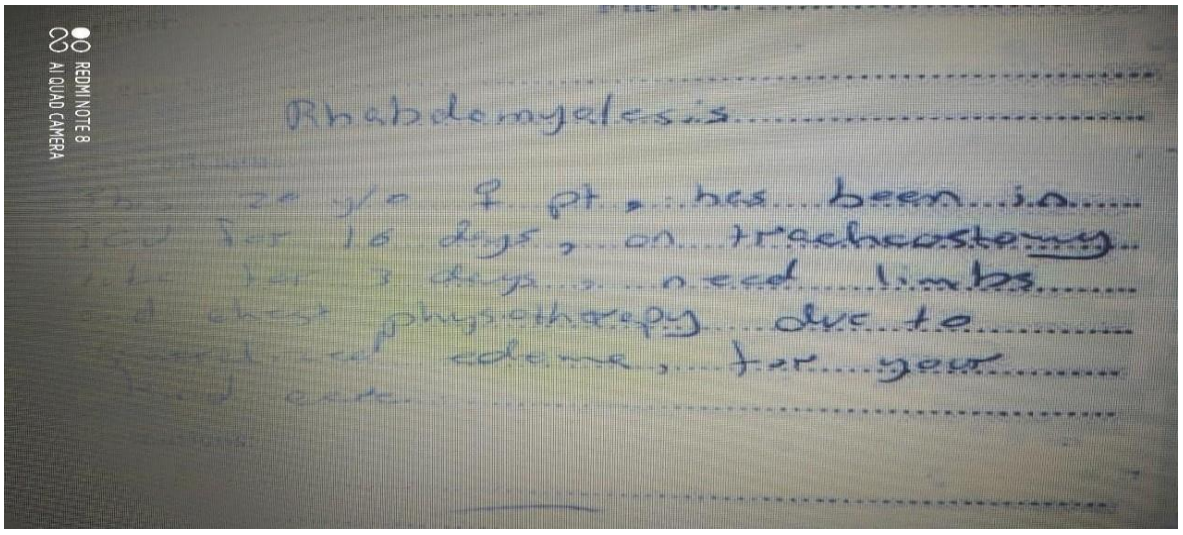
She Still feverish all time, changing folly catheter, central line and sputum culture were done, Positive sputum C/S – pseudomonas spp, sensitive for pipracillin alone Central line C/Positive urine-Sensitive for meropenem and doxycycline negative.

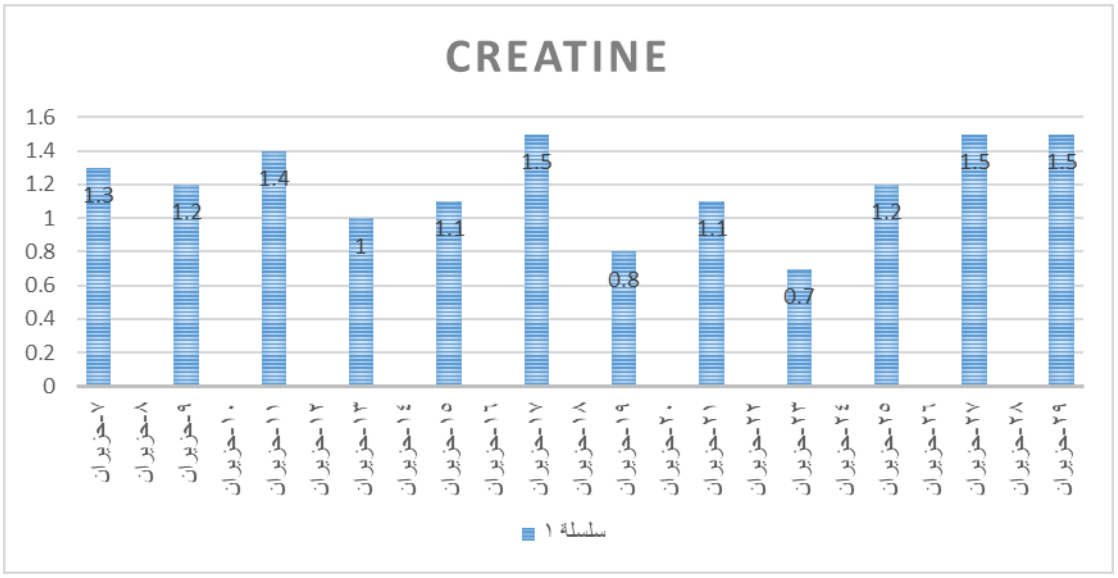


Tracheostomy was done after 10 days, put still on MV with sedation, no bleeding or any complication after tracheostomy.

