e-ISSN: 2980-1400

ICJEM

The Intercontinental Journal of Emergency Medicine

Volume: 1 Issue: 1 Year: 2023

MHA
MEDIHEALTH ACADEMY

EDITOR-IN-CHIEF

ASSOCIATE EDITORS-IN-CHIEF

EDITORS

EDITORIAL BOARD

EDITORIAL BOARD

LANGUAGE EDITOR

STATISTICS EDITOR

The Intercontinental Journal of
Emergency Medicine

EDITORIAL

Dear Colleagues, Our Valuable Readers,

It is with great pleasure that I welcome you to the first issue of our peer-reviewed academic journal named "**Intercontinental Journal of Emergency Medicine (ICJEM)**". As the editor-in-chief, I am thrilled to introduce this exciting new journal dedicated to advancing the field of emergency medicine. This first issue brings together a collection of interesting research and case reports, providing valuable insights and advancements in the field of emergency medicine. Our aim is to promote the dissemination of knowledge and stimulate discussion and collaboration among medical professionals, researchers, and academics. In this issue, we present five compelling articles, covering a range of topics in emergency medicine.

Our first article, "*Investigation of changes in MDH enzyme activity in epilepsy*" explores the potential use of malate dehydrogenase (MDH) as a biomarker for seizure classification. The authors conducted a prospective study on 65 patients with epilepsy and found a statistically significant difference in MDH levels between focal-onset and generalized-onset epilepsy patients. The second article delves into the topic of "*Infectious diseases that can occur after an eearthquake: a review*". The authors discuss the factors that facilitate the development of infections after earthquakes, including lack of clean water, food, hygiene, and problems with city infrastructure. The article highlights the importance of monitoring infectious diseases and implementing appropriate public health measures to prevent epidemics. In the third article, "*Lemmel syndrome: a rare cause of obstructive jaundice (case report)*" the authors present a case of the periampullary duodenal diverticulum (PAD) causing obstructive jaundice. The fourth article, "*Paraplegia and fatal outcome following cervical fracture and dislocation due to blunt trauma: a case study*" presents a rare case of cervical fracture secondary to blunt trauma, leading to death. Finally, the fifth article, "*Case report: Sudden death of a female adolescent due to inferior vena cava thrombosis and pulmonary embolism*" discusses a rare and deadly case of pulmonary embolism in a young patient. The authors urge clinicians to consider the possibility of vena cava inferior thrombosis and pulmonary embolism, even in young patients, and to be prepared to manage such challenging cases.

As we embark on this new journey together, I would like to thank the authors, reviewers, editorial team and publisher for their hard work and dedication in bringing this first issue to fruition. I also would like to thank our readers for their support and interest in the **Intercontinental Journal of Emergency Medicine (ICJEM)**. We look forward to providing you with the latest insights and developments in emergency medicine, and we welcome your feedback and suggestions.

Sincerely,

Umut OCAK, MD
Editor-in-Chief

The Intercontinental Journal of
Emergency Medicine

CONTENTS

Volume: 1 Issue: 1 Year: 2023

ORIGINAL ARTICLE

REVIEW

CASE REPORTS

The Intercontinental Journal of Emergency Medicine

Original Article

DOI: 10.51271/ICJEM-0001

Investigation of changes in MDH enzyme activity in epilepsy

Bahadır Taşlıdere[1], Emre Can Güven[2], Beyza Oflaz Güven[2], Bedia Gülen[3]

[1]Department of Emergency Medicine, Faculty of Medicine, Bezmialem Vakif University, Istanbul, Turkey
[2]Basaksehir Public Hospital, Istanbul, Turkey
[3]Department of Emergency Medicine, Istanbul Medipole University, Istanbul, Turkey

Cite this article: Taşlıdere B, Güven EC, Oflaz Güven B, Gülen B. Investigation of changes in MDH enzyme activity in epilepsy. *Intercont J Emerg Med.* 2023;1(1):1-3.

Corresponding Author: Bahadır Taşlıdere, drbahadir@yahoo.com

Submit Date: 15/03/2023 **Accept Date:** 30/03/2023

ABSTRACT

Aims: Epilepsy is one of the most common neurological diseases. It is classified into three groups: focal onset, generalized onset and unclassifiable. A seizure type is difficult to determine. The malate dehydrogenase (MDH) enzyme has a critical role in the excitability of the brain, and it has been reported that recurrent seizures are seen when its regulation is disturbed. We investigated the contribution of MDH enzyme levels as a biomarker to seizure classification.

Methods: Our study was conducted prospectively by patients who were admitted to the emergency department of our hospital within a six-month period. Included in the study were 65 patients diagnosed with epilepsy, according to the International League Against Epilepsy (ILAE) classification. The patients were divided into two groups according to seizure type: focal onset and generalized onset. They were compared in terms of enzyme activity.

Results: The MDH values of the patients with primary epilepsy were found to be 105.94±111.43 and those with secondary epilepsy were 81.03±121.59 (p=0.141). The same test was used to compare the distribution of MDH values between seizure type groups. The MDH value was 47.56±38.65 in patients with focal-onset epilepsy and 109.76±128.44 in patients with generalized-onset epilepsy. A statistically significant difference was observed in the comparisons between the groups (p= 0.031)

Conclusion: Our results reveal the potential of MDH as a biomarker that can be used in epilepsy. In addition, the statistically significant difference between focal-onset and generalized-onset epilepsy indicates that it can be a usable biomarker in seizure classification. MDH enzyme level has a cut-off value of 109.76±128.44 IU/L strongly underlines that it should be used in the diagnosis.

Keywords: Epilepsy, malate dehydrogenase, generalized-onset

INTRODUCTION

Epilepsy is one of the most common neurological diseases and affects 1–3% of the general population.[1] Epilepsies with known etiology are called secondary epilepsy, while epilepsies whose causes cannot be determined are called idiopathic or primary epilepsy.[2] According to the new classifications made by the ILAE in 2017, epilepsies are categorized into three groups: focal onset (focal clonic, focal myoclonic, and focal tonic), generalized onset (tonic-colonic, absence), and unclassifiable.[3,4] Determining the seizure type is important for the treatment, follow-up, and prognosis of the disease. There are many mechanisms responsible for the pathogenesis of epilepsy, which result in different clinical manifestations.[5,6] One of them is oxidative stress and neurodegeneration.[7]

MDH is an enzyme involved in the mitochondrial tricarboxylic acid cycle, and oxidative stress can cause the enzyme activity to change. MDH has a critical role in brain excitability (encoded by the voltage-gated sodium channel α-subunit type I -SCN1A gene). When this gene regulation is disrupted, recurrent seizures may be observed. It has been shown that this enzyme level is increased in epilepsy patients.[8,9]

Appropriate biomarkers should be used to prevent progression and predict the prognosis of epilepsy, which has a very complex pathophysiology and classification. In particular, the distinction between focal and generalized seizures is important in the treatment and follow-up of patients. In our study, we first investigated the level of MDH in focal-onset and generalized-onset epilepsies. Then, we examined the change in MDH enzyme activity in primary and secondary epilepsy. Finally, we investigated whether changes in MDH enzyme activity could be used as a biomarker in the classification of epilepsy.

METHODS

The study was carried out with the permission of Bezialem Vakıf University Non-invasive Researches Ethics Committee (Date: 19.05.2021, Decision No: 2021/177). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

The study was conducted prospectively by analyzing patients who were admitted to the emergency department of

our hospital during seven-month period between 12/1/2019 and 06/30/2020. Included in the study were 65 patients diagnosed with epilepsy as a result of neuroradiological and electroencephalographic examinations. The patients were informed about their participation in the study, and their written consent was obtained. Excluded from the study were patients who did not give consent (and for whom there was a lack of data) and patients with seizures whose onset could not be determined and were therefore unclassified.

The diagnosis of epilepsy was made by the presence of typical interictal electroencephalogram (EEG) findings and clinical presentations, in accordance with ILAE epilepsy classifications. Excluded from the study were patients with a history of any traumatic brain injury, cerebral ischemia, transient ischemic attack, stroke, or neuroendocrinal tumors; patients with a history of neuropsychiatric procedures; patients with a history of psychoactive or central nervous system depressant drug use; patients with a history of drug or alcohol abuse; pregnant and lactating women; and patients with any hepatic dysfunction. Routine laboratory tests and malate dehydrogenase levels were determined from blood samples taken from the patients. Patients were divided into primary and secondary epilepsy groups. According to the seizure types, they were collected in two groups as focal-onset and generalized-onset seizures. They were then compared in terms of enzyme activity. Before administering any medication, at least 2 cc of study blood was taken into a gel-free biochemistry tube from patients with informed consent. The tubes were centrifuged at 3000/min for 10 minutes and the serums were separated and stored in eppendorf tubes at -80 degrees Celsius until the operating time.

Statistics

Behaviors of quantitative variables were expressed using centralization and measures of variance: Mean±SD. To show the behavioral differences of the group averages; Kruskal-Wallis H Test (number of groups>2) and Mann-Whitney U Test (number of groups=2) were used in cases where the assumptions of normality and homogeneity were not met. Statistical significance was determined as p = 0.05 for all cases. Statistical analyzes were provided with the IBM SPSS (Statistics Package for Social Sciences for Windows, Version 21, Armonk, NY, IBM Corp.) package program.

RESULTS

The mean age of the 65 patients participating in the study was 43.75±19.49 years. Of all the patients, 29 were female (44.6%) and 36 were male (55.4%). The mean age of the women was 46.3 ±19.4 years and the men 41.2±19.4 years. Thirty-five (53.8%) of the patients had primary epilepsy and 30 (46.2%) had secondary epilepsy. There were 16 patients (24.6%) with focal-onset epilepsy and 49 patients (75.4%) with generalized-onset seizures (Table 1).

The Mann–Whitney U test was used to compare the distribution of MDH values between epilepsy type groups. The MDH values of the patients with primary epilepsy were found to be 105.94±111.43 and those with secondary epilepsy were 81.03±121.59. No statistically significant difference was observed in the comparisons between groups (p=0.141).

The same test was used to compare the distribution of MDH values between seizure type groups. The MDH value was 47.56±38.65 in patients with focal-onset epilepsy and 109.76±128.44 in patients with generalized-onset epilepsy. A statistically significant difference was observed in the comparisons between the groups (p= 0.031) (Table 2). The results of routine blood tests taken from the patients are given in Table 3.

