Case Report

Journal of Emergency Medicine Case Reports

Persistent High Fever After Metchloropramide Treatment; Neuroleptic Malignant Syndrome

Fatih Cemal Tekin[1], Can Sezer[1]
[1]Cihanbeyli State Hospital, Emergency Department, Konya, Türkiye

Abstract

Neuroleptic Malignant Syndrome (NMS) is a state neurotransmitter levels fluctuate subsequent to administration of neuroleptic agents. Etiology of NMS is unclear. It's a neurological emergency includes symptoms like altered mental status, rigidity, fever and dysautonomia. Our aim in here is to mention the side effects of metoclopramide, which is commonly used in Emergency Departments (ED). In our case, subsequent to intravenous admission of metoclopramide, patients clinic worsened with NMS symptoms and this was quite unexpected and unwanted for ED doctors. By this case report it is wanted to raise awareness against, side effects of metoclopramide and NMS. Subsequent to intravenous administration of single dose metoclopramide to relieve abdominal pain into 21 years old male with no history of chronical diseases, symptoms of fever, muscle rigidity, confusion and fluctuating blood pressure levels quickly emerged along with leukocytosis and high levels of creatine phosphokinase. ED Doctors suspected NMS. Further laboratory and imaging studies has excluded other central nervous system pathologies and infections. Eventually, patient's clinic got better with symptomatic therapy and he was discharged fully recovered. NMS due to metoclopramide is quite rare, but usage of therapies includes metoclopramide at ED's are quite often. Subsequent to administration of this drug, if patients clinical state worsens with fever, confusion and muscle rigidity, physicians should keep NMS in mind.

Keywords: Adverse reactions, metoclopramide, neuroleptic malignant syndrome

Introduction

Neuroleptic Malignant Syndrome (NMS) is associated with anti-psychotic (neuroleptic) agents. It's a life threating neurologic emergency with altered mental status, muscle rigidity, fever and autonomic dysfunction. Dysautonomic symptoms and systemic complications are direct causes of mortality (1).

NMS, a disease of young and adult males, widely accepted as a situation of the fluctuating neurotransmitter levels subsequent to administration of tranquilizing and anti-psychotic agents but there are also other reasons and it's etiology remains unclear. When associated drugs analyzed, medications (Table 1) formerly known as neuroleptic agents which are first generation of anti-psychotics with high (haloperidol) and low potencies (chlorpromazine), second generation of anti-psychotics (clozapine, risperidone, olanzapine) and anti-emetic drugs (metoclopramide) mostly have blamed as a cause (2, 3).

Patients of NMS may show symptoms and sings of hyperthermia, muscle rigidity, autonomic dysfunction

Table 1: Medications Associated with Neuroleptic Malignant Syndrome

Typical Neuroleptics	Atypical Neuroleptics	Antiemetics	Others	Dopaminergic Agents (withdrawal)
Haloperidol	Clozapine	Droperidol	Tetrabenazine	Levodopa
Chlorpromazine	Risperidone	Domperidone	Reserpine	Amantadine
Fluphenazine	Quetiapine	Metoclopramide	Amoxapine	Tolcapone
Thioridazine	Ziprasidone	Promethazine	Diatrizoate	Dopamine agonists
Trifluordazine	Aripiprazole	Prochlorperazine	Lithium	
Thiothixene	Zotepine	Droperidol	Phenelzine	
Loxapine	Amisulpride		Dosulepin	
Bromperidol	Olanzapine		Trimipramine	
Promazine			Desipramine	
Clopenthixol				

*This table was created using data from Reference 4.

Corresponding Author: Fatih Cemal Tekin
e-mail: fatihcemal.tekin@sbu.edu.tr
Received: 06.05.2022 • **Revised:** 25.07.2022 • **Accepted:** 03.08.2022
DOI: 10.33706/jemcr.1068447

Cite this article as: Tekin FC, Sezer C. Persistent high fever after metchloropramide treatment; neuroleptic malignant syndrome. Journal of Emergency Medicine Case Reports. 2022;13(4): 101-103

DOI: 10.33706/jemcr.1112956
Journal of Emergency Medicine Case Reports. 2022;13(4): 101-103

Table 2: The DSM-V Criteria for Diagnosing Neuroleptic Malignant Syndrome are as Follows

Major Criteria (all required)	Other Criteria (at least two required)
Exposure to dopamine-blocking agent	Diaphoresis
Severe muscle rigidity	Dysphagia
Fever	Tremor
	Incontinence
	Altered level of consciousness
	Mutism
	Tachycardia
	Elevated or labile blood pressure
	Leukocytosis
	Elevated creatine phosphokinase

*This table was created using data from Reference 4.

(such as sweating, dysphagia, sialorrhea, pallor, urinary incontinence), tachycardia, tachypnea, hypertension or postural hypertension and altered mental status. High levels of creatine phosphokinase (CK) and white blood cell (WBC) can be found. Other diagnostic criteria must be assessed too. For differential diagnosis infectious diseases (encephalitis, meningitidis, brain abscess, rabies, tetanus, sepsis etc.), endocrine diseases (pheochromocytoma, thyrotoxicosis etc.), intoxications (drug abuse, heavy metal poisoning, lithium, salicylates) and other pharmacologic syndromes (serotonin syndrome, malignant hyperthermia, drug withdrawal or drug overdose) should keep in mind (3, 5).

Many patients apply to ED with fever. Causes from quite a wide range can be the trigger of fever and before deciding the therapy, doctors need to find the origin of fever, which can be very hard sometimes. For instance, just like in our case, when a patient with no fever quickly suffered from fever after drug therapy, ED doctors should immediately consider drug side effects and drug interactions for fever's origin. Some of drugs shown at Table-1 are oftenly use in EDs, therefore when a situation like our case emerged, ED doctors should consider NMS as a possible fever origin. In our case, the occurrence of NMS after a single dose of metoclopramide is unexpected and undesirable situation for emergency physicians. Thereafter with our case report we wanted to point out NMS which may be cause of the fever. It's rare, nevertheless it is a neurological emergency that every physician should keep in mind.

Case Report

21 years old male with no medical history applied the ED with abdominal and dorsal pain. His pain had started 3-4 hours ago and it was getting more painful. There was no personal or family history of known disease and drug usage. During his physical examination, he's current situation was good, he was oriented and cooperated, no neurological pathologic signs had found. His vitals were, blood pressure: 126/68, fever: 36.7°C, pulse: 76 per minute, sO2: 95%. There was a minimal tenderness at left lower quadrant of abdomen but no signs of defense, rebound or costovertebral angle tenderness. His electrocardiography (ECG) was normal sinus rhythm. His radiologic imagines were normal except for distension due to intestinal gas. Blood and urinary samples had taken from patient and to relieve his symptoms intravenous metoclopramide, proton pump inhibitor and hyoscine-n-butyl bromide medications had began. Then he was taken to observation room.

Not long after, ED doctors were informed that patient was shivering and patient was re-assessed quickly. His body temperature was 40°C. Muscle rigidity and minimal neck stiffness had emerged quickly. Intravenous hydration and anti-pyretic therapy had began. Infection markers and CK levels had studied. Despite the efforts, his fever was persistent. His laboratory studies were, WBC: 8.9, Alanine aminotransferase (ALT): 20, Aspartate Aminotransferase (AST): 20, Creatinine: 0.95, Troponin: <0.01, CK: 225, C-reactive protein (CRP): 6.4. At the same time, he was being confused and blood pressure levels were getting lower. His pupillary light reflex was +/+, pupils were isochoric, deep tendon reflexes and muscle strength were normal. ED doctors had suspected NMS and referred him to neurologist for further investigation and therapy. His central nervous system (CNS) imagines were reported normal and neurological consultant report was stating that NMS can not be excluded but other CNS pathologies and infections were excluded. Approximately 1000 cc of saline was applied intravenously to the patient in the first hour and 1000 cc of saline was given as maintenance. After enough clinical observing and symptomatic therapy, his clinic got better and his CK levels decreased to 215. Blood tests were re-evaluated at approximately 4-hour intervals and urine output was more than 1 ml/kg/hour in the follow-up. He was discharged from the hospital fully recovered after 6-8 hours of observation.

Discussion

The incidence of NMS changes between 0.02% and 3%. Majority of NMS cases are young and adult males. It's mortality rate has reduced to 10%, which is still too high but better than former situation. About this reduction, growing awareness against NMS could be an important factor (1, 5). The case we presented is also supports scientific studies about NMS.

It's main trigger is oftenly first generation of anti-psychotics with high potencies but anti-psychotics with low potencies, atypical anti-psychotics, anti-emetics, tricyclic anti-depressants and lithium can also be a trigger

(3, 6). In our case, NMS emerged after administration of metoclopramide. Metoclopramide mostly causes tardive dyskinesia, dystonia and parkinsonism due to dopamine antagonism. But some studies reported that, it can cause NMS rarely, especially in older population. According to studies, NMS can be triggered either subsequent to single dose of metoclopramide or following administrations (3, 7).