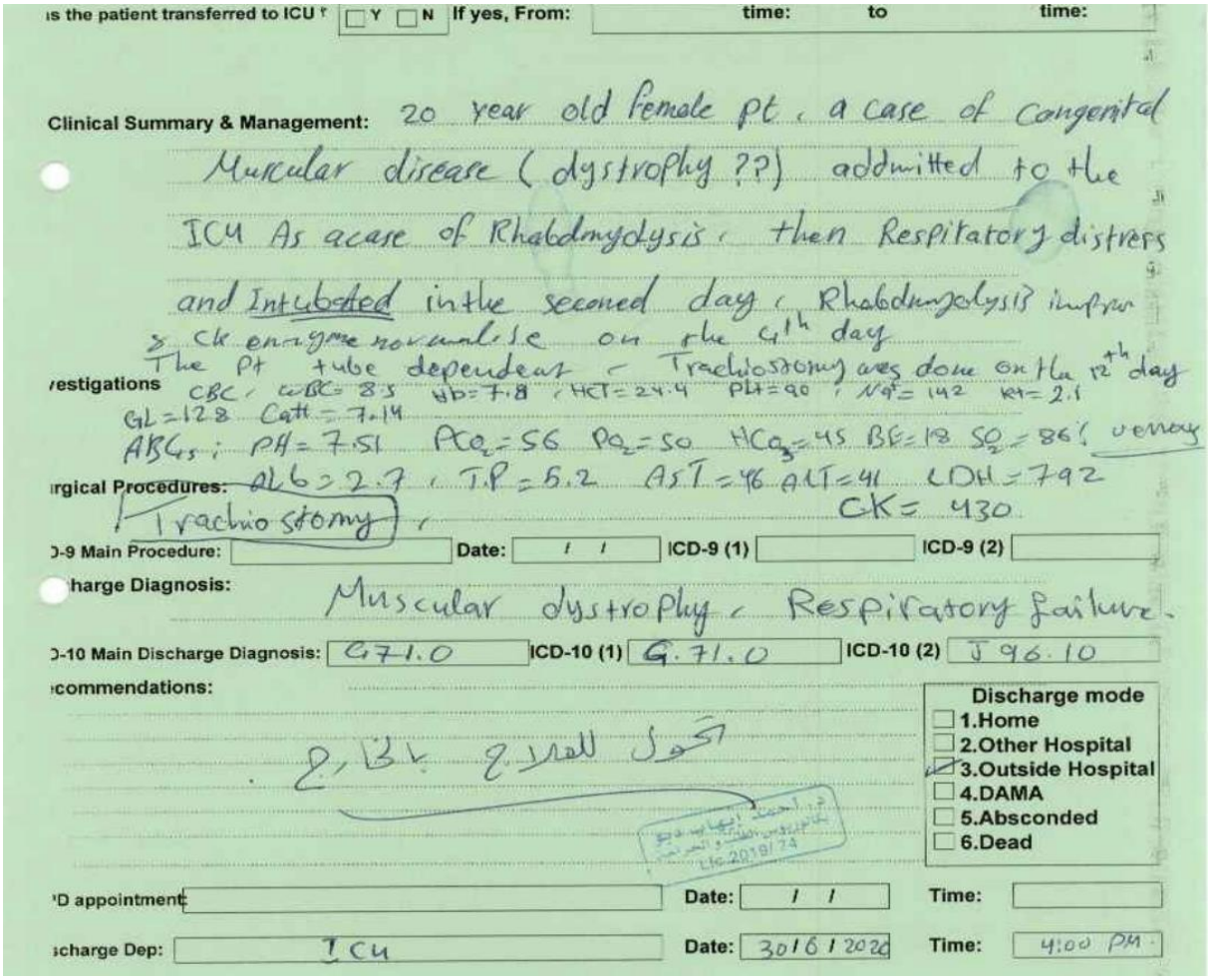


We stop sedation, she was conscious and oriented, unable to do movement of upperand lower limbs and we started physiotherapy to her chest and limbs.