Table 2. The relationship between epilepsy and MDH

	MDH		P (m)
	Mean ± SD/	Median (Min–Max)	
Type of epilepsy			0.142
Primary	105.9 ± 101.4	71 (13 - 512)	
Secondary	81.0 ± 91.5	44 (5 - 658)	
Seizure types			0.031
Generalized-onset	109.76 ± 118.4	71 (13 - 658)	
Focal-onset	47.56 ± 38.65	34 (5 - 141)	

MDH: Malate dehydrogenase m=Mann Whitney U Test

Table 3. Laboratory results

Parameters	Unit	Values
MDH	U/L	95.06±116.7
WBC	10^9/L	10.9±4.7
Hemoglobin	gr/dl	13.1 ±2.8
Platelet	10^9/L	260±89.4
BUN	mg/dL	14.8±7.5
Creatinine	µmol/L	0.88±0.45
AST	IU/L	27.2±22.3
ALT	IU/L	26.5±18.2
Sodium	mEq/L	137±3.8
Potassium	mmol/l	4.6±4.3
Calcium	mmol/L	10.6±10.8
Neutrophil	10^9/L	7.6±7.9
Lymphocyte	10^9/L	2.9±1.9

MDH: Malate dehydrogenase, WBC: White blood cell, BUN: Blood Urea Nitrogen, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase

DISCUSSION

It has been shown that the incidence of epilepsy is higher in men than in women, and the male to female ratio varies between 1.42 and 2.12.[10-12] In our study, we found this ratio to be 1.24. The mean age of our patients was 43.75±19.49 years. In other studies, 47% of the patients were found to be 40 years or younger.[13] Epilepsies in which there is no underlying cause that triggered the seizures are called idiopathic or primary epilepsy. Most of the time, this condition is caused by genetic predispositions. According to studies, the most common cause of seizures in adults is stroke (23%). In our study, patients were grouped according to the etiological cause. Thirty-five (53.8%) were classified as primary epilepsy and 30 (46.2%) as secondary epilepsy.

Table 1. Age, gender, epilepsy

Female	Male	Type of epilepsy n (%)		Seizure types n (%)		Age
		Primary epilepsy	Secondary epilepsy	Generalized-onset seizures	Focal-onset epilepsy	Mean ± SD
29 (44.6)	36 (55.4)	35 (53.8)	30 (46.2)	49 (75.4)	16 (24.6)	43.7 ± 19.4

n: number, %: percent

In the latest current classification made by the ILAE, the terms "focal onset," "generalized onset," and "unclassifiable" epilepsy are used. Generalized epilepsies constitute approximately 40% of all epilepsies, and there is no obvious etiology other than genetic predisposition.[14,15] Focal seizures are more common in adults than are generalized seizures. Many studies have reported the frequency of generalized seizures to be 23–35% among all epilepsy syndromes.[16-18] In some studies, this rate goes up to 59%.[19] In our study, generalized-onset seizures were more common than focal seizures. There were 16 patients (24.6%) with focal-onset epilepsy and 49 (75.4%) with generalized-onset seizures. Because it causes unconsciousness more frequently, emergency admission rates are higher in epilepsies with generalized onset.[20]

The relationship between the MDH enzyme and epilepsy has been demonstrated in experimental epilepsy studies.[21,22] In addition, severe neurological disorders have been observed in MDH enzyme mutations in the Krebs cycle.[23] Studies have found 46.6±4.9 international units per liter (IU/L) in MDH control groups. According to the receiver operating characteristic (ROC) analysis, the cut-off value of MDH is 51.2 IU/L.[24] In our study, the MDH value was 47.56±38.65 IU/L in patients with focal-onset epilepsy, while the MDH value was 109.76±128.44 IU/L in patients with generalized-onset epilepsy. A statistically significant difference was observed in the comparison between groups (p= 0.031).

CONCLUSION

The result we obtained showed that MDH—one of the oxidative markers—can be used in epilepsy. Diagnostic difficulties in distinguishing between focal seizures and generalized seizures may ultimately cause serious difficulties in the follow-up and treatment of patients. A misdiagnosis as focal epilepsy may lead to inappropriate antiepileptic use and even unnecessary epilepsy surgery. The MDH enzyme level can be used as a biomarker instead of separating focal epilepsy from generalized epilepsy based only on semiology.[25] Moreover, the fact that the MDH enzyme level has a cut-off value of 109.76±128.44 IU/L strongly underlines that it should be used in the diagnosis.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Bezialem Vakıf University Non-invasive Researches Ethics Committee (Date: 19.05.2021, Decision No: 2021/177).

Informed Consent: Written informed consent was obtained from all participants who participated in this study.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: This study was supported by the Scientific Research Project Department of Bezmialem Vakif University (TTU-2020-0302).

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Fiest KM, Sauro KM, Wiebe S, et al. Prevalence and incidence of epilepsy: a systematic review and meta-analysis of international studies. *Neurology.* 2017;88(3):296-03.
2. Shorvon S, Walker M. Status epilepticus in idiopathic generalized epilepsy. *Epilepsia.* 2005;46(9):73–9
3. Falco-Walter JJ, Scheffer IE, Fisher RS. The new definition and classification of seizures and epilepsy. *Epilepsy Res.* 2018;139:73–9
4. Fisher RS, Cross JH, French JA, et al. Operational classificationof seizure types by the International League Against Epilepsy: Position Paper of the ILAE Commission for Classification and Terminology. *Epilepsia.* 2017;58:522-30
5. Menon B, Ramalingam K, Kumar RV. Oxidative stress in patients with epilepsy is independent of antiepileptic drugs. *Seizure.* 2012;21(10):780-4
6. Nemade ST, Melinkeri RR. Effect of antiepileptic drugs on antioxidant status in epilepsy. *Curr Neurobiol.* 2010;1(2):109–12
7. Menon B, Ramalingam K, Kumar RV. Oxidative stress in patients with epilepsy is independent of antiepileptic drugs. *Seizure.* 2012;21(10):780–4
8. Shi Q, Gibson GE. Up-regulation of the mitochondrial malate dehydrogenase by oxidative stress is mediated by miR-743a. *J Neurochem.* 2011;118(3):440–8
9. Laur D, Dozieres-Puyravel B, Ilea A, et al. Focal epilepsy due to de novo SCN1A mutation. *Epileptic Disord.* 2021;23(3):459–65
10. Benn EK, Hauser WA, Shih T, et al. Estimating the incidence of first unprovoked seizure and newly diagnosed epilepsy in the low-income urban community of Northern Manhattan, New York City. *Epilepsia.* 2008;49(8):1431–9
11. Hesdorffer DC, Tomson T, Benn E, et al. ILAE Commission on Epidemiology; Subcommission on Mortality. Combined analysis of risk factors for SUDEP. *Epilepsia.* 2011;52(6):1150–9
12. Muralidhar V, Venugopal K. New onset seizures: Etiology and co-relation of clinical features with computerized tomography and electroencephalography. *J Sci Soc.* 2015;42(2):82-87.
13. Chalasani S, Kumar MR. Clinical profile and etiological evaluation of new onset seizures after age 20 years. *IOSR J Dent Med Sci.* 2015;14(2):2279-2861.
14. Kanitkar SA, Gaikwad AN, Kalyan M, et al. Study of seizure disorder in elderly: Etiology, types, EEG and image findings. *Transworld Med J.* 2013;1:24–5
15. Steinlein OK. Genes and mutations in idiopathic epilepsy. *Am J Med Genet.* 2001;106(2):139–45
16. Beghi E. The Epidemiology of Epilepsy. *Neuroepidemiology.* 2020;54(2):185–91
17. Aaberg KM, Suren P, Søraas CL, et al. Seizures, syndromes, and etiologies in childhood epilepsy: The International League Against Epilepsy 1981, 1989, and 2017 classifications used in a population-based cohort. *Epilepsia.* 2017;58(11):1880-1891.
18. Alexandre V Jr, Capovilla G, Fattore C, et al. Characteristics of a large population of patients with refractory epilepsy attending tertiary referral centers in Italy. *Epilepsia.* 2010;51(5):921-925.
19. Hirani MM, Shrivastva S. Clinical profile of new onset seizures in adults. *Indian J Appl Res* 2015;5:19–21.
20. Galizia EC, Faulkner HJ. Seizures and epilepsy in the acute medical setting: presentation and management. *Clin Med (Lond).* 2018;18(5):409–13.
21. Lerche H, Jurkat-Rott K, Lehmann-Horn F. Ion channels and epilepsy. *Am J Med Genet.* 2001;106(2):146–59.
22. Xu X, Guo F, Lv X, et al. Abnormal changes in voltage-gated sodium channels Na(V)1.1, Na(V)1.2, Na(V)1.3, Na(V)1.6 and in calmodulin/calmodulin-dependent protein kinase II, within the brains of spontaneously epileptic rats and tremor rats. *Brain Res Bull.* 2013;96:1–9
23. Ait-El-Mkadem S, Dayem-Quere M, Gusic M, et al. Mutations in MDH2, encoding a krebs cycle enzyme, cause early-onset severe encephalopathy. *Am J Hum Genet.* 2017;100(1):151–9.
24. Selek S, Gul AZ, Atakul N, Meydan S, Sarikaya A, Koktasoglu F, et al. Investigation of serum isocitrate dehydrogenase, malate dehydrogenase and glutamate dehydrogenase activities with related oxidative stress markers in preeclampsia. *Authorea.* 2021;3:1–2.
25. Cheng C, Sirven JI, Ryan DJ, Feyissa AM. Looks can be deceptive: A primary generalized epilepsy mimicking a lesional focal-reflex epilepsy. *Seizure.* 2021;10(91):114–6.

The Intercontinental Journal of Emergency Medicine

Review

DOI: 10.51271/ICJEM-0002

Infectious diseases that can occur after an eearthquake: a review

Ayşegül Tuna

Department of Infectious Diseases and Clinical Microbiology, Faculty of Medicine, Kırıkkale University, Kırıkkale, Turkey

Cite this article: Tuna A. Infectious diseases that can occur after an eearthquake: a review. *Intercont J Emerg Med*. 2023;1(1):4-8.

Corresponding Author: Ayşegül Tuna, draaslan87@gmail.com

Submit Date: 14/03/2023 **Accept Date:** 30/03/2023

ABSTRACT

Due to uncontrolled urbanization and rapid population growth, earthquakes can have major effects on human life, society and economic systems. Infectious diseases can be seen depending on the factors that facilitate the development of infection in the long term after earthquakes. Lack of clean water, food and hygiene are important causes of infectious disease for individuals. Problems in the city's mains systems such as water and electricity are also among the reasons that increase the possibility of infectious disease. Lack of adequate medication may cause delays in treatment. For this reason, the surveillance of infectious diseases, which increased after the earthquake, has an important place in the fight against infectious diseases and epidemics. Earthquakes can lead to an increase in latent infections such as skin and soft tissue infections, gastroenteritis caused by contaminated food and water, respiratory system infections transmitted by droplets, rash diseases, meningitis, and tuberculosis due to limited treatments.