In most of the cases altered mental status, muscle rigidity, hyperthermia and autonomic dysfunction are four major components of NMS. Those symptoms can emerge in 3 days, after drug administration (1, 4, 5). In our case, after intravenous administration of metoclopramide fever, muscle rigidity, altered mental status and fluctuating blood pressure levels emerged in 30-60 minutes.

NMS is a diagnosis of exclusion. Serotonin syndrome, malignant hyperthermia, malignant catatonia, other syndromes related with drug use and central nervous system infections must be excluded. In laboratory studies leukocytosis and high levels of CK, electrolyte imbalance due to possible renal failure can be expected (1, 5, 8). In our case leukocytosis and high levels of CK has been observed. For differential diagnosis central nervous system imaging and lumber puncture may be needed.

When approaching to patients who diagnosed with NMS, first of all administration of the drug which triggered NMS must be stopped. After that intensive care observation may be needed. Anti-pyretic therapy and external cooling methods can be used. Patient must be observed about cardiorespiratory failure, renal failure, aspiration pneumonia and coagulopathy. Even though there are different opinions, dantrolene, bromocriptine, amantadine and benzodiazepines can be administered into patient. For some cases, electroconvulsive therapy may helpful (1, 5, 9). Despite studies reported some cases that intensive care unit observation and mechanical ventilation are needed, with early diagnosis and therapy most of the cases can fully recover without any complication, just like our case (7, 8).

Conclusion

NMS related to metoclopramide is quite rare, but usage of metoclopramide is very often in EDs. When considered, if single dose of metoclopramide is enough to trigger NMS, patients who received metoclopramide therapy must be observed about emergence of NMS symptoms. If imaging and laboratory studies are not suitable for differential diagnosis, patient should be transferred to a more comprehensive center.

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Case Report

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A Case of Acute Coronary Syndrome Under Immunosuppression Who is the Criminal Neutrophils or T Cells?

İrem Oktay[1], Ahmet Lütfü Sertdemir[1], Abdullah İçli[1]
[1] Necmettin Erbakan Üniversitesi Meram Tıp Fakültesi Hastanesi, Konya, Türkiye

Abstract

Chronic granulomatous disease (CGD) is a primary immunodeficiency characterized by recurrent, life-threatening bacterial and fungal infections of the skin, airways, lymph nodes, liver, brain, and bones. These infections most commonly occur in organs in contact with the outside world (lungs, gastrointestinal tract, and skin), as well as in lymph nodes that drain these structures. While involvement can be seen in many organs, there is no known cardiovascular involvement. Our case is an ACS case that has a different place in the literature because acute coronary syndrome (ACS) was seen in a twenty years old male patient with a diagnosis of chronic granulomatous disease.

Keywords: Acute coronary syndrome, bone marrow replacement therapy, chronic granulomatous disease, coronary angiography, graftversus host disease

Introduction

Chronic granulomatous disease (CGD) is an immunodeficiency syndrome characterized by recurrent, life-threatening bacterial and fungal infections of the skin, airways, lymph nodes, liver, brain, and bones. These infections most commonly occur in organs in contact with the outside world (lungs, gastrointestinal tract, and skin), as well as in lymph nodes that drain these structures. These children are normal at birth. However, infections can be quite severe in the early period. Chronic granulomatous disease is genetically inherited. Although initially thought to have only an X-linked form of inheritance, its discovery in girls in 1968 enabled the recognition of autosomal recessive forms. (1) Although incidence rates are higher in geographies where consanguineous marriage is more common, CGD is thought to affect one in 20000 to 250000 live births without ethnic preference. (2-5) As an immunodeficiency syndrome, it causes severe persistent bacterial and fungal infections such as tuberculosis, aspergilloma and osteomyelitis. (6-7) In disease management, it is aimed to quickly identify and treat acute infections and to prevent secondary granulomatous complications, together with prophylactic antibacterial, antifungal and immunomodulatory drugs. Although hematopoietic stem cell transplantation appears to be the only widely available curative treatment for patients with CGD, recent advances in gene therapy may also provide a safer and more direct option. (8) However, bone marrow transplant cannot be a definitive solution and graft versus host disease (GVHD) can be seen in patients. Although the risk of transplant related mortality and GVHD is high, survival in children has been shown to be significantly higher. The probability of developing GVHD with the increase in age has been found to be higher in studies. (9)

Case Report

A 20 years old male patient with a known diagnosis of chronic granulomatous disease, who underwent bone marrow transplant in 2008 and 2019 and was followed up in pediatric immunology with the suspicion of graft versus host disease, was consulted from the emergency department with the complaint of chest pain in the retrosternal region. From the anamnesis, it was learned that the patient was followed up with the diagnosis of chronic granulomatous disease, the last bone marrow transplant was performed in 2019, and immunosuppression treatment was started a week ago with

Corresponding Author: İrem Oktay **e-mail:** iremoktay.io42@gmail.com
Received: 31.05.2022 • **Revision:** 20.09.2022 • **Accepted:** 18.10.2022
DOI: 10.33706/jemcr.1124235

Cite this article as: Oktay I, Sertdemir AL, Icli A. A case of acute coronary syndrome under immunsupression who is the criminal neutrophils or t cells? Journal of Emergency Medicine Case Reports. 2022;13(4): 104-106

the diagnosis of graft versus host. When his family history was questioned, it was learned that his sister also died at a young age due to chronic granulomatous disease. The patient applied to the emergency department with the complaint of compressive chest pain in the retrosternal region. In the drugs he uses, methylprednisolone 16 mg tablet, ursodeoxycholic acid 200 mg tablet, calcium carbonate 1000 mg effervescent tablet, cyclosporine 25 mg tablet, mycophenolate mofetil 500 mg tablet, posacanazol 100 mg tablet, zinc sulfate 50 mg tablet, Cetirizine dihydrochloride 10 mg tablet, pantoprazole 40 mg tablet, levofloxacin 500 mg tablet, acyclovir 800 mg tablet were seen. It was learned that he had no history of smoking and dyslipidemia. On physical examination, blood pressure was 110/60 mmHg and saturation was %95 , heart rate was 116/min. Electrocardiography (ECG) of the patient with typical anginal symptoms showed normal sinus rhythm and diffuse ST elevations. (figure 1) Echocardiography was performed on the patient. The ejection fraction was %40 anterior, and wall motion defect in the septum and first degree mitral regurgitation were observed. In laboratory results, troponin is 5.39 ug/l, ck-mb is higher than 300 ug/l, creatinine normal alanine aminotransferase (ALT) and aspartate aminotransferase are high, C-reactive protein (CRP) is within normal limits, hemoglobulin 19.6 g/dl, platelet 190 thousand, white blood cells was seen as 12 thousand. The patient, who had acute coronary syndrome findings on ECG and ECHO, describing typical anginal symptoms, had elevated troponin and was taken to coronary angiography (CAG) with a preliminary diagnosis of ACS. The right coronary artery (RCA) was normal, the proximal circumflex (CX) lesion was %70, and the left anterior descending artery (LAD) proximal was %100 occluded. The total occluded lesion proximal to the LAD was predilated with a 2.5*30 mm balloon, and then a 2.5*33 mm drug-coated stent was implanted. Then, the stent was post dilated with a 2.75*12 mm balloon. Full disclosure has been achieved and the process has been terminated. (Figure 2) The patient was taken to the coronary intensive care unit. Dual antiaggregant therapy was started. The patient with elevated hemoglobin levels was consulted to hematology. One unit of phlebotomy was performed and mycostatin oral suspension was started. The patient, who was followed in isolation in the coronary intensive care unit for two days, was discharged with full recovery after medical treatment was arranged.

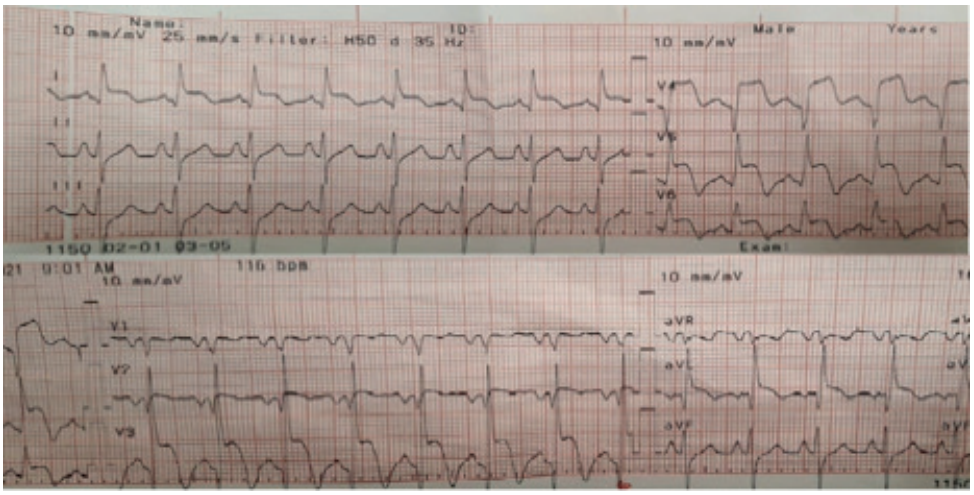

Figure 1.