She was referred to Saint Joseph Hospital in 30/06/2020 for more investigations while she still on MV, but she died on the second day of referral.



The ethical and legal dilemmas that faced patient and her family

- Her parents want to have children but are afraid that the children will die whenthey grow up.
- Delaying the insurance of a referral for treatment outside the Gaza Strip, despite the urgent need for treatment.

Psychosocial issue related to the case

The presence of the disease in the girl had an impact on

many psychological factors, such as the parents' involvement in anxiety, extreme tension and frustration, stress of what would happen to their daughter and intense fear of her death, as happened with her sister and ignorance of the consequences.

The patient's psyche was affected by intense sadness and crying when her fiancé did not visit her and separated from her during illness.

The fact that her mother stayed by her side inside the care department after obtaining approval and aimed to alleviate the psychological effects of her daughter and the feeling of safety, had an effect on the husband and children at home and the feeling ofanxiety and the loss of the mother from her duties at home, which made their social life irregular.

Psychosocial interventions done by emotional support and psychosocial support to deal with this issue to family and patient by team and Psychologist as the next plan

1. Assess psychological and physiological state of the patient.
2. Support the pt. psychological and emotionally and though other closed family members or her close friends.
3. Tell the pt. that an advanced treatment is widely available now so she can restore her good health when she commits with the treatment program.
4. Bring a real example of patients who restored their good health with samediagnosis after treatment.

DISCUSSION

Rhabdomyolysis is a medical emergency that requires high clinical suspicion and an early treatment. The index case highlights that rhabdomyolysis may be multifactorial and may resolve completely with appropriate management.

Rhabdomyolysis has several causes that can be broadly categorized as hypoxic, physical, chemical and biologic. Chemical causes are responsible for most of the cases of rhabdomyolysis. Apart from the intrinsic factors (electrolyte abnormalities), many extrinsic factors (alcohol consumption, psychiatric medications and illicit drug use) are known to cause rhabdomyolysis.

Physical causes of rhabdomyolysis are the most common and can be due to either the external (direct trauma) or internal (voluntary and involuntary) factors. However, several of these factors may contribute. Also, chronic alcohol consumption predisposes an individual to rhabdomyolysis due to electrolyte abnormalities, malnutrition and limited energy stores.

The symptoms of rhabdomyolysis are nonspecific and the diagnosis may be overlooked especially in absence of classical triad of myalgia, muscle weakness and dark colored urine.

Diagnosis is established by elevated CK levels (usually >10 times the upper limit of normal) and myoglobinuria that is attributed to muscle destruction and its release into the circulation. Management of rhabdomyolysis primarily involves preventing further muscle injury, and potential complications (cardiac arrhythmias and acute renal failure). This is especially important when multiple causes of rhabdomyolysis coexist. After eliminating the precipitating cause of rhabdomyolysis, the management is aimedat preventing renal insult. Acute kidney injury may develop in up to 60% patients with rhabdomyolysis. Use of aggressive fluid resuscitation (with isotonic saline), is recommended to prevent further renal damage. The aggressive fluid management is continued till the resolution of rhabdomyolysis as evidenced by falling CK levels or when oliguric AKI precludes further fluid infusion. In those with oliguric or anuric kidney injury, an early renal support therapy with hemodialysis should be considered. Alkalinization of urine and forced diuresis with diuretics or mannitol, are not preferred currently.

In conclusion, rhabdomyolysis is a medical emergency that may have multifactorial etiologies. Identifying and correcting each component is important to prevent further muscle injury and organ failure. Early identification and institution of treatment is the cornerstone to prevent AKI and other serious complications of rhabdomyolysis.

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