Keywords: Earthquake, infection disease, precaution

INTRODUCTION

Earthquakes are natural events that occur at depths of several kilometers under the earth's crust and can be devastating at the epicenter, however the number of earthquakes that cause damage are very few.[1] The rapid population growth worldwide, in addition to the decrease in rural living areas and an increase in uncontrolled urbanization, has resulted in many people losing their lives in earthquakes, having to migrate to other settlements, infrastructure systems being adversely affected and irreparable economic losses occurring in the country.[2,3] Natural disasters such as earthquakes may cause death by direct destruction and indirect illness For individuals, the stress caused by the earthquake, the lack of hygiene due to the lack of access to clean water and food and sanitization, the lack of clean air due to collapsed buildings, injuries due to being trapped under debris can be considered the most important cause of infection, while electricity, water, damages in gas and sewerage networks and disruption of the supply chain are among the reasons that increase the likelihood of infection.[4,5] Moreover, during natural diseases the loss of medicines used by people, damage to hospitals and health facilities, and the decrease in emergency capacity despite the increase in the number of people in need of emergency intervention also affect medical treatment.[6] Throughout all these unfavourable conditions, the fact that people try to live in camp areas where there is insufficient living space, leads to a problem in their access to uncontaminated food and water, and a susceptibility to infection arises.[2] For this reason, increasing the surveillance of contagious diseases has an important place in the fight against post-earthquake infectious diseases and epidemics.[2]

If we look at all these stages in order, we need to divide the process of infection transmission into 3 parts. The first stage is the period when the individuals affected by the disaster are rescued and the first aid services are mainly carried out. In cases of infections that are found in tissues with impaired integrity, it is important to be careful. This period generally refers to the first 4 days of the process. The second period defines the initial month in which there is an increase in the frequency of infections from contaminated food and water and the incidence of droplet transmitted infections because of communal living. The third period is the period in which infections with a long latent period or incubation period are seen. Endemic diseases and epidemics specific to the disaster area can be seen. This is the period when infections are most common due to large human migrations, crowded living areas, excess stagnant water, difficulty in accessing clean water, inadequate hygiene, malnutrition, inadequate vaccination, and rapid increase in the number of vectors.[7]

In a meta-analysis that was perfromed, gastrointestinal and hepatic infections were shown as the most common infections (163.4 cases per 100,000 people). This was followed by dermal infections (84.5 cases per 100000 persons), respiratory tract infections (9.9 cases per 100000 persons), and central nervous system infections (0.5 cases per 100000 persons).[8] Infections that are encountered following the earthquake are summarized in **Table 1**.

Tablo 1. Post-earthquake infections and their characteristics 14,15

Infections	Transmission	Pathogen	Clinic	Incubation Time
Wound Infections				
Tetanus	Wounds with contaminated with dirt, feces and soil	Clostiridium tetani	Difficulty swallowing, trismus	3-21 days
Skin and soft tissue infections	Bacterial colonization	Gram negative ve Gram positive bacteria	Fever, skin rash, myonecrosis, gangrene	1-14 days
Waterborne infections				
Gastroenteritis	Fecal/oral transmission	V. cholerae, Shigella dysenteriae,	Watery diarrhea, bloody diarrhea, nausea	12 hours -5 days
Leptospirosis		Leptospira spp.	Fever, headache, chills, myalgia	2-28 days
Hepatitis		Hepatitis A and E	Icterus, abdominal pain, nausea, diarrhea	15-50 days
Infections due to communal living spaces				
Viral URT	Droplet and contact	Influenza, COVID-19	Fever, myalgia, respiratory distress	1-14 days
Pneumonia		S. pneumoniae, H. influenza	Fever, cough, tachypnea	1-3 days
Measles		Rubeola	Fever, rash	10-12 days
Menengitis		N. meningitidis	Fever, headache, neck stiffness, altered consciousness	2-10 days
Tuberculosis		Mycobacterium tüberculosis	Cough, night sweats, fever, weight loss	12 weeks-3 years
Vector-borne infections				
Malaria	Mosquito	P. vivax	Fever, chills, myalgia	7-30 days

INFECTIONS LINKED TO IMPAIRED TISSUE INTEGRITY

Aside from the first diseases that come to mind such as tetanus, crush syndrome, which may include the crushing of tissues, edema in the muscles, kidney failure, heart failure, respiratory failure and shock can also be seen, especially due to the time spent under the debris and pressure.[9]

According to the recommendation guide prepared for the management of Crush syndrome, through the venous catheter which is to be opened while still under debris, initiation of isotonic sodium chloride at a rate of 1000 ml for adults and 15-20 ml/kg/hour for children for the first two hours, avoidance of crystalloids containing potassium (Ringer lactate, etc.); at the end of two hours, it is advised to reduce the maintenance dose by half.[10] The presence of infection accompanying Crush syndrome is associated with high mortality and morbidity.[9] In a study, it was shown that the mortality rate was 3 times higher in patients who developed sepsis due to an earthquake related wound.[4] Especially in earthquake victims, 81% of the fasciotomies performed due to compartment syndrome were shown to be infected, which confirms that it is also a risk factor for mortality.[4] The increase in the duration of stay under debris can also be considered a risk factor since it increases the risk of both pathogen exposure and the development of crush syndrome. Cases of organ failure and malnutrition that are seen in disaster survivors may predispose hospitalized patients to healthcare-associated infections.

Due to all these reasons, infection can be seen in 22.8% to 67.2% of the disaster victims. More than half of these infections are in the form of skin and subcutaneous infections seen in the areas of injury.

Especially in cases of crushing and injuries, the presence of necrotic tissue and foreign body causes bacterial colonization and ultimately infection. In a study, wound infections due to resistant Gram-negative bacteria were found most frequently.[9,11]

After the earthquake and tsunami in Indonesia in 2004, after the Pakistan earthquake in 2005, and after the great eastern Japan earthquake in 2011, there was an increase in cases of tetanus due to the lack of vaccination.[2,12]

In injuries related to collapses of infrastructures, a delay in starting care of the wound or immediate closure of the wound, can lead to the dvelopment of deep soft tissue infections. Gas gangrene is a part of the group of vital infections. Therefore, it is recommended to pay attention to proper wound care.

In the case of patients that are followed up in clinics, contact isolation is advised against resistant Gram-negative bacteria until the culture test results are clear.[11,13]

The bone, soft tissue and neurovascular statuses of the wounds should be evaluated, contaminated areas and areas of necrosis should be carefully cleaned, foreign bodies should be removed, and appropriate debridement should be performed.

The primary suture should not be rushed and should only be applied to properly cleaned wounds within the first 6 hours after injury.[11] In other wounds, additional debridements should be performed at the 48th hour of the wound occurrence, and closed once it is concluded that it is clean. Alongside wound care, it is also recommended to use systemic antimicrobials instead of topical antimicrobials as part of treatment.[13] For wounds without significant contamination and before closed fracture operations, a single dose of prophylactic antibiotic is recommended, whereas for open fractures and dirty wounds a 7-day treatment is recommended. Antibiotic prophylaxis is not recommended for injuries with intact skin.[4,11]

Tetanus

Tetanogenous wound; is defined as a star-shaped crushed wound older than 6 hours, deeper than 1 cm, containing a foreign body or necrotic tissue. Injuries with fractures are also risk factors for tetanus. For this reason, when evaluating the wounds of the victims, their vaccination status should also be evaluated. It is recommended to administer both the tetanus vaccine and tetanus immunoglobulin for those injured who have not had 3 doses of vaccine in the past or whose vaccination status is unknown. No additional recommendation is needed for those who have a full dose of vaccine and who have received a booster vaccine in the last five years, but a single dose of the vaccine is recommended for those who have not received a booster vaccine in the last five years.[4,11]

Infections Due To Lack Of Sanitization

In the case of developing countries, 40% of deaths in the shelter camps of disaster survivors are due to gastroenteritis.[14] In poorly planned and crowded camps, there may be an increase in enteritis cases due to the difficulty in the access to clean water as a result of damage to water networks, the lack of tools used to prepare food and cleaning materials such as soap to clean hands.[15] Studies have shown Salmonella enterica serotype paratyphi A, Vibrio cholerae, and norovirus as contributing factors in particular. For this reason, hydration is the most important treatment to be performed outside of health institutions.[2,16]

Following natural disasters, the problem of housing and open water resources causes an increase in contact of rodents such as mice with water, mixing of rodent urine with water, and consumption of food prepared with that water. As a result, there was an increase in leptospirosis cases. It has been reported that there may be an increase in hepatitis E and hepatitis A cases in cases where the sewage system and drinking water have mixed.[2]

Hepatitis A and hepatitis E

Although hepatitis A and E infections are associated with travel to endemic countries, they can lead to epidemics due to faecal contamination of drinking water and lack of sanitization after natural disasters such as earthquakes. The main approach should be supportive treatment. Both infections do not become chronic, but it should be kept in mind that hepatitis E may progress in a fulminant way in pregnant women.[17]

Cholera

It is a disease that is transmitted by consuming drinking water contaminated with faeces and can lead to an epidemic. While mortality is 50% in untreated cases, mortality decreases to 1% in treated patients. Severe cases should be followed up by hospitalization. Oral hydration is recommended in patients with good clinical status. Contamination can be prevented through safe and clean water sources and observance of personal hygiene rules.[18]

Leptospirosis

Spirochetes, which are found in the urine of many animal species including dogs, when mixed with water result in disease in humans. Preventing contact with contaminated water and fighting mice are methods to control the transmission of the disease.[18]

Typhoid

Typhoid is a febrile disease caused by Salmonella typhi, which is faecal-oral transmission as a result of faeces contaminating clean water. Especially after natural disasters such as earthquakes and tsunamis, epidemics can occur due to the decrease in clean water areas, restriction of access to antibiotics, and failure to comply with hygiene rules in public living areas. The most serious complications are bleeding and intestinal perforation.[19]

Infections Caused By Living In Communities

Especially in young children, acute respiratory disease (ARI) and pneumonia are responsible for approximately 20% of deaths. Valley fever and whooping cough are also among the diseases that can be seen following an earthquake.[20] Lack of clean water and personal hygiene, and housing problems leading to close contact between individuals can be considered as causes of this situation.[21]

There is an increase in the frequency of diseases transmitted by other droplets in places where large groups live together where ventilation conditions are poor, and nutrition and shelter conditions are not provided. There is also an increase in cases of rash diseases such as measles and chickenpox, and meningitis due to N. meningitidis.[2,3]

In situations where there is both a lack of hygiene and personal items such as sheets, towels, mattresses and slippers, the incidences of ectoparasites such as scabies increases.[8]