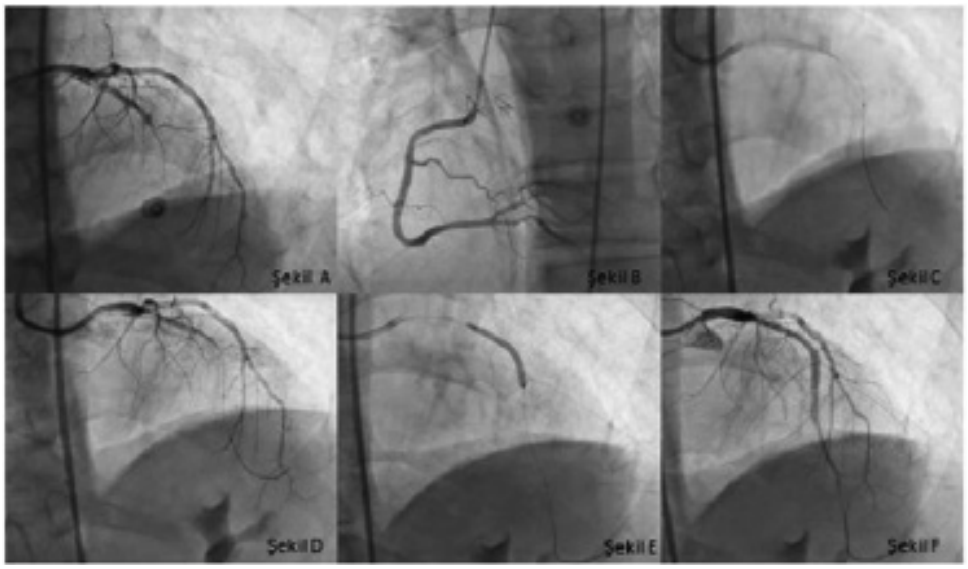

Figure 2.

DOI: 10.33706/jemcr.1124235
Journal of Emergency Medicine Case Reports. 2022;13(4): 104-106

Discussion

Chronic granulomatous disease is a disease that affects the immune system and progresses with the formation of granulomas as a result of the inability of microorganisms to be phagocytosed by neutrophils. Many organs involvement can be seen, but cardiovascular involvement is unknown. However, there are also granulomatous diseases with cardiovascular involvement. When we look at the literature, we see examples of this. For example, acute coronary syndrome and left anterior descending artery occlusion similar to our case were detected in a young male patient with Wegener's granulomatosis. (10) In another example, in two different patient groups with Takayasu artery and rheumatoid arthritis, early atherosclerosis was observed in those with Takayasu arteritis.(11) This was thought to be because atherosclerosis was associated with chronic systemic inflammation without cardiovascular risk factors. It has been accepted that vascular remodeling and endothelial activation play a role in the development of atherosclerosis in autoimmune granulomatous diseases. Considering the classification of myocardial infarction, type 1 myocardial infarction develops due to atherosclerotic plaque rupture, while type 2 myocardial infarction develops due to an ischemic cause due to an imbalance of myocardial oxygen supply and demand. In the light of all these, our patient was evaluated as type 1 myocardial infarction in the presence of ST elevation on ECG, elevated cardiac biomarkers and anginal symptoms. Although antibiotics, intravenous immunoglobulin and bone marrow transplantation are used in the treatment of chronic granulomatous diseases, GVHD can be seen. In cases where GVHD develops, the only treatment that can be done is to suppress the immune system. For immunosuppressive treatment, drugs such as methylprednisolone, cyclosporine, mycophenolate mofetil are usually used in the first stage. Although macrophages are in the first place in the pathogenesis of atherosclerosis, both CD8 and CD4 T lymphocytes are seen at every stage of atherogenesis. The presence of these cells in the atherosclerotic lesion suggests that atherosclerosis occurs as a result of an immune or perhaps an autoimmune response. As a matter of fact, in a study conducted in the literature, subendocardial ischemia was observed in the imaging performed with adenosine in patients with Wegener's granulomatosis, and it was shown that myocardial ischemia disappeared after cyclophosphamide and steroid.(12) Considering all these studies and our case, the increased number of neutrophils in granulomatous diseases and the high number of T cells brought about by an autoimmune mechanism raises the question of whether neutrophils should be responsible for the etiology of atherosclerosis or whether T lymphocytes should be responsible. Our case enters the literature as a special case emphasizing this contradiction.

Informed consent: *Informed consent was obtained from the patient for the publication of the case report and the accompanying images.*

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Case Report

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Kounis Syndrome That Recurs in A Short Time Period: A Case Report

İlker Akbaş[1], Abdullah Osman Koçak[2], Sinem Doğruyol[3]

[1]Department of Emergency Medicine, Kahramanmaras Sutcu Imam University, Kahramanmaras, Turkey.

[2]Department of Emergency Medicine, Faculty of Medicine, University of Ataturk, Erzurum, Turkey.

[3]Department of Emergency Medicine, Haydarpasa Numune Training and Research Hospital, Istanbul, Turkey.

Abstract

This case report is unique for the occurrence of a quickly recurring Kounis Syndrome (KS) due to re-exposure to the same agent. A 40-year-old male was brought to our ED with a diagnosis of non-ST-segment elevation myocardial infarction. He stated that he had taken one dose of amoxicillin-clavulanate 1,000 mg at 03.00 AM. After taking the drug, chest pain and vomiting began. ECG which was taken at the the rural hospital, revealed a normal sinus rhythm with no ischemic changes. The value of cardiac troponin I 0.34 ng/ml in the rural hospital. The patient was consultated to the cardiology clinic with a pre-diagnosis of KS. Percutaneous coronary intervention showed that coronary arteries were normal and no plaque formation was found. The patient, who was diagnosed with type I KS, left the hospital at his own request at 14:12 PM. The patient presented to our ED again at 22:30 PM with chest pain and shortness of breath after accidentally using the same allergenic drug ~eight hours after leaving our hospital. ECG showed > 0.5 cm ST-segment elevation in leads DII, DIII, and aVF. Quickly recurring KS was due to accidental reuse of the same agent may be more severe than the first occurrence.

Keywords: Kounis syndrome, acute coronary syndrome, allergic infarction, recurring Kounis syndrome

Introduction

Kounis syndrome (KS) is defined as the concurrent occurrence of an acute coronary syndrome (ACS) with a hypersensitivity reaction (1, 2). KS is an entity that develops coincidentally after exposure to allergen, that can affect all races and age groups and that can be seen in many geographical regions (3). Food, drugs and especially environmental agents are frequently encountered etiological causes of this entity (4). When the high risk of causing allergic reactions is considered, non-steroidal anti-inflammatories and antibiotics are the drugs that are most frequently associated with this syndrome (5).

Kounis syndrome is a lesser-known entity (3). However, in the literature, there are many cases of KS that have developed due to different agents. But 'recurrent' KS that has developed as a result of recurrent use of the same agent has been seen quite rare (5). In this case report, we aimed to present a recurrent case of KS with two different types of ACS, which developed in a short time after reuse of the same drug.

Case Report

A 40-year-old male patient was brought to our emergency department (ED) by an ambulance from a rural hospital with a diagnosis of non–ST-segment elevation myocardial infarction (NSTEMI). The past medical history of the patient was unremarkable in terms of coronary disease risk. There was no known allergy history. The patient stated that he had taken one piece of the prescribed medicine containing Amoxicillin-Clavulanate 1000 mg (Klavon 1000 mg - Husnu Arsan Drug Co., Turkey) at 03.00 AM. He went to the hospital with complaints of chest pain and vomiting which had started 10 minutes after the drug intake. He described his pain in the form of pressure, starting from the retrosternal region and spreading to the left arm and chin. The chest pain was accompanied by nausea, vomiting, and abdominal pain. Vital findings were recorded as follows: blood pressure 81/55 mmHg and pulse 106/min. His physical examination was unremarkable. The electrocardiogram (ECG) taken at the 15th minute of his admission revealed a normal sinus rhythm with no ischemic change. The value of cardiac

Corresponding Author: İlker Akbas **e-mail:** akbasilker@gmail.com
Received: 06.06.2022 • **Revised:** 07.11.2022 • **Accepted:** 08.11.2022
DOI: 10.33706/jemcr.1126723

Cite this article as: Akbas I, Kocak AO, Dogruyol S. Kounis syndrome that recurs in a short time period: a case report. Journal of Emergency Medicine Case Reports. 2022;13(4): 107-110

DOI: 10.33706/jemcr.1126723
Journal of Emergency Medicine Case Reports. 2022;13(4): 107-110

troponin I (cTnI) was determined as 0.34 ng/ml (reference < 0.3 ng/ml) in the rural hospital. Other laboratory tests were within normal limits.

The patient was admitted to our ED as NSTEMI around 06.00 AM. He had no complaints on admission. The vital signs and physical examination were normal. No significant ischemic change was observed in his ECG (Figure-1). The cTnI values studied in our ED were above the normal limit (2.96 ng/ml). Therapy for ACS was started in the ED. The present status of ACS in the patient was associated with anaphylaxis that had developed due to drug use. The patient was consulted to the cardiology clinic with a pre-diagnosis of KS. The coronary arteries were considered normal in percutaneous coronary intervention (PCI). No plaque formation was found. The patient, who was diagnosed with type I KS, left the hospital at his own request while he was on follow-up (14:12 PM).

The patient re-presented to our ED at 22.30 PM, complaining of chest pain and shortness of breath. He had accidentally used the same allergenic drug once again about 8 hours after leaving the hospital. He stated that his complaints had started shortly after the drug use. The chest pain was similar to his previous pain. But the patient stated that the pain was more severe. The vital signs and physical examination findings were normal. His ECG showed > 0.5 cm ST-segment elevation in leads DII, DIII, aVF, and > 0.5 cm reciprocal ST-segment depression in leads DI, aVL (Figure-2). The cTnI value was 9.12 ng/ml. Treatment for anaphylaxis and ACS was started in the patient who was considered to have KS and an associated STEMI. He was discharged from the cardiology clinic after a 3-day follow-up and treatment and was informed about the need to avoid drugs containing amoxicillin-clavulanate.

Discussion

There is a quite wide literature on KS and each new case opens up new horizons for allergens that play a role in aetiology. This case, reported by us, can be considered quite ordinary if considering only the etiological agent (6). However, what makes our case special is that it is a recurrent KS that recurs in a very short time due to re-exposure to the same agent. Recurrent KS that has developed due to the same agent is a quite rare condition in the literature (5, 7, 8, 9, 10). KS with a quite short recurrence time is another important feature that differs from the literature in our case. Gunaydin et al. mentioned a case of KS that developed one year apart due to bee sting (7).

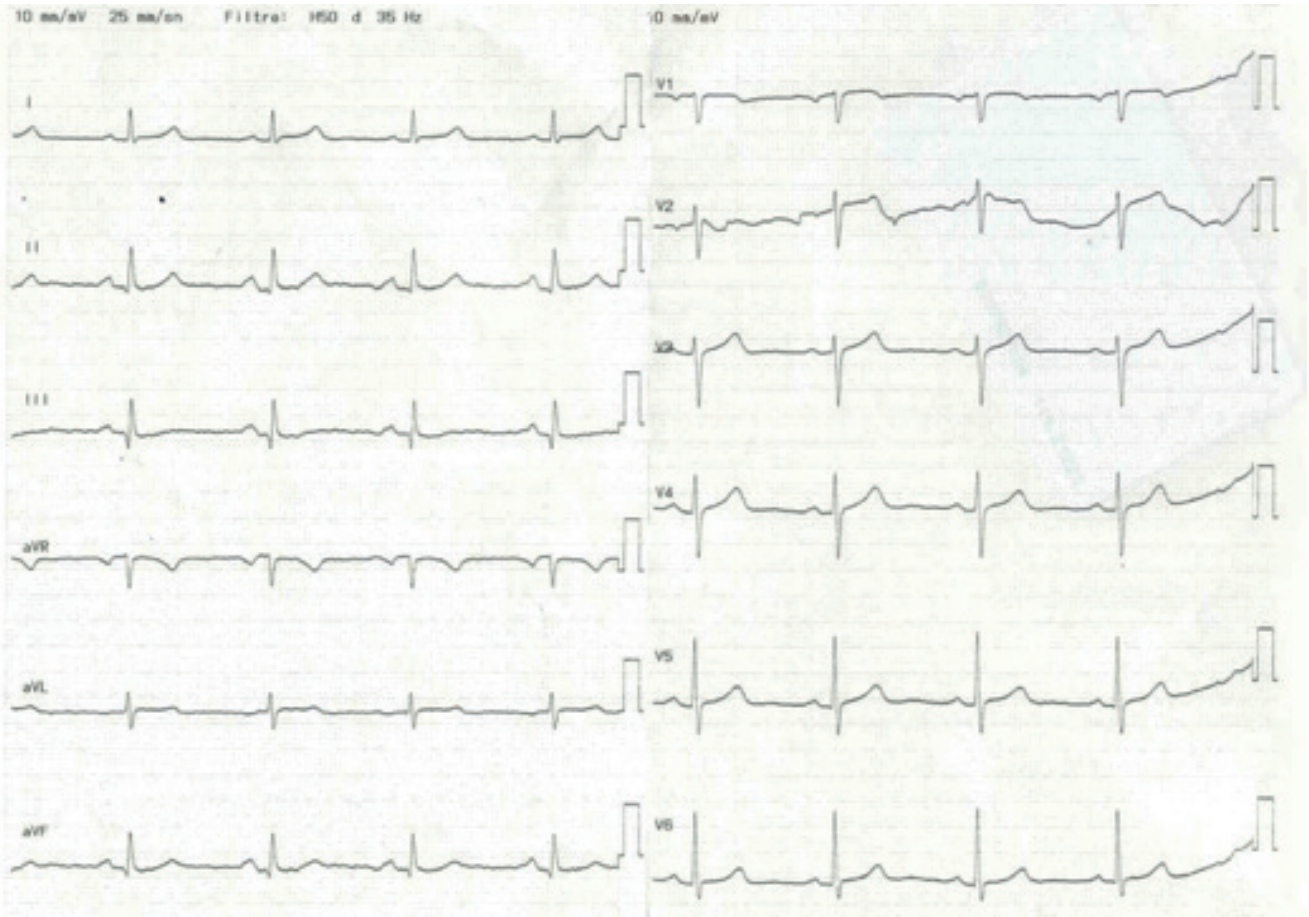

Figure 1. Initial ECG in ED revealed no significant ischemic changes

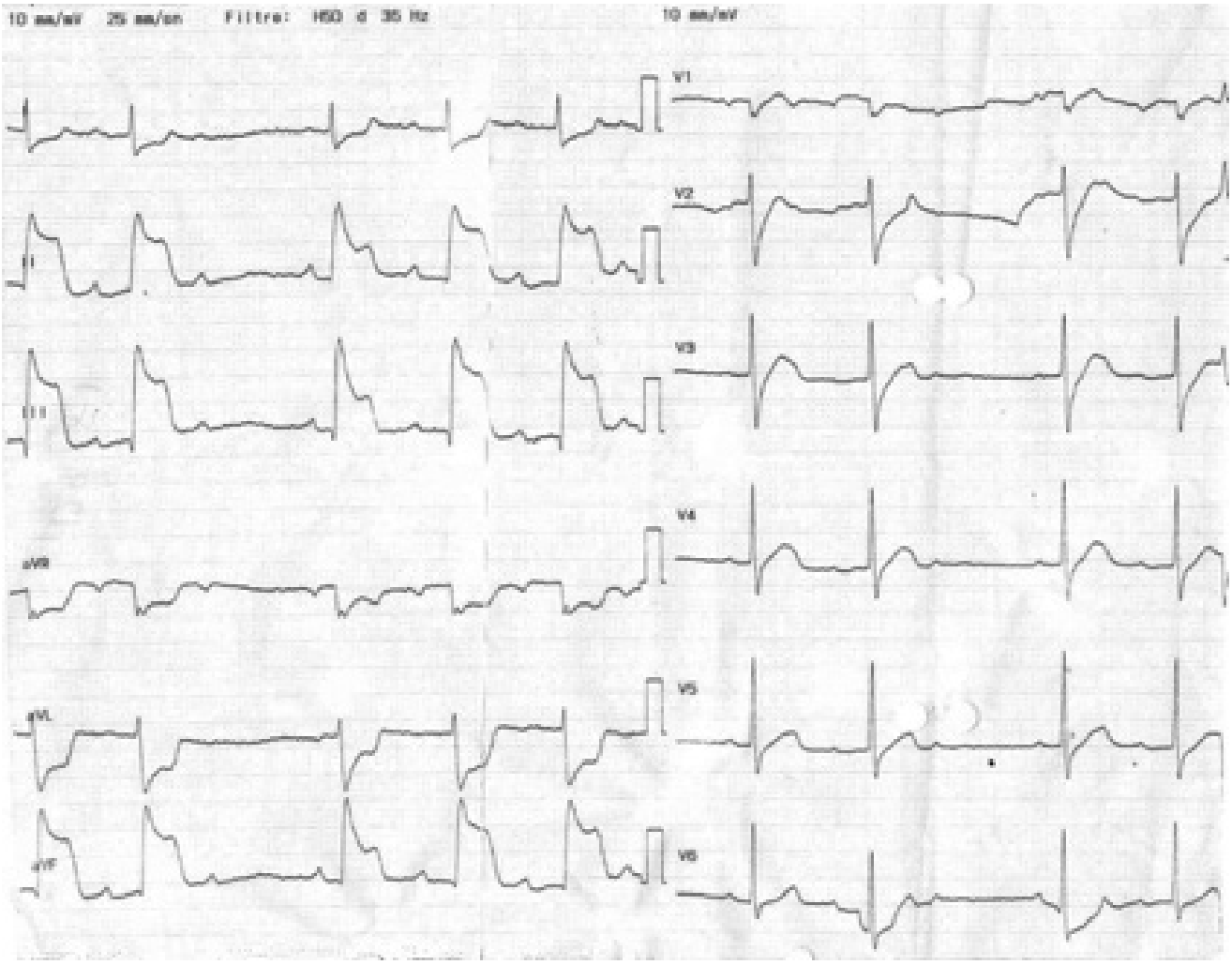

Figure 2. ECG showed > 0.5 cm ST-segment elevation in leads DII, DIII, and aVF and > 0.5 cm reciprocal ST-segment depression in leads DI and aVL

Celiker et al. reported a case of KS that occurred at 8-months interval due to pseudoephedrine use (5). In another case report, a case of KS that developed due to general anaesthetics that were given at 4-week intervals was reported (9, 10). Patane et al. presented a case of allergy and allergy-related ACS as a result of clopidogrel use. In this case, recurrence developed as a result of reuse of the same drug after three days (8). In our case, KS recurrence time is approximately 18 hours. To the best of our knowledge, this time period is much shorter than those so far reported in other cases.