Meningococcal Meningitis

In cases related to N. Meningitidis affecting children, young adults and individuals living in communities, the meningitis agent transmitted by droplets can be seen. If possible, these cases should be isolated and those who encounter these cases should receive chemoprophylaxis with ceftriaxone, rifampicin or ciprofloxacin.[22]

Scabies

In areas where personal hygiene rules are not followed, Sarcoptes scabiei infestation can be seen since people are in close contact to each other for a long duration of time and communal toilets are not cleaned. However, it should be kept in mind that the transmission is not due to short-term contact such as shaking hands, but rather to long-term contact and the use of communal materials.[23]

Measles

It is a rash disease with a high mortality rate, especially in children. At the beginning of the epidemic, a rapid vaccination program should be carried out to cover all individuals in society, including 6-month-old babies. In epidemics that occur during disasters, it is not recommended in practice, as there is not enough space to carry out isolation and quarantine practices in such situations.[18]

Viral Upper Respiratory Infection (URTI)

After the earthquake, people living collectively in areas without proper ventilation conditions, the drinking of contaminated water, as well as inadequate personal hygiene practices are predisposing factors for contracting viral upper respiratory tract infections.[24]

Especially in seasons with a temperature difference between day and night, viral exacerbation may be diagnosed more frequently within a month following the earthquake.[25]

Latent Infections

Occurring in the late period following the disaster; there has also been an increase in tuberculosis cases due to delays in diagnosis due to immigration, disruption in drug supply, or inadequacy of healthcare providers.[8]

Tuberculosis

This spread of this disease is seen due to migration from endemic areas and due to collective living areas. It can be controlled by facilitating access to medication for individuals receiving treatment with directly supervised treatment and the screening of individuals that have been contacted by the disease.[26]

Vector-Associated Infections And Zoonoses

As a result of the increase in the number of mosquitoes, there may be an increase in diseases with high fever such as dengue fever, malaria, and leishmaniasis in endemic areas. For example, one-third of the cases reported in Asia occurred in the Iran earthquake.[20,28] Therefore, necessary precautions should be taken according to the regional and climatic conditions.

Malaria

Malaria is a disease that is transmitted through the bites of Anopheles mosquitoes in endemic areas and is characterized by seizures of chills with accompanying fever. In addition to personal measures such as the use of insecticide-treated bed nets and fly repellent, regional measures such as the prevention of swamp formation and stagnant water should also be taken.[29]

Precautions

Access to clean water, proper toilet and sewer infrastructure, and ensuring food safety can aid in the prevention of Gastroenteritis.[4] Information should be provided on proper hand washing and personal hygiene practices, before and after eating, following sneezing and coughing, information should be given on washing with soap and water or rubbing with alcohol-containing solutions.[30] For drinking water, primarily closed and bottled water should be preferred. Boiled then chilled water or water disinfected with bleach are other alternatives. Three drops of conventional household bleach containing 4% chlorine provides sufficient chlorination for each 1 litre of water, and vegetables and fruits can be washed with this water.[15]

It is recommended that the bodies of those who lost their lives in the earthquake be buried one by one in recommended areas with local guidelines.[30] It is recommended to wear a mask to cover the mouth and nose, especially in closed areas, to use tissue paper when coughing and sneezing if possible, and to maintain a distance of 1-2 meters from people with symptoms of upper respiratory tract infection.[2] In patient care, masks should be used in combination with other measures such as hand hygiene and physical distancing to be effective in preventing the spread of infections and if possible, for caregivers, it is recommend to follow local vaccination guidelines and prioritize vaccines that are most relevant to the specific setting and population.[31] Considering that the standard resources in the affected areas may be insufficient, planning should be made by local authorities, healthcare providers, and communities for the transportation of the necessary medical equipment and materials for infection prevention and treatment.[32] It is recommended to prioritize strengthening existing healthcare systems and building local capacity to respond to disasters and other emergencies.[33]

The correct choice of shelters, their proximity to sources of water, access to clean water and sanitization, removal of garbage and other wastes with appropriate methods, and combating mosquitoes can prevent the spread of diseases that can be transmitted by vectors. In addition to this, it is recommended that pest control is carried out regularly.[34] In order to prevent the spread of zoonotic diseases such as rabies and brucellosis, it is necessary to do animal vaccinations, quarantine zones should be established if necessary, and the gathering of healthy animals and sick animals should be prevented.[30]

CONCLUSION

In order to prevent infectious diseases in the earthquake area, it is necessary to comply with hygiene conditions, give importance to sanitization, use appropriate personal protective equipment, give priority to vaccination and act in line with the recommendations of local public health authorities.

ETHICAL DECLARATIONS

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Leith A, Sharpe J. A. Deep-focus earthquakes and their geological significance. *J Geology.* 1936;44(8):877-917.
2. Kouadio IK, Aljunid S, Kamigaki T, Hammad K, Oshitani H. Infectious diseases following natural disasters: prevention and control measures. *Expert Rev Anti Infect Ther.* 2012;10(1):95-104. doi:10.1586/eri.11.155
3. Suk JE, Vaughan EC, Cook RG, Semenza JC. Natural disasters and infectious disease in Europe: a literature review to identify cascading risk pathways. *Eur J Public Health.* 2020;30(5):928-935. doi:10.1093/eurpub/ckz111
4. Bartels SA, VanRooyen MJ. Medical complications associated with earthquakes [published correction appears in Lancet. 2012 Feb 25;379(9817):712]. *Lancet.* 2012;379(9817):748-757. doi:10.1016/S0140-6736(11)60887-8
5. Berariu R, Fikar C, Gronalt M, Hirsch P. Understanding the impact of cascade effects of natural disasters on disaster relief operations. *Int J Disaster Risk Reduct.* 2015;12:350–6.
6. Pourhosseini SS, Ardalan A, Mehrolhassani MH. Key Aspects of Providing Healthcare Services in Disaster Response Stage. *Iran J Public Health.* 2015;44(1):111-118.
7. Aghababian RV, Teuscher J. Infectious diseases following major disasters. *Ann Emerg Med.* 1992;21(4):362-367. doi:10.1016/s0196-0644(05)82651-4
8. Najafi S, Akahavan Rezayat A, Beyzaei SF, et al. Incidence of infectious diseases after earthquakes: a systematic review and meta-analysis. *Public Health.* 2022;202:131-138. doi:10.1016/j.puhe. 2021.11.005
9. Chen X, Zhong H, Fu P, Hu Z, Qin W, Tao Y. Infections in crush syndrome: a retrospective observational study after the Wenchuan earthquake. *Emerg Med J.* 2011;28(1):14-17.doi:10.1136/emj. 2009.077859
10. Sever MS, Vanholder R; RDRTF of ISN Work Group on Recommendations for the Management of Crush Victims in Mass Disasters. Recommendation for the management of crush victims in mass disasters. *Nephrol Dial Transplant.* 2012;27 Suppl 1:i1-i67. doi:10.1093/ndt/gfs156
11. Wuthisuthimethawee P, Lindquist SJ, Sandler N, et al. Wound management in disaster settings. *World J Surg.* 2015;39(4):842-853. doi:10.1007/s00268-014-2663-3
12. Aoyagi T, Yamada M, Kunishima H, et al. Characteristics of infectious diseases in hospitalized patients during the early phase after the 2011 great East Japan earthquake: pneumonia as a significant reason for hospital care. *Chest.* 2013;143(2):349-356. doi:10.1378/chest.11-3298
13. Hollands M. Facilitating optimal wound care. *World J Surg.* 2015;39(4):854-855. doi:10.1007/ s00268-014-2842-2
14. Connolly MA, Gayer M, Ryan MJ, Salama P, Spiegel P, Heymann DL. Communicable diseases in complex emergencies: impact and challenges. *Lancet.* 2004;364(9449):1974-1983.doi: 10.1016/S0140-6736(04)17481-3
15. Waring SC, Brown BJ. The threat of communicable diseases following natural disasters: a public health response. *Disaster Manag Response.* 2005;3(2):41-47. doi:10.1016/j.dmr.2005.02.003
16. Dube A, Moffatt M, Davison C, Bartels S. Health Outcomes for Children in Haiti Since the 2010 earthquake: a systematic review. *Prehosp Disaster Med.* 2018;33(1):77-88. doi:10.1017/S1049023X 17007105

17. Shrestha AC, Flower RL, Seed CR, et al. Hepatitis E virus seroepidemiology: a post-earthquake study among blood donors in Nepal. *BMC Infect Dis.* 2016;16(1):707. Published 2016. doi:10.1186/s12879-016-2043-8
18. Gökçekuş H, Barlas C, Almuhise M, Eyni N. Doğal ve İnsan Kaynaklı Afetler, Sonuçları ve Afet Yönetimi. İnşaat Mühendisliği Bölümü Yakın Doğu Üniversitesi, Lefkoşa, 2018.
19. Sutiono AB, Qiantori A, Suwa H, Ohta T. Characteristics and risk factors for typhoid fever after the tsunami, earthquake and under normal conditions in Indonesia. *BMC Res Notes.* 2010;3:106. Published 2010 Apr 17. doi:10.1186/1756-0500-3-106
20. Aflatoonian MR, Sharifi I, Poursmaelian S, Hakimi-Parizi M, Ziaali N. The emergence of anthroponotic cutaneous leishmaniasis following the earthquake in southern villages of bam district, southeastern iran, 2010. *J Arthropod Borne Dis.* 2013;7(1):8-14.
21. Kawano T, Hasegawa K, Watase H, Morita H, Yamamura O. Infectious disease frequency among evacuees at shelters after the great eastern Japan earthquake and tsunami: a retrospective study. *Disaster Med Public Health Prep.* 2014;8(1):58-64. doi:10.1017/dmp.2014.15
22. Kartal ED. Sağlık Personelinde Aşılama ve Kemoprofilaksi. *Turkiye Klinikleri J Inf Dis-Special Topics.* 2010;3:54-61.
23. Peyravi M, Ahmadi Marzaleh M, Khorram-Manesh A. An overview of the strengths and challenges related to health on the first 10 days after the large earthquake in the west of Iran, 2017. *Iran J Public Health.* 2019;48(5):963-970.
24. Mavrouli M, Mavroulis S, Lekkas E, Tsakris A. The ımpact of earthquakes on public health: a narrative review of ınfectious diseases in the post-disaster period aiming to disaster risk reduction. *Microorganisms.* 2023;11(2):419. Published 2023 Feb 7. doi:10.3390/microorganisms 11020419
25. Akbari ME, Farshad AA, Asadi-Lari M. The devastation of Bam: an overview of health issues 1 month after the earthquake. *Public Health.* 2004;118(6):403-408. doi:10.1016/j.puhe.2004.05.010
26. T.C. Sağlık Bakanlığı, Halk Sağlığı Genel Müdürlüğü. Tüberküloz Tanı ve Tedavi Rehberi, 2. Baskı Ankara, Mayıs 2019.
27. Feng J, Xia Z, Zhang L, Cheng S, Wang R. Risk Assessment of malaria prevalence in Ludian, Yongshan, and Jinggu Counties, Yunnan Province, After 2014 Earthquake Disaster. *Am J Trop Med Hyg.* 2016;94(3):674-678. doi:10.4269/ajtmh.15-0624
28. Jafari N, Shahsanai A, Memarzadeh M, Loghmani A. Prevention of communicable diseases after disaster: A review. *J Res Med Sci.* 2011;16(7):956-962.
29. Kınıklı S, Cesur S. Afetlerde enfeksiyon kontrol önlemleri. *Uluslararası Modern Sağlık Bilimleri Dergisi.* 2020;1(1): 15-23.
30. Çalışkan C, Özcebe H. Afetlerde Enfeksiyon Hastalıkları Salgınları ve Kontrol Önlemleri. *TAF Preventive Medicine Bulletin,* 2013;12:5.
31. Esen Ş. İzolasyon Önlemleri. *Turkiye Klinikleri J Inf Dis-Special Topics.* 2010;3: 62-6.
32. Tierney KJ. Emergency medical preparedness and response in disasters: The need for interorganizational coordination. *Public Administration Review.* 1985;45:77-84.
33. Rawls CG, Turnquist MA. Pre-positioning of emergency supplies for disaster response. *Transportation Research Part B: Methodological.* 2010;44(4):521-534.
34. Salama P, Spiegel P, Talley L, Waldman R. Lessons learned from complex emergencies over past decade. *Lancet.* 2004;364(9447):1801-1813. doi:10.1016/S0140-6736(04)17405-9

The Intercontinental Journal of
Emergency Medicine

Case Report

DOI: 10.51271/ICJEM-0003

Lemmel syndrome: a rare cause of obstructive jaundice (case report)

Fatma Sinem Hatipoğlu, Afşin İpekci

Department of Emergency Medicine, Cerrahpaşa School of Medicine, İstanbul University-Cerrahpaşa, İstanbul, Turkey

Cite this article: Hatipoğlu FS, İpekci A. Lemmel syndrome: a rare cause of obstructive jaundice (case report). *Intercont J Emerg Med*. 2023;1(1):9-11.

Corresponding Author: Fatma Sinem Hatipoğlu, f.sinemhatipoglu@gmail.com

Submit Date: 10/03/2023 **Accept Date:** 30/03/2023

ABSTRACT

Obstructive jaundice through periampullary duodenal diverticulum (PAD), without gallstones or neoplasms, was first described by Lemmel in 1934 as Lemmel syndrome. PAD can be diagnosed incidentally or suspected in patients with obstructive jaundice and nonspecific abdominal pain. It is a rare and benign condition that can be misdiagnosed as malignancy. Ignoring this, can cause morbidity and mortality. Definite diagnosis is made by Endoscopic Retrograde Cholangiopancreatography (ERCP). An accurate diagnosis is very important to ensure patient management and to avoid complications of delayed care. We present a case who coincidentally was diagnosed with PAD and thus obstructive jaundice.

Keywords: Lemmel sydrome, emergency medicine, obstructive jaundice, endoscopic retrograde cholangiopancreatography

INTRODUCTION

Abdominal pain is one of the chief complaints for presenting at emergency department. Although it is a reflex to associate the pathology with choledocholithiasis or pancreaticobiliary tumors in patients presenting with right upper quadrant pain and obstructive jaundice, a pathophysiology caused by periampullary diverticulum (PAD), which presents with similar signs and symptoms should be considered.[1] In rare occasions that PAD causes mechanical obstruction of the main bile duct from the distal section, it is termed as Lemmel Syndrome.[2] Lemmel syndrome is difficult to diagnose because it mimics other pancreaticobiliary diseases and malignancies.[3] It is an underdiagnosed and underreported syndrome due to the lack of specific pathognomonic symptoms or signs.[2] Duodenal diverticula are more common in adults aged 70's and older, and have a prevalence of 11%-22% in the general population in postmortem studies. It is independent of gender; however, the detection rate varies depending on the imaging modalities.[3,4]

In this case, we present a patient admitted to emergency department with right upper quadrant pain and jaundice. He was coincidentally diagnosed with PAD and developed obstructive jaundice as a result.

CASE

66-year-old male patient is admitted to emergency room with right upper quadrant pain that had been going on for a week. His known diagnoses are hypothyroidism, coronary artery disease, hypertension and hyperlipidemia. He has a history of gastric perforation, a horseshoe kidney operation and bilateral thyroidectomy.

On physical examination, patient's general condition was good, his consciousness was clear, his orientation was cooperative, Glasgow Coma Scale: 15/15. Vital signs were as follows: fever: 36.4, pulse: 79/min, blood pressure: 102/63 mmHg, sPO_2: 98% in room air, respiratory rate: 18/min. Abdomen was distended, no defense/rebound, rectal examination was normal stool smear. Last defecation was 2 days ago, he had flatus. Lung examination was normal, without rale/rhoncus. Posteroanterior chest x-ray was obtained with no pneumo/hemothorax (**Figure 1**). Cardiac auscultation was normal without murmur. Electrocardiography showed only mild incomplete right branch block.

Figure 1. The posteroanterior chest X-ray

Blood tests showed elevated liver enzymes and amylase. Hyperbilirubinemia with direct dominance was present (**Table 1**).

Table 1. Summary of the Patient's Laboratory Values During the Hospital Stay

	Day 1	Day 2	Day 3		After ERCP Day 1	Discharge	Reference Range	Units
WBC	14.4	11.1	10		7.9	7.8	4.3 - 10.3	$10^3/\mu L$
HGB	13.3	11.9	12.5		11.2	11.3	13.6 - 17.2	g/dl
HCT	40.3	36.1	36.7		33.2	33.3	42 - 52	%
Creatinin	1.4	0.99	0.72		0.54	0.92	0.7 - 1.2	mg/dL
Urea	46	58	49		41	37	17 - 49	mg/dL
AST	382	199	447		139	71	< 40	IU/L
ALT	484	323	388		222	122	< 41	IU/L
ALP	637	492	622	ERCP	456	274	40 - 130	U/L
GGT	938	710	490		102	177	< 60	IU/L
Amylase	207	573	346		93	89	28 - 100	U/L
Total Bilirubin	7.85	6.24	6.91		2.44	1.08	0.2 - 1.2	mg/dL
Direct Bilirubin	7.16	5.81	6.66		1.92	1.05	< 0.3	mg/dL
INR	1.17	1.12	1.21		1.12	1.02	0.85 - 1.15	
C-reactive Protein	137	144	95		22	2.3	< 5	mg/L
pH	7.4	7.4	7.4		7.4	7.4	7.35 - 7.45	
Lactat	1.8	1.1	0.9		0.9	1.2	0.5 - 1.6	mmol/L

Imaging was conducted through hepatobiliary ultrasonography, no acute pathology in liver parenchyma was reported. Neither cystic or solid lesions nor perihepatic free fluid were detected. Gallbladder was enlarged, hydropic and 120×44 mm in size with 3 mm wall thickness. Sludge image that levels within the lumen was observed. Sonographically selectable calculus image was not detected in the lumen. No pericholecystic fluid was detected. Intrahepatic biliary tract appeared dilated and measured 4 mm at the distal site. Choledochus, pancreas and midline structures could not be evaluated due to the dense gas shadows. There was not any free fluid in abdominal compartments.

The patient underwent intravenous contrast-enhanced computed tomography (CT). Radiologist's verbal comment suggested a distended gallbladder, dilatation in intra and extra biliary tract, 1 cm diverticulum in duodenum's second segment that presses distally to the choledochus (**Figure 2**).

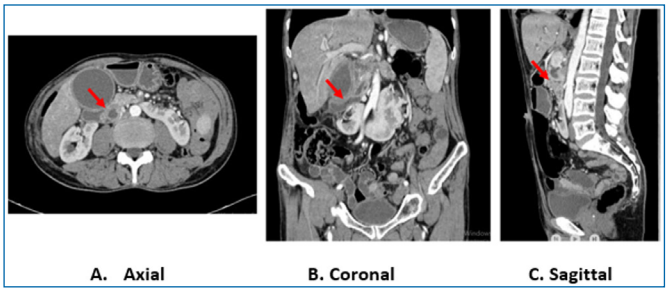

Figure 1. IV contrast-enhanced computed tomography (CT). The PADD is indicated by the red arrow at the A. Axial, B. Coronal, C. Sagital

The patient's oral regimen was stopped. Pantoprazole, metronidazole and saline infusion were administered. General surgery and Gastroenterology consultations were obtained. Patient underwent ERCP by gastroenterology that resulted in malpositioned papilla-sphincter oddi dysfunction, choledochal and intrahepatic biliary tract dilatation. Needle knife sphincterotomy (fistulotomy) and stent insertion into the choledochus duct were performed.

Patient was able to tolerate oral intake. His general condition rapidly improved. After final blood tests were obtained and showed regression in liver enzymes, the patient was discharged (**Table 1**).

DISCUSSION

Duodenum is the most common site of diverticula in digestive system after colon.[5] Duodenal diverticulum (DD) was first described by Chomel in 1710 and documented by Morgagni in 1762, and its first radiological demonstration was performed by JT Case in 1913.[5,6] PAD is an extraluminal diverticulum of duodenal wall located at a distance of 2 to 3 cm from the ampulla vater. Obstructive jaundice due to PAD, which is observed in the absence of gallstones or neoplasms, was first described by Lemmel in 1934 as Lemmel syndrome. Its incidence is 1 - 5% in radiological series, 11-22% in autopsy series.[1,7] The highest incidence is between the ages of 50 and 60, regardless of gender.[5,6,8] Although the physiopathology is not definite and specific, there are three main possible mechanisms:

1. Direct external compression of the common bile duct / ampulla, which leads to blockage,
2. Dysfunction of the sphincter of Oddi,
3. Irritation of ampulla, causing chronic inflammation and eventually leading to papilla fibrosis.[2,10]

A majority of diverticula are asymptomatic.[4,5,7] Even if only 1%-2% of cases may become symptomatic with acute abdominal pain in the form of biliopancreatic colic, our patient right upper quadrant pain that had been going on for a week. Leukocytosis, acute phase reactants increase, liver and/or pancreatic enzyme elevation, bilirubin elevation may be observed at patients' laboratory.[1,5,8] In our case, all laboratory parameters related to obstructive jaundice were elevated, so further examinations for definitive diagnosis were performed.