KS can be presented with many different clinical pictures. However, this syndrome should definitely come to mind in the presence of allergic symptoms accompanying chest pain (11, 12). The clinician's suspicion is the most important step in diagnosis. Diagnosis begins with a detailed medical history. It is confirmed based on symptoms and signs, ECG changes, laboratory findings, echocardiography, and angiography at the timed of admission (4, 5). In our case, both the medical history and accompanying ECG and laboratory findings were the clues that allowed us to think about KS in preliminary diagnosis. The most important point that should not be forgotten in a diagnosis is that clinical findings may not always reflect KS fully. While silent angina may be seen in some patients, in others, as in our case, the clinical manifestations of allergy may not be clear (2). In some cases, the presence of hypotension without skin manifestations may be the only clue in the diagnosis of anaphylaxis (7).

Three different types of KS have been described: Type 1 coronary vasospasm, Type 2 native plaque destabilization and Type 3 stent thrombosis (7, 12). In the type I variant, there is no risk factor for coronary artery disease and the coronary arteries are angiographically normal. Coronary vasospasm developing due to an allergic reaction is responsible for the disease. If vasospasm progresses when cardiac enzymes are normal, it may increase due to myocardial damage (2, 7, 13). Our case also had no risk factors for AKS and the coronary angiography was normal. Therefore, the case was considered as KS Type 1 variant. In addition, our case was also compatible with the literature, which states that KS Type 1 is more common in cases of KS induced by amoxicillin than other variants (14).

ECG changes in KS can range from ST segment elevation to depression, and from any degree of heart block to cardiac arrhythmias (15). The first admission ECG of our

DOI: 10.33706/jemcr.1126723
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case was normal, however, ECG showed elevation of the ST segment in the inferior leads in the second admission. Although it is thought that the clinical status in the second reaction to the allergen will be worse, it can be is seen that the clinical symptoms are similar in recurrent KS cases in the literature (5, 8, 10). However, since this situation may not be valid for all cases, these patients who develop KS should be sufficiently informed and the list of drugs that are to be avoided should be stated.

As a result, KS can be seen for the second time in a very short time due to the accidental use of the same agent.

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Case Report

Journal of Emergency Medicine Case Reports

Coexistence of Late Diagnosed Pericardial and Diaphragmatic Ruptures Caused by Rib Fracture

Mehmet Ağar[1], Semih Koçyiğit[1]
[1]Department of Thoracic Surgery, Fethi Sekin City Hospital, Elazıg, Turkey.

Abstract

While rib fractures due to blunt traumas are common, pericardial and diaphragmatic injuries caused by rib fractures are rarely seen. Diagnosis is challenging due to the difference in clinical symptoms which may have severe clinical consequences. A 58-year-old female patient who had a traffic accident was referred to our center due to left pericardial effusion while being followed up for tibial fracture. Left diaphragmatic rupture was detected in the radiographs taken. Intraoperative pericardial rupture was observed in the operated patient. The defects were repaired primarily and the patient was discharged in good health. Although the diagnosis is difficult in multitraumas including thoracic trauma, especially in cases with multiple rib fractures, one should be more attentive considering the possibility of diaphragmic and pericardial ruptures.

Keywords: rib fracture, pericardial rupture, diaphragmatic rupture

Introduction

Pericardial and diaphragmatic ruptures, which may lead to serious complications, may be asymptomatic in multi-trauma patients with rib fractures. Diagnosis of these clinical conditions is difficult and can be missed due to the hasty, and acute approach to the multi-trauma patient.

Cardiac herniation is the most common complication and cause of death in pericardial injury, which constitutes 0.4%-2% of blunt trauma cases (1). Rib fractures have been shown to be the most common cause of pericardial rupture (2). Depending on the size of the pericardial defect, patients may present with symptoms of pericardial tamponade, but may also progress asymptomatically.

The most common cause of diaphragmatic injuries, which constitute less than 1% of traumatic injuries, is rib fractures (3). Small diaphragmatic defects are asymptomatic, while large defects may present with symptoms and respiratory distress due to intrathoracic organ herniation.

Case Report

A 58-year-old female patient was operated by the orthopedics department for multiple rib fractures in the left hemithorax and comminuted fractures of the left tibia due to an in-vehicle traffic accident. The patient, who was followed up in the ward for 15 days, was consulted to our clinic after detection of left pleural effusion in her radiographs. Her general condition was good, and her physical examination revealed swelling due to a possible subcutaneous hematoma on the lateral left hemithorax, and tenderness on palpation. Thoracic computed tomography of the hemodynamically stable patient performed in our center displayed multiple, displaced and non-displaced rib fractures in the left hemithorax (left 2nd-8th ribs), and spleen herniated into the thorax. Surgery was decided in the same session organized with the department of orthopedics. After the orthopedic surgery, the thorax was entered by performing left uniportal video-assisted thoracoscopic surgery (VATS).

On exploration, the lung, intrathoracic spleen and stomach firmly adhered to the thoracic wall were observed (Figure 1a). When the adhesions of the anterior surface of the lung were removed, ruptured pericardium was seen (Figure 1b). Thoracotomy was decided due to restricted exposure and double lumen intubation problem. The heart was evaluated and any sign of trauma was not found. The pericardium was primarily repaired by intermittently suturing the ruptured ends with a 2/0 silk suture (Figure 2). The spleen, stomach and omental tissue in the thorax were tried to be pushed towards the abdomen without success, thus laparotomy was

Corresponding Author: Mehmet Ağar
e-mail: md.mehmetagar@gmail.com
Received: 15.06.2022 • **Revision:** 08.08.2022 • **Accepted:** 15.08.2022
DOI: 10.33706/jemcr.1131413

Cite this article as: Agar M, Kocyigit S. Coexistence of late diagnosed pericardial and diaphragmatic ruptures caused by rib fracture: case report. Journal of Emergency Medicine Case Reports. 2022;13(4): 111-113

DOI: 10.33706/jemcr.1131413
Journal of Emergency Medicine Case Reports. 2022;13(4): 111-113

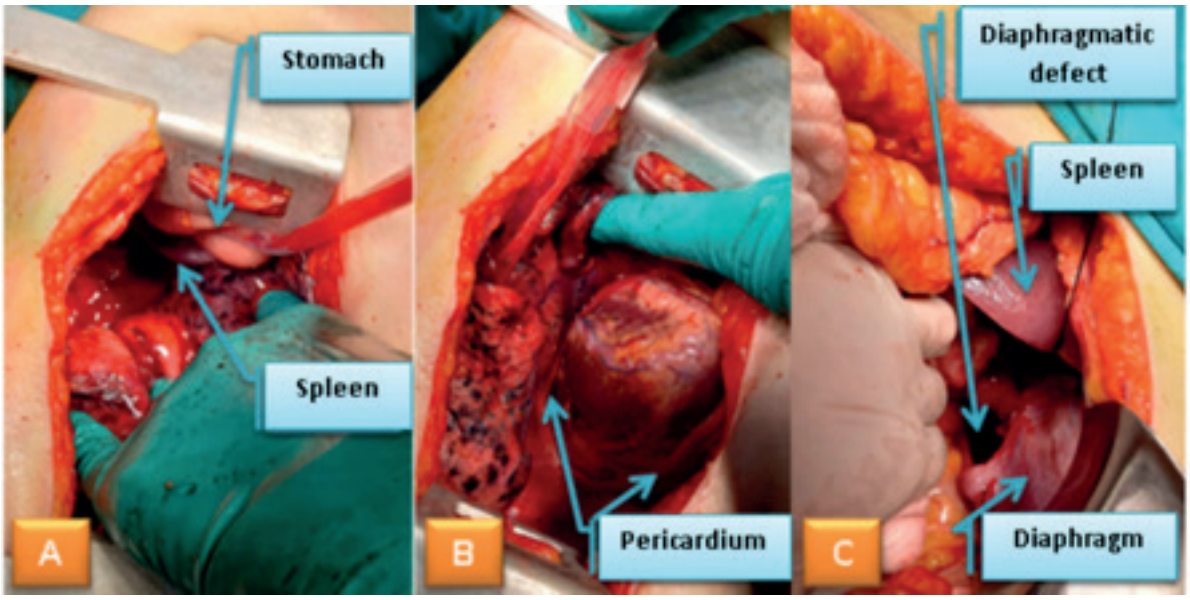

Figure 1. Intrathoracic stomach and spleen [A], pericardium [B] and diaphragm rupture [C].

performed on the patient due to the size of the defect and exposure problem. By laparotomy, intrathoracic structures were retracted into the abdomen and diaphragmatic rupture was observed (Figure 1c). The resulting diaphragmatic defect was primarily repaired with 1/0 silk sutures and the operation was terminated. The patient, who was extubated after the operation, was followed up in the intensive care unit for one day, and then she was then transferred to the service. On the 4th day, the chest tube of the patient was removed when sufficient pulmonary expansion without any air leakage and fluid drainage from the chest drain was observed on PA AC radiographs. The patient was discharged after orthopedic follow-ups.