In addition, in 5% of cases, there may be various complications including:

- Non-pancreaticobiliary complications such as bleeding, diverticulitis, perforation or fistula formation.
- Pancreaticobiliary complications such as acute pancreatitis, cholangitis, bile duct stones or Lemmel Syndrome.

In the absence of choledocholithiasis or any other etiology of obstructive jaundice like in our case, Lemmel syndrome should be considered.[2]

Although ERCP is the gold standard diagnostic test; ultrasound, CT and Magnetic resonance cholangiopancreatography (MRCP) are first line diagnostic methods.

- Ultrasonography (USG) is usually preferred as the first imaging method in biliary obstruction since it shows the expansion of the bile ducts.
- MRCP is preferred when evaluating biliary tract anomalies and eliminating differential diagnosis to confirm the actual diagnosis.
- CT scan with intravenous and/or oral contrast is usually preferred due to its rapid acquisition and availability.
- In our case, we used a USG as a first imaging method, and it was non-diagnostic. Diagnosis of Lemmel syndrome was made by CT scan and confirmed by ERCP.

Since PAD is usually asymptomatic, treatment is only necessary when the patient becomes symptomatic. Its treatment in the literature is still controversial. Diverticulectomy is considered the gold standard treatment method. Nevertheless as a conservative medical treatment in oligosymptomatic patients, it is used in broad-spectrum antibiotic therapy in case of nasogastric decompression and perforation. In addition, endoscopic sphincterotomy or papillary balloon dilatation with ERCP is the first preferred treatment option because it is possible and has a good success rate. However, the recurrence rate is high in alternative treatments (the recurrence rate is 10%-24%). The cannulation rate in ERCP is 94.9%; complications such as bleeding or perforation are 7.8%. In case of unsuccessful endoscopic treatment, a surgical approach is preferred. But these procedures are difficult, their morbidity and mortality are high. Operative mortality is 20%-30%, while morbidity is 30%-40%. That's why perforation, etc. in cases without complications, the first choice is conceptive therapy or ERCP.[2,4-7] In our case, there was an Oddi sphincter defect and sphincteretomy was performed with ERCP.

CONCLUSION

Lemmel syndrome is a difficult diagnosis to establish and can be found by chance or suspected in patients with obstructive jaundice, pancreaticobiliary disease, nonspecific abdominal pain in the absence of stones or tumors like in our case. It is a rare and benign cause of obstructive jaundice. Differential diagnosis is important since it can mimic a malignant neoplasm. Ignoring this possibility, can lead to recurrent jaundice and, in some cases, cholangitis, which is related with high morbidity and mortality. Lemmel Syndrome should be considered in the etiology of obstructive jaundice and accurate diagnosis is essential to ensure proper patient care management and avoid complications of delayed management. Early diagnosis without complications as in our case, endoscopic or conservative approach may be preferred.[1,5,10]

ETHICAL DECLARATIONS

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Bernshteyn M, Rao S, Sharma A, Masood U, Manocha D. Lemmel's Syndrome: Usual Presentation of an Unusual Diagnosis. *Cureus.* 2020;12(4):e7698. doi:10.7759/cureus.7698
2. Aourarh B, Tamzaourte M, Benhamdane A, et al. An Unusual Cause of Biliary Tract Obstruction: Lemmel Syndrome. *Clin Med Insights Case Rep.* 2021;14:11795476211063321. doi:10.1177/11795476211063321
3. Gao AR, Matta A, Seth R, Bande D. Lemmel's syndrome secondary to common bile duct compression by an inflamed duodenal diverticulum. *Cureus.* 2021;13(8):e16959. doi:10.7759/cureus.16959
4. Ergin A, Fersahoğlu MM, Kaya B, Onur E. Lemmel sendromu: duodenal divertiküle bağlı karın ağrısı ve sarılığın nadir bir sebebi. *Haydarpasa Numune Med J.* 2017;57(2):104–106. Doi: 10.14744/hnhj.2017.58077
5. Volpe A, Risi C, Erra M, Cioffi A, Casella V, Fenza G. Lemmel's syndrome due to giant periampullary diverticulum: report of a case. *Radiol Case Rep.* 2021;16(12):3783-3786. doi:10.1016/j.radcr.2021.08.068
6. Alzerwi NAN. Recurrent ascending cholangitis with acute pancreatitis and pancreatic atrophy caused by a juxtapapillary duodenal diverticulum: a case report and literature review. *Medicine (Baltimore).* 2020;99(27):e21111. doi:10.1097/MD.0000000000021111
7. Chavez JCM, Andrade LAM, Ramos ADJ, et al. Cholangitis secondary to Lemmel syndrome. case report. *Arch Clin Gastroenterol.* 2016;2(3):077-079.
8. Bellamlih H, Echchikhi M, El Farouki A, Moatassim Billah N, Nassar I. An unusual cause of obstructive jaundice: Lemmel's syndrome. *BJR Case Rep.* 2020;7(2):20200166. doi:10.1259/bjrcr.20200166
9. Azzam AZ, Alsinan TA, Alrebeh GA, Alhaider T, Alnaqaeb LJ, Amin TM. Lemmel syndrome as a rare cause of prolonged right hypochondrial pain: a case report. *Cureus.* 2021;13(12):e20093. doi:10.7759/cureus.20093
10. Oliveira DM, Correia C, Cunha F, Dias P. A rare cause of abdominal pain with fever. *BMJ Case Rep.* 2019;12(3):e228401. doi:10.1136/bcr-2018-228401

The Intercontinental Journal of
Emergency Medicine

Case Report

DOI: 10.51271/ICJEM-0004

Paraplegia and fatal outcome following cervical fracture and dislocation due to blunt trauma: a case study

Mustafa Alpaslan[1], Necmi Baykan[2]

[1]Department of Emergency Medicine, Nevsehir Public Hospital, Nevşehir, Turkey
[2]Department of Emergency Medicine, Kayseri City Hospital, Kayseri, Turkey

Cite this article: Alpaslan M, Baykan N. Paraplegia and fatal outcome following cervical fracture and dislocation due to blunt trauma: a case study. *Intercont J Emerg Med*. 2023;1(1):12-14.

Corresponding Author: Mustafa Alpaslan, mustafalpaslan@gmail.com

Submit Date: 17/03/2023 **Accept Date:** 30/03/2023

ABSTRACT

Vertebral traumas are most common in the cervical vertebrae. While most intstead of traumas are due to traffic accidents and falling from a height, the rate of cervical fractures secondary to blunt trauma is extremely low. In this case, cervical fracture caused by blunt trauma in the neck region with animal blow and death secondary to paraplegia and other complications that developed afterward were reported. According to the literature, cervical fracture and dislocation are rare traumas with high morbidity and mortality rates. Taking protective measures will always be lifesaving.

Keywords: Emergency medicine, cervical fracture, paraplegia

INTRODUCTION

Vertebral fractures as a result of trauma are common in emergency departments. Early diagnosis and correct treatment methods in the management of patients are very important in reducing the rate of morbidity and mortality. The most common causes of vertebral fractures due to trauma are traffic accidents and falls from a height.[1] Although vertebral traumas are most common in the cervical vertebrae (55%), thoracic, lumbar and lumbosacral traumas are seen at similar frequencies.[2] The rate of fractures and dislocations in the cervical region is less than in other vertebral traumas, and it is essential because the clinical course is more negative.[3] In this case report, sudden paraplegia and complications after cervical vertebral fracture and dislocation due to blunt trauma, which are rare, are mentioned.

CASE

A 54 year old male patient received blunt trauma to the neck region as a result of the startle of the animal during the slaughter of cattle in the morning hours of the feast of sacrifice and then fell to the ground. The patient was brought to the emergency room by ambulance teams due to urinary incontinence and weakness in the legs after neck trauma. Vital signs were examined and blood pressure was 140/80 mmHg, pulse was 96, fever was 36.7°C, and oxygen saturation was 98. On physical examination, the patient was oriented and cooperative. The patient had neck pain and a feeling of weakness and numbness in the lower extremities. There was no response to painful stimuli in the lower extremities. Neurological examination revealed a 3/5 loss of motor strength in the legs and hypoesthesia on sensory examination. Urinary incontinence also occurred during the incident. Laboratory tests and computed tomography (CT) scans were performed on the patient. No pathology was detected in the lower extremities on direct X-ray. Anterolisthesis and facet joint locking were observed at the C6 and C7 levels of the cervical vertebrae (**Picture 1**), (**Picture 2**). Magnetic resonance (MR) imaging of the cervical vertebra region was performed on the patient (**Picture 3**). The patient was evaluated by a neurosurgeon and referral to a higher health institution was planned due to advanced tissue damage in the spinal cord and the development of paraplegia in the patient. During this period, analgesic therapy and intravenous 250 mg methylprednisolone therapy were administered. The clinical course of the patient was followed in another center and it was concluded that he did not benefit from the surgical operation and that he was hospitalized in the intensive care unit for a long time due to respiratory failure and aspiration pneumonia approximately two months after the operation. As a result, the patient developed signs of paraplegia and insufficiency of the diaphragm muscles. Tracheostomy was opened to the patient and muscle strengthening studies were started with physical therapy methods. While the patient was living at home depending on the bed and respirator, he died approximately four months after the trauma.

Figure 1. Cervical fracture transverse section tomography image

Figure 2. Cervical fracture and dislocation sagittal tomography image

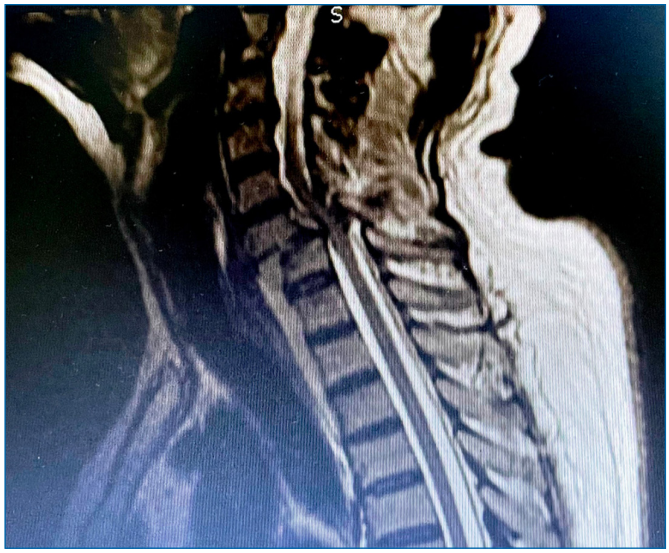

Figure 3. Cervical fracture and spinal cord compression sagittal MR image

DISCUSSION

The region most frequently traumatized in the entire vertebra is between C3-C7, which is defined as the lower cervical region, and the morbidity and mortality rate is high.[4,5] The incidence of fracture or dislocation of the cervical vertebrae secondary to blunt trauma is extremely low.[6] Among blunt traumas, cervical region injuries are around 2-5%, and the annual number of cases is approximately 4-5/100,000.[8] It is usually seen after traumas such as traffic accidents, falling from a height and assault. However, in this case, cervical fracture and spinal cord damage occurred due to the blow received by the cattle, which is very rare. Serious complications may occur in patients with cervical region traumas. For this reason, all regions adjacent to the area of pain from the cranial region should be examined in detail. CT is recommended as the gold standard for scans. MRI should be performed to detect spinal cord tissue damage.[8] Cervical vertebral fractures are most commonly seen in the C5-C7 region.[9] In this case, there were fractures and dislocations at the C6 and C7 levels. Dislocation may accompany in most cases by in cervical vertebral fractures. As in this case, in the study of Ozay et al.[7] dislocation was observed in almost all cases with lower cervical vertebral fractures. As result of studies conducted in cervical spine surgery, decompression and stabilization procedures performed in the first 24 hours have proven that more positive results can be obtained.[11,12] The patient in this case report was referred to a higher health institution after trauma, but the surgical procedure was over 24 hours. Exceeding the recommended ideal time may have reduced the rate of benefit from surgery. There was no significant improvement in neurological functions after surgery in this patient. Similarly, in a study, it was observed that 77,8% of the patients did not improve their neurological functions after the surgical procedure after cervical fracture.[7] Although the effectiveness of methylprednisolone treatment in reducing the neurological symptoms seen in post traumatic patients has not been proven yet, there are opinions suggesting that it will benefit from its anti-inflammatory and antiedema effect.[7] Intravenous 250 mg dexamethasone treatment was administered to this patient.

Spinal cord injury and muscle weakness, urinary and stool incontinence, hemiplegia, paraplegia, and in the future, thromboembolism, infections and atelectasis in the lung may occur in vertebral fractures.[9] In this case, spinal cord injury and subsequent paraplegia and urinary incontinence were observed. In the later period, aspiration pneumonia was observed. Studies show that complications such as pneumonia and meningitis develop frequently in patients after surgical operations in the cervical vertebral region due to trauma, and that the process is prolonged in intensive care follow up and there are fatal cases.[7,12,13]

CONCLUSION

This case is one of the rare cases seen secondary to blunt trauma in cervical vertebral fractures. As in the studies mentioned in the literature, a neurological improvement could not be achieved in this patient, and due to the complications that developed in the advanced stage, the patient first became bedridden and dependent on a ventilator and eventually

died. Although it is not possible to predict when and how the trauma will occur, taking protective measures is always life-saving. In this case, it was observed that the trauma caused by inexperienced, unconscious and careless animal slaughter resulted in mortality.

ETHICAL DECLARATIONS

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Saltzherr TP, Fung Kon Jin PH, Beenen LF, Vandertop WP, Goslings JC. Diagnostic imaging of cervical spine injuries following blunt trauma: a review of the literature and practical guideline. *Injury.* 2009;40(8):795-800. doi:10.1016/j.injury.2009.01.015
2. Looby S, Flanders A. Spine trauma. *Radiol Clin North Am.* 2011;49(1):129-163. doi:10.1016/j.rcl.2010.07.019
3. Heinemann U, Freund M. Diagnostic strategies in spinal trauma. *Eur J Radiol.* 2006;58(1):76-88. doi:10.1016/j.ejrad.2005.12.007
4. Kwon BK, Vaccaro AR, Grauer JN, Fisher CG, Dvorak MF. Subaxial cervical spine trauma. *J Am Acad Orthop Surg.* 2006;14(2):78-89. doi:10.5435/00124635-200602000-00003
5. Vaccaro AR, Hulbert RJ, Patel AA, et al. The subaxial cervical spine injury classification system: a novel approach to recognize the importance of morphology, neurology, and integrity of the disco-ligamentous complex. *Spine (Phila Pa 1976).* 2007;32(21):2365-2374. doi:10.1097/BRS.0b013e3181557b92
6. Eskesen TG, Baekgaard JS, Peponis T, et al. Cervical spinal cord injury after blunt assault: Just a pain in the neck?. *Am J Surg.* 2019;217(4):648-652. doi:10.1016/j.amjsurg.2018.06.022
7. Kwon BK, Vaccaro AR, Grauer JN, Fisher CG, Dvorak MF. Subaxial cervical spine trauma. *J Am Acad Orthop Surg.* 2006;14(2):78-89. doi:10.5435/00124635-200602000-00003
8. Hasler RM, Exadaktylos AK, Bouamra O, et al. Epidemiology and predictors of cervical spine injury in adult major trauma patients: a multicenter cohort study. *J Trauma Acute Care Surg.* 2012;72(4):975-981. doi:10.1097/TA.0b013e31823f5e8e
9. Aebi M. Surgical treatment of upper, middle and lower cervical injuries and non-unions by anterior procedures. *Eur Spine J.* 2010;19 Suppl 1(Suppl 1):S33-S39. doi:10.1007/s00586-009-1120-8
10. B M M, Hegde D, Hussain PS. Management of unstable thoracolumbar spinal fractures by pedicle screws and rods fixation. *J Clin Diagn Res.* 2014;8(2):121-123. doi:10.7860/JCDR/2014/5505.4025
11. Feuchtbaum E, Buchowski J, Zebala L. Subaxial cervical spine trauma. *Curr Rev Musculoskelet Med.* 2016;9(4):496-504. doi:10.1007/s12178-016-9377-0
12. Joaquim AF, Patel AA. Subaxial cervical spine trauma: evaluation and surgical decision-making. *Global Spine J.* 2014;4(1):63-70. doi:10.1055/s-0033-1356764
13. O'Dowd JK. Basic principles of management for cervical spine trauma. *Eur Spine J.* 2010;19 Suppl 1(Suppl 1):S18-S22. doi:10.1007/s00586-009-1118-2

The Intercontinental Journal of Emergency Medicine

Case Report

DOI: 10.51271/ICJEM-0005

Case report: Sudden death of a female adolescent due to inferior vena cava thrombosis and pulmonary embolism

Kazım Ersin Altınsoy[1], Mehmet Göl[2], İbrahim Halil Türkbeyler[3]

[1]Department of Emergency Medicine, Ersin Arslan Education and Research Hospital, Faculty of Medicine, Gaziantep Islam Science and Technology, Gaziantep, Turkey
[2]Department of Physiology, Faculty of Medicine, Gaziantep Islam Science and Technology University, Gaziantep, Turkey
[3]Division of Geriatrics, Department of Internal Medicine, Faculty of Medicine, Gaziantep Islam Science and Technology University, Gaziantep, Turkey

Cite this article: Altınsoy KE, Göl M, Türkbeyler İH. Case report: Sudden death of a female adolescent due to inferior vena cava thrombosis and pulmonary embolism. *Intercont J Emerg Med*. 2023;1(1):15-17.

Corresponding Author: Mehmet Göl, fatih172@gmail.com

Submit Date: 24/03/2023 **Accept Date:** 30/03/2023

ABSTRACT

Despite the fact that considerable advances were seen in diagnosis and treatment of pulmonary embolism since last decades, it is still a seriously mortal condition. Although pulmonary embolism is very rare in children and young people, it can be extremely deadly. Vena cava inferior thrombosis might give rise to pulmonary embolism. Association of vena cava inferior thrombosis and pulmonary embolism might be unthought of particularly in adolescents or young adults, and difficult to manage for the clinicians.

Keywords: Adolescent, pulmonary embolism, vena cava inferior thrombosis.

INTRODUCTION

Pulmonary embolism (PE) due to pulmonary bed obstruction may progress to severe acute right ventricular failure. Contrary to widespread belief, PE is not an uncommon condition. Hospital admissions due to PE was encountered as being 65 per 100.000, in USA, in 2012.[1] On the other hand, PE is reported to be diagnosed in two million cases globally with 3% mortality rate.[2] 5 to 10% of in-hospital deaths are attributed to PE.[3] Non-specific signs and symptoms of PE makes it difficult to diagnose and the clinicians also do not perform an extra effort not to overlook the PE. However, the diagnosis and emergent intervention of PE is of vital significance.

As it is well-known, PE mostly seen in the elderly population. The elderly over 70-year old are at risk of PE as high as 3-fold in comparison to those individuals between the ages of 45 to 69, and the individuals between the ages of 20 to 44 have a 3-fold lower risk of PE than those individuals who are between 45 and 69.[4] PE in adolescents is thought to be very rare, displaying an incidence as much incidence as 1/50 of the elderly population. As for the children, it is found to be between 8.6-57/100.000 in hospitalized children, and 0.14-0.9/100.000 in non-hospitalized children.[5,6]

We sometimes come across with the cases include both vena cava inferior (VCI) thrombosis and PE. Recently, a case is reported, which involves both the inferior vena cava thrombosis and PE simultaneously. However, that report associates of both clinical entities of VCI thrombosis and PE with adverse effects of SARS-CoV-2 vaccine. Accordingly, a 27-year-old young male is reported. He admits to the hospital with chief complaints of cough, hemoptysis and epigastric pain which are reported to begin 10 days later receiving the second dose of mRNA-1273 (Moderna, Cambridge).[7] It is reported that he has no history of trauma or surgical intervention. Also, he and his family are reported to have no history of venous thromboembolism and any coagulation disorder. In another study, a PE case due to VCI thrombosis in a 29-year-old male with chronic pancreatitis is reported.[8]

In this case report, a 17-year-old female adolescent with both PE and VCI thrombosis was presented.