Discussion

Diagnosis of pericardial injury is quite challenging due to the fact that the entities are often caused by multitraumas, and few cases are diagnosed at their first presentation. While only 18% of patients are correctly diagnosed preoperative, the diagnosis is made intraoperatively or in autopsy series in most cases (4). In our patient, the diagnosis could not be made in the acute period, and she was sent to our center on suspicion after 15 days of follow-up.

Patients may manifest symptoms consistent with cardiac tamponade, but if the defect is large, clinical deterioration may not be observed. Pericardial tears occur in the diaphragmatic or

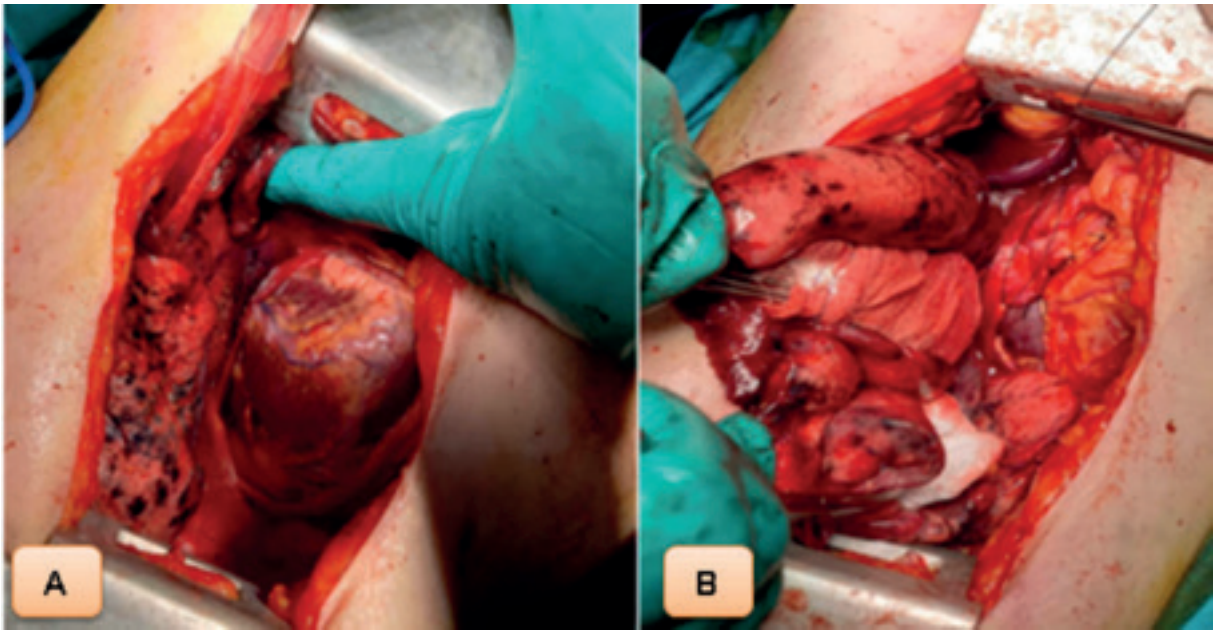

Figure 2. Ruptured pericardium [A] and its primary repair [B]

pleural pericardium. Pericardial tears are seen most commonly in the left pleural pericardium, followed by diaphragmatic and right pleural pericardium in decreasing frequency (5).

Chest X-rays, ECG cardiac biomarkers, echocardiography, computed tomography and MR (magnetic resonance) imaging are being used as diagnostic tools. Surgical exploration should be performed immediately when pericardial rupture occurs. Because of the risk of cardiac herniation, primary repair of 8-12 cm-long ruptures is recommended (6). Posterolateral thoracotomy, including median sternotomy, and minimally invasive thoracoscopic approaches are being used in the treatment.

In the literature review conducted by Hongbin W. et al. the cause of rupture was found to be rib fractures in 21 of 42 patients with blunt trauma (2). Torah O. et al. detected pericardial rupture in 3 of 10 patients with diaphragmatic rupture due to blunt trauma (7).

Diaphragmatic injuries represent less than 1% of traumatic injuries. Penetrating diaphragmatic injuries are more common than blunt injuries. Although acute large diaphragmatic injuries manifest themselves both clinically and radiologically, diaphragmatic injuries are usually asymptomatic and diagnosed at a late stage (8). The clinical manifestations vary according to the size of the defect. While small defects are asymptomatic, large defects may cause severe respiratory distress. Our patient was asymptomatic during the follow-up period and did not show any clinical symptoms.

Diaphragmatic injuries occur most frequently on the left side, and the most common cause of injury is rib fractures (9). Surgical repair of the defect is performed in the patient with diaphragmatic injury. The defect can be repaired through laparotomy or thoracotomy. Laparoscopic or thoracoscopic methods can be tried in patients with small defects. Beng Leong L. et al. found diaphragmatic ruptures in 46 of more than 13 thousand trauma patients (10).

Conclusion

Although the coexistence of pericardial and diaphragmatic ruptures is a rare condition that poses a diagnostic challenge in multitrauma patients, it can lead to serious clinical consequences. These two entities should be kept in mind and should be treated more attentively, especially in patients with multitrauma, including those with rib fractures.

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Case Report

Journal of Emergency Medicine Case Reports

Soft Tissue Tuberculosis Mimicking Ewing Sarcoma: A Case Report

Mehmet Fatih Orhan[1], Mustafa Büyükavcı[1], Olena Erkun[2], Saliha Çıracı[3], Huri Tilla İlçe[4]

[1] Sakarya University Faculty of Medicine, Department of Pediatric Hematology and Oncology, Sakarya, Turkey.

[2] Sakarya University Faculty of Medicine, Department of Pediatrics, Sakarya, Turkey.

[3] Sakarya University Training and Research Hospital, Department of Radiology, Sakarya, Turkey.

[4] Sakarya University Faculty of Medicine, Department of Nuclear Medicine, Sakarya, Turkey.

Abstract

Mycobacterium tuberculosis is a disease seen in every tissue and organ. Although it often involves the lung and pleura, it can also progress into extrapulmonary tuberculosis. Soft tissue and bone tuberculosis are the least common of all tuberculosis types. In some cases, the lesions may appear like bone tumors or metastatic lesions. Bacteriological and histopathological studies reach a definitive diagnosis because of the biopsy. We present a case suggestive of Ewing's sarcoma with clinical and imaging findings but diagnosed as soft tissue tuberculosis resulting from the biopsy. A two-year-old girl was admitted to our clinic with the complaint of palpable swelling on the left side of her chest. Ewing sarcoma was considered with the findings of Computed Tomography (CT), Magnetic Resonance Imaging (MRI), and PET-CT. Biopsy material was reported as casefied granulomatous inflammation, and M. tuberculosis complex DNA was detected by PCR examination of the tissue. The patient was successfully treated with rifampin, isoniazid, pyrazinamide, and ethambutol. Today, it should be kept in mind that tuberculosis is a common disease, rarely isolated soft tissue or bone involvement, and can be confused with malignancy.

Keywords: Child, Ewing sarcoma, Chest wall mass, tuberculosis

Introduction

Mycobacterium tuberculosis can be seen in every tissue and organ. Although it often involves the lung and pleura, it can also progress into extrapulmonary tuberculosis. Soft tissue tuberculosis is the least common of all tuberculosis types. Rarely, they may involve the soft tissues and skin and cause very different clinical manifestations. Since it does not give specific symptoms and signs, it is difficult to diagnose, and there may be a delay in diagnosis. In cases where primary pulmonary tuberculosis is not accompanied, or the disease is asymptomatic, it may be confused with thoracic wall tumor, tuberculous osteomyelitis, tuberculosis cold abscess, and malignancy[1,2]. In some cases, the lesions may appear similar to bone tumors or metastatic formations. Definitive diagnosis is reached with bacteriological and histological studies resulting from biopsy[3].

Ewing sarcoma is one of the small round blue cell tumors. The most common places are; the trunk, pelvis, vertebrae, thorax, and extremities. The most common presenting symptoms are swelling, pain, limitation of movement, and tenderness in the affected area. Respiratory distress may also be seen in patients with sizeable primary chest wall tumours[4]. Ewing sarcoma is one of the first diseases that come to mind when a mass or a lesion originating from the rib is seen on the chest wall[5]. Here, we present a case who presented with the complaint of swelling in the chest wall and was initially thought to be Ewing's sarcoma on imaging but was diagnosed as soft tissue tuberculosis by biopsy.

Case Report

A two-year-old female patient was admitted to our clinic with a palpable swelling on the left side of her chest for 20 days (Picture 1). The patient's and family history was unremarkable. The schedule completed the patient's vaccinations, and there was a 5mm scar on the left arm. On physical examination, a 3x3 cm, semi-mobile, firm, and painless mass was detected under the nipple on the left side of the chest wall. No pathological findings were found in other system examinations. The patient's body weight was 11 kg (10-25 percentile), and his height was 84 cm (25-50 percentile). In laboratory examination, Complete blood count, serum electrolytes, and kidney and liver functions were within the normal reference range, and the erythrocyte sedimentation rate was 36 mm/hour. Immunoglobulins were within the normal range, lymphocyte subgroups were within the normal reference range: CD3 66.5%, CD4 43.3%, CD19

Corresponding Author: Mehmet Fatih Orhan
e-mail: forhan@sakarya.edu.tr
Received: 18.06.2022 • **Accepted:** 13.09.2022
DOI: 10.33706/jemcr.1132704

Cite this article as: Orhan FM, Buyukavci M, Erkun O, Ciraci S, Tilla Ilce S. Soft tissue tuberculosis mimicking ewing sarcoma: a case report. Journal of Emergency Medicine Case Reports. 2022;13(4): 114-117

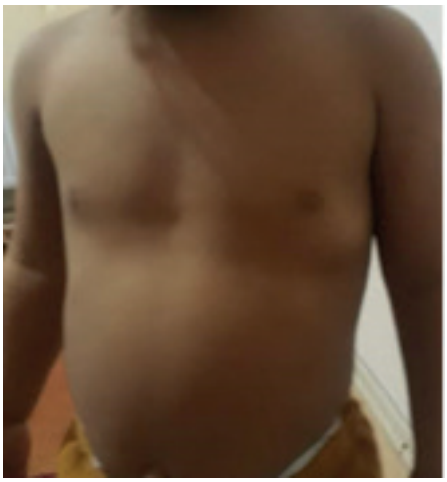

Figure 1. The appearance of a mass lesion on the chest wall.

23.4%, NK 7.7%, CD4/CD8 2.3. Ultrasonography revealed a 30x36 mm heterogeneous lesion with solid and cystic areas and amorphous calcifications in the left anterior-lateral wall of the chest, which could not be distinguished from the rib. The patient's chest X-ray was normal. Computer Tomography (CT) (Picture 2a, 2b) and Magnetic Resonance Imaging (MRI) (Picture 2c, 2d, 2e) images show a mass lesion originating from the 6th rib, 24x32x30 mm in size, extending towards the

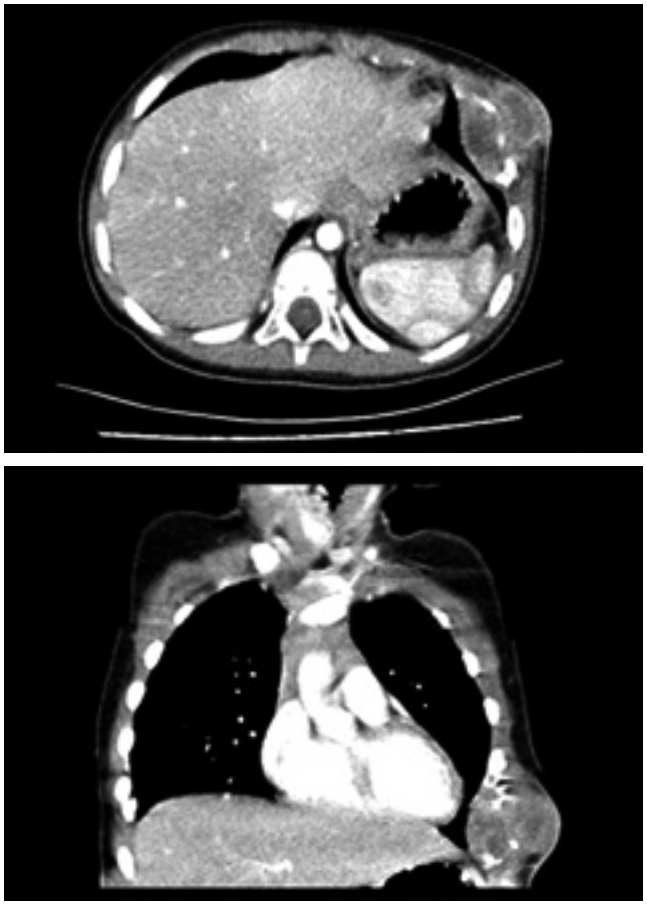

Figure 2a, 2b. On thorax CT examination: A mass lesion on the 6th rib, 26x33mm in size, hypodense in the center, cystic-necrotic structure, hyperdense solid in the periphery, peripherally contrasting, expanding-destroying the rib and extending into the soft tissue.

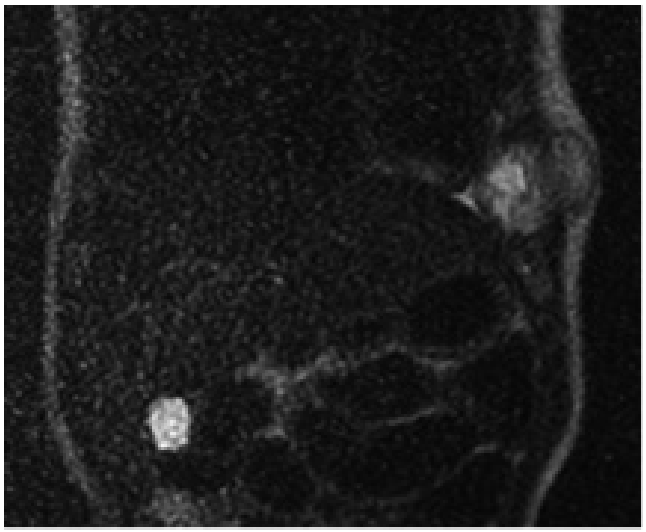

Figure 2c. Mass lesion with a central hyperintense appearance, cystic-necrotic structure, hypointense solid structure on the periphery, and extending into the soft tissue in coronal T2W MRI examination

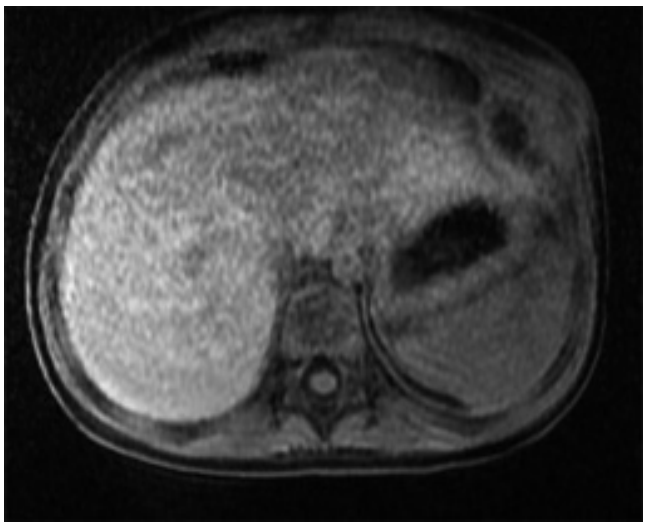

Figure 2d. On T1W MRI with axial contrast, a mass lesion originating from the 6th rib, 24x32x30 mm in size, hypointense in the central, cystic-necrotic structure, peripheral hyperintense solid construction, peripheral enhancement, extending into the soft tissue.

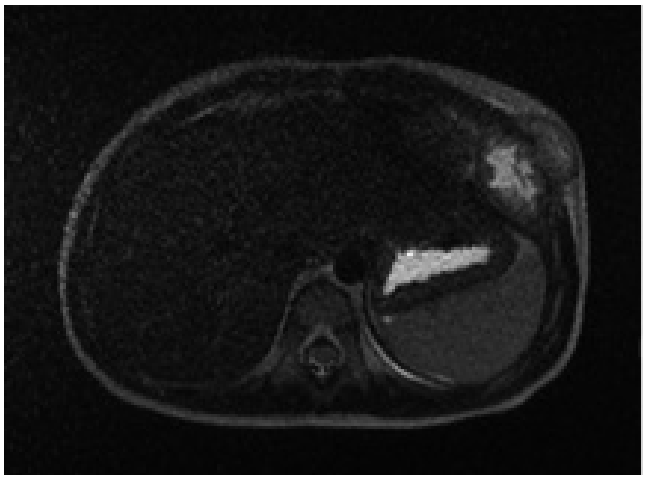

Figure 2e. Mass lesion with central hyperintense appearance, cystic-necrotic structure, peripheral hypointense, solid structure, extending into soft tissue in axial T2W MRI examination

DOI: 10.33706/jemcr.1132704
Journal of Emergency Medicine Case Reports. 2022;13(4): 114-117

abdomen and under the skin, destroying the rib. The lesion had central necrosis and peripheral contrast enhancement. In the whole body PET-CT examination, a malignant mass was reported in the left intercostal space, lysing the 6th rib, medially adjacent to the liver and stomach corpus, extending to the cutaneous-subcutaneous plane, and showing pathological FDG uptake (SUVmax: 7.7). All these reports are suggestive of Ewing's sarcoma. Histopathological review of the tru-cut biopsy samples reported granulomatous inflammation. Tuberculin skin test was negative. In the polymerase chain reaction (PCR) examination of the tissue samples, M. tuberculosis complex DNA was detected. Treatment with rifampin, isoniazid, pyrazinamide, and ethambutol was started. In the 6th month of her treatment, a significant clinical and radiological regression was observed in the size of the mass (Figure 3a, 3b).