CASE

A 17-year-old female adolescent admitted to the emergency department of Ersin Arslan Training and Research Hospital with acute abdominal pain complaint. Informed consent form was approved by the relatives of the patient. She reported that she had a widespread and vague abdominal pain with a feeling of shortness of breath, since last three to four hours. The patient was not obese and was of medium height. Her medical history revealed neither any surgical intervention nor pregnancy/miscarriage/abortion. The history of mRNA vaccination for SARS-COV-2 pandemic was questioned. The patient was understood to have a sum of four doses vaccination for SARS-CoV-2, firstly two doses of conventional type of vaccine and the two doses of mRNA type of vaccine. It

was revealed that about 10 months had passed since the last mRNA vaccination. According to the knowledge of her past medical history received from relatives, she had never smoked or used alcohol. They could not provide reliable information to exclude the existence of a kind of familial thrombophilia and could not precisely report whether there is venous thromboembolism case or not in their family. When the patient was asked, it was uncovered that she did not ever use oral contraceptives or any other regular medication. Vital signs were as follows: respiration was 18/min, heart rate was 98/min, body temperature was 38°C, blood pressure was 85/55 mmHg, and oxygen saturation was 95%. The hematological parameters were as follows: white blood cell count was 17.6×10^9/L (4-10×10^9/L), neutrophil count was 16.7×10^9/L (2-7×10^9/L), lymphocyte count was 0.5×10^9/L (0.8-4×10^9/L), monocyte count was 0.3×10^9/L (0.2-0.8×10^9/L), eosinophil count was 0.0×10^9/L (0.02-0.5×10^9/L), hemoglobin was 13.4 g/dL (11.5-14.7 g/dL), hematocrit was 39.5% (36.9-49.1%), red blood cell count was 4.35×10^{12}/L (4-6×10^{12}/L), thrombocyte count was 233×10^9/L (150-400×10^9/L), mean corpuscular hemoglobin was 30.8 pg (27-33 pg), mean corpuscular hemoglobin concentration was 33.9 g/dL (32-36 g/dL), red cell distribution width standard deviation was 40.7 fL (36-50 fL), and mean corpuscular volume was 90.9 fL (80-100 fL). According to the arterial blood gas parameters, pH was 7.246 (7.35-7.45), pO_2 was 20.5 mmHg (80-108 mmHg), and pCO_2 was 41.6 mmHg (32-48 mmHg). In biochemical profile, albumin was 43.3 g/L (35-52 g/L), urea was 18.8 mg/dL (17-43 mg/dL), alkaline phosphatase was 69 U/L (30-120 U/L), lipase was 15.8 U/L (0-67 U/L), indirect bilirubin was 0.88 mg/dL (0-1 mg/dL), direct bilirubin was 0.461 mg/dL (0-0.5 mg/dL), creatinine was 0.82 mg/dL (0.5-0.95 mg/dL), glucose was 142.9 mg/dL (70-100 mg/dL), Na+ was 141.2 mmol/L (135-145 mmol/L), K+ was 3.9 mmol/L (3.5-5 mmol/L), Cl- was 104.1 mmol/L (98-109 mmol/L), Ca^{++} was 2.28 mmol/L (2.17-2.51 mmol/L), and C-reactive protein was 6.48 mg/L (0-5 mg/L). D-dimer was 4.27 mg/L (0-0.55 mg/L). Prothrombin time was 14.5 s and active partial thromboplastin time was 36.5 s. Fibrinogen was 160 mg/dL (150-400 mg/dL).

While an abdominal ultrasonography screening was being planned to reveal the etiology of abdominal pain in the emergency department, an urgent computed tomography pulmoner angiogram (CTPA) screening was decided upon encountering a high D-dimer value. No finding was found in the lower extremity Doppler ultrasonography screening. According to the CTPA report, filling defects consistent with acute pulmonary thromboembolism were observed in the segmental and subsegmental branches of both main pulmonary arteries and thrombosis in inferior vena cava (**Figure 1, 2** and **3**). 6. The case was intervened with tissue plasminogen activator (100 mg/2 h).

After the patient was as diagnosed as PE, she was admitted to intensive care unit. The patient was also consulted the thoracic surgery department to get the patient considered for VCI filter insertion or aspiration thrombectomy. However, the blood pressure decreased to 75/50 mmHg, palpitations, and paradoxical pulse was observed. She passed away within ten minutes immediately after supportive care (hydration, oxygenation, ventilation etc.) started in intensive care unit.

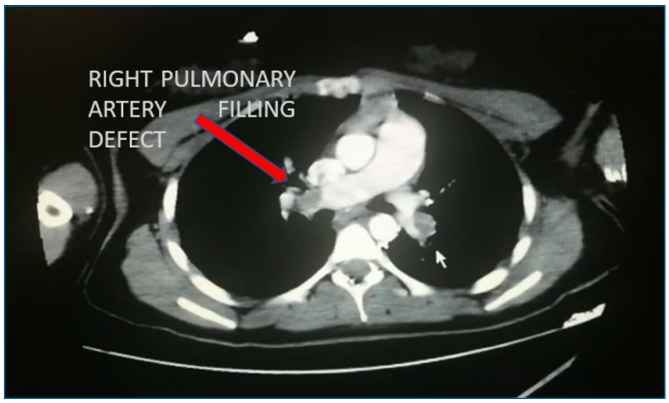

Figure 1: Right pulmonary artery filling defect in computed tomography pulmoner angiogram.

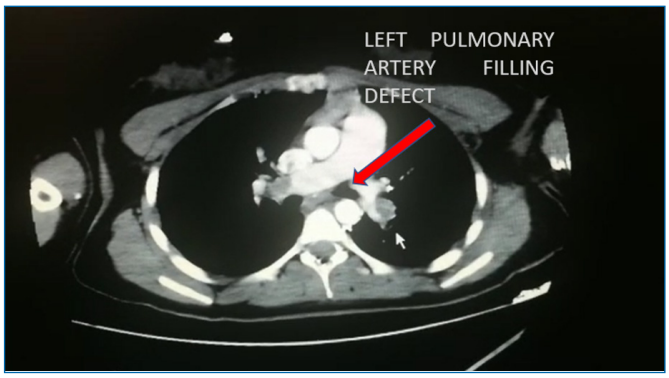

Figure 2: Left pulmonary artery filling defect in computed tomography pulmoner angiogram.

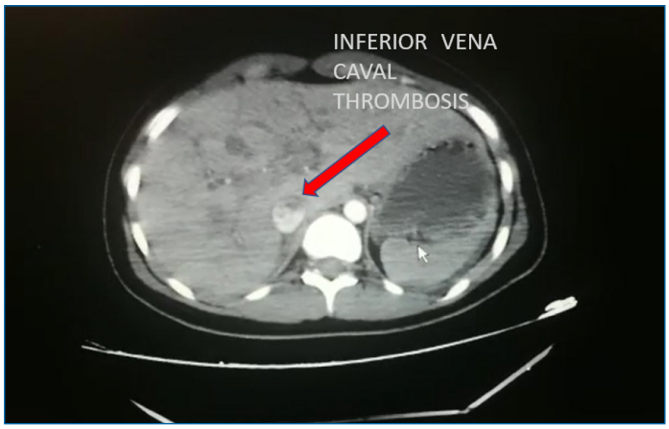

Figure 3: Observation of inferior vena caval thrombosis in computed tomography pulmoner angiogram.

DISCUSSION

According to a recent study, only 9 of 728 acute PE patients were in the adolescent age group, in a period of 16 years of follow-up. Despite the incidence rate of the PE is observed as nearly 1.5 times high in women as it is in men, there seems not to be a significant difference between genders, in the age range of 15 to 20. This is attributed to the less incidence of abortion and oral contraceptive usage, which are well-known contributors of PE, in that range of age.[6] In another study, in which the patients aged between 10 to 17 are 52.2%, most commonly detected risk factors of PE in children are revealed to be central venous catheter (39.2%), malignancy (34.8%), recent surgery (34.8%), infection (30.4%), drugs such as corticosteroids and asparaginase (21.7%), Nephrotic Syndrome (13.0%), congenital heart disease (8.7%), trauma (4.3%), polycythemia (4.3%), and splenectomy (4.3%). Unfortunately, the total number of the patients included in the study is 23, very low in order to be considered sufficient to

rely on the outcomes, like many other studies conducted with children or adolescents PE patients. 16 out of 23 patients are determined to have D-dimer levels above 0.5 mg/L.[9]

Although deep vein thrombosis (DVT) is the most significant one among the causes of PE, it should not be ignored that VCI thrombosis may also result in PE. Along with the certain recent case reports,[7,8] it is revealed that PE is one of leading outcomes among of acute or chronic complications of VCI thrombosis, with a ratio of 30%. It is also stated that the mortality of VCI is twice that of DVT.[10] Unfortunately, rapid loss of the patient in the case we presented did not allow us to carry out further investigations to elucidate the etiology.

CONCLUSION

Although Pulmonary Embolism is a disease that is known to be mostly specific to the elderly, it can cause dramatic results in adolescents as well. VCI thrombosis might simultaneously prompt to PE.

ETHICAL DECLARATIONS

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Smith SB, Geske JB, Kathuria P, et al. Analysis of national trends in admissions for pulmonary embolism. *Chest*. 2016;150(1):35-45. doi:10.1016/j.chest.2016.02.638
2. Al Sayegh F, Almahmeed W, Al Humood S, et al. Global risk profile verification in patients with venous thromboembolism (GRIP VTE) in 5 Gulf countries. *Clin Appl Thromb Hemost*. 2009;15(3):289-296. doi:10.1177/1076029608315168
3. Alikhan R, Peters F, Wilmott R, et al. Fatal pulmonary embolism in hospitalised patients: a necropsy review. *J Clin Pathol*. 2004;57(12):1254-1257. doi:10.1136/jcp.2003.013581
4. Naess IA, Christiansen SC, Romundstad P, et al. Incidence and mortality of venous thrombosis: a population-based study. *J Thromb Haemost*. 2007;5(4):692-699. doi:10.1111/j.1538-7836.2007.02450.x
5. Zaidi AU, Hutchins KK, Rajpurkar M. Pulmonary embolism in children. *Front Pediatr*. 2017;5:170. doi:10.3389/fped.2017.00170
6. Ma QB, Yao WZ, Chen JM, et al. Pulmonary embolism in adolescents. *Chin Med J (Engl)*. 2012;125(6):1089-1094.
7. Ahn EY, Choi H, Sim YS, Shin TR, Kim T. Pulmonary embolism and ınferior vena cava thrombosis in a young male patient after mRNA-1273 (Moderna) immunization: a case report. *Tuberc Respir Dis (Seoul)*. 2022;85(4):361-363. doi:10.4046/trd.2022.0050
8. Hanterdsith B. Fatal Pulmonary Thromboembolism due to inferior vena cava thrombosis. *Ann Vasc Dis*. 2011;4(2):121-123. doi:10.3400/avd.cr.10.00021
9. Lira LAS, Celeste DM, Garanito MP, et al. Pulmonary embolism in pediatrics: A 10-year experience from a tertiary center in Brazil [published online ahead of print, 2022 Apr 2]. *Hematol Transfus Cell Ther*. 2022;S2531-1379(22)00042-6. doi:10.1016/j.htct.2022.02.005
10. Lin HY, Lin CY, Shen MC. Review article inferior vena cava thrombosis: a case series of patients observed in Taiwan and literature review. *Thromb J*. 2021;19(1):43. doi:10.1186/ s12959-021-00296-5