Discussion

The skeletal system is one of the most common sites of extrapulmonary tuberculosis infection and constitutes 1-3% of all tuberculosis cases. The most commonly affected bones in children are; the metaphysis of long bones such as the femur, tibia, fibula and humerus, vertebrae, pelvis, and skull bones. Although the infection occurs due to the spread of the microorganism from the lungs by the lymphohematogenous route, a primary pulmonary lesion may not be seen at the time of diagnosis[6,7]. The history of contact with a possible tuberculosis case is also very low[8]. Skeletal tuberculosis is more common in immunocompromised patients, especially multifocal bone lesions in these patients[2]. Immune system deficiency was not detected in our case. In addition, no other tuberculosis focus was detected in the lung and musculoskeletal system. No tuberculosis infection was seen as a result of screening of family members.

Since clinical and radiological findings are not specific in the early period in children, the diagnosis can be easily missed[5,6]. Pain, swelling, and limping are the most common complaints. Fever, weight loss, night sweats, weakness, muscle atrophy around the joint, and pathological fracture are rare findings[9]. Tuberculin skin test, erythrocyte sedimentation rate, complete blood count, and chest x-ray are auxiliary diagnostic methods[1,2,6]. Our case presented with the swelling in the thoracic wall without pain and systemic symptoms. Erythrocyte sedimentation rate was slightly elevated. With these findings, we first thought of costal malignancy in our patient.

Tissue swelling can be detected only in the affected area in the early X-ray of patients with soft tissue and bone tuberculosis. In the late period, a lytic, oval, lobulated structure with multiple sclerotic cystic appearances extending from the metaphysis to the epiphysis is typical and is often accompanied by osteoporosis[2,6,8]. Computed Tomography and MRI examinations will help determine the degree of bone involvement. The preliminary diagnosis was reported as malignancy in all imaging studies performed on this patient, including ultrasonography, CT, MR, and PET CT.

Similar radiological findings may be seen in tuberculous bone lesions, such as eosinophilic granuloma, primary/secondary malignancies, or fungal infections. In children, lytic lesions may resemble bone pathologies such as leukemia, neuroblastoma, and Langerhans cell histiocytosis[2,7]. Multifocal bone involvement may occur[6,8]. Malignancies, including Ewing's sarcoma, are primarily considered in the differential diagnosis of mild tissue tuberculosis[2,3,5].

Ewing sarcoma is one of the small round blue cell tumors. The most common sites are the trunk, pelvis, vertebrae, thorax, and extremities. The most common presenting complaints are swelling, pain, limitation of movement, and tenderness in the affected area. Respiratory distress may also be seen in patients with a large primary chest wall tumor. Lytic lesions on direct X-ray and characteristic onion skin appearance due to periosteal reaction should suggest Ewing's sarcoma. In radiological imaging, the tumor often crosses the cortex and enters the soft tissue, and the soft tissue component can sometimes be huge. There is no calcification in the smooth tissue extension4. In the differential diagnosis, osteomyelitis due to bacteria, fungi, and bone tumors that cause lytic lesions should be investigated. Therefore, the biopsy is recommended in suspicious cases[2,5,8].

Detection of granulomatous inflammation in the histopathological examination of the tissue specimen supports the diagnosis of tuberculosis. Acid-fast bacteria can be seen with Ziehl-Neelsen staining. M. tuberculosis growth in culture is diagnostic[8]. However, since M. tuberculosis culture takes a long time, it may cause a delay in treatment. Polymerase chain reaction (PCR), one of the nucleic acid amplification methods, can be used for faster diagnosis. In our case, the diagnosis of tuberculosis was made due to the detection of M. tuberculosis complex DNA in the PCR examination and the absence of malignant cells in the biopsy sample, together with the findings of granulomatous inflammation.

Anti-tuberculosis drugs should be given in the treatment of skeletal tuberculosis. While many authors in the literature suggest that medical treatment alone is sufficient, some believe that surgical debridement and drainage are necessary. Surgical intervention may be needed in refractory or complicated cases, advanced lesions with caseation, or nonresponsive patients to drug therapy[7,8]. The medical treatment of skeletal tuberculosis includes 9-12 months of drug therapy[11]. Our patient received isoniazid, rifampicin, ethambutol, and pyrazinamide for six months and responded clinically and radiologically.

In conclusion, although clinical and radiological evaluations indicate malignancy in children presenting with a mass in the chest wall, it should be kept in mind that there may be tuberculosis infection in the etiology, and lung lesions may not accompany the findings.

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Case Report

Journal of Emergency Medicine Case Reports

An Unusual Pediatric Monteggia Equivalent Lesion: A Rare Case Report

Metin Celik[1], Emre Arikan[2], Omer Faruk Yilmaz[3]

[1]Department of Orthopaedics and Traumatology, Malatya Education and Research Hospital, Malatya, Turkey.

[2]Department of Orthopaedics and Traumatology, Cekirge State Hospital, Bursa, Turkey.

[3]Department of Orthopaedics and Traumatology, Bolu Abant Izzet Baysal University, Bolu, Turkey.

Abstract

Monteggia lesion is a rare fracture in which ulna shaft fracture and radial head dislocation occur together. Monteggia equivalent is the lesion associated with ulna shaft fracture and radius head and/or neck fracture. We offer a case of Monteggia equivalent in a 9-years-old boy. In the surgery of the patient, anatomical plating was performed for ulna shaft fracture and intramedullary nail was applied for radius neck fracture. When we look at the literature, such case reports are very rare. In the 1-year follow-up of the patient, there was no loss in flexion, extension, supination and pronation range of motion.

Keywords: Internal fixation, monteggia equivalent, radius neck fracture, ulna shaft fracture

Introduction

Monteggia is an uncommon lesion where fracture of the ulna is accompanied by dislocation of the radio-humeral joint. It was first expressed by Milanian Giovanni Batista Monteggia in 1814[1]. Monteggia fracture dislocation is a lesion that occurs in less than 1% of all pediatric elbow injuries[2]. In addition, lesions named as Monteggia equivalent were defined as private forms by Bado. These lesions are even less common in children than a Monteggia fracture[1]. In the literature, Monteggia equivalent lesions associated with both ulna shaft or olecranon fracture and radius neck or head fracture have been reported rarely. We present our Monteggia equivalent case associated with 1/3 proximal fracture of the ulna and radius neck fracture.

Case Report

A 9-years-old boy fell from a height of 3 meters and applied to the emergency room. The patient had an isolated right forearm and right elbow injury. On physical examination, there was deformity and limitation of motion in the elbow and forearm. Neuro-vascular and skin examination were normal. Bidirectional radiograph of the elbow revealed a fracture in the radius neck and 1/3 proximal of the ulna without dislocation. (Figure 1). The patient was operated on the same day. After osteosynthesis with a LCDCP plate with a posterio-medial approach to the ulna, it was observed that the radius neck fracture was not reduced under fluoroscopic control. Considering the patient's age and the shape of the deformity, the radius neck fracture was reduced using the Metaizeau technique (Figure 2). No postoperative complications were observed. Postoperative immobilization was provided on the splint for 4 weeks. The intramedullary nail was removed after 3 months. The clinical and radiological examination was uneventful in the patient's controls. In the 1-year follow-up, the fracture lines were completely fused, the elbow was stable, painless, and the movements were complete (Figure 3) (Figure 4).

In this case report, written informed consent was obtained from the patient and his parents for the publication of all images.

Discussion

Monteggia lesion, by Giovanni Battista Monteggia in 1814; It is defined as a forearm injury in which ulna fracture and radial head dislocation occur together. Monteggia injury is very rare in children[3]. Monteggia lesion occurs in less than 1% of pediatric fractures and can be considered a rare injury with an incidence of 2% to 5% of forearm fractures[2]. We thought to share our extremely rare patient with ulna shaft fracture and ipsilateral radius neck fracture in the literature (Figure 1).

Corresponding Author: Emre Arıkan
e-mail: dremrearikan@gmail.com
Received: 19.06.2022 • **Accepted:** 07.07.2022
DOI: 10.33706/jemcr.1132878

Cite this article as: Celik M, Arikan E, Yilmaz OF. An unusual pediatric monteggia equivalent lesion: a rare case report. Journal of Emergency Medicine Case Reports. 2022;13(4): 118-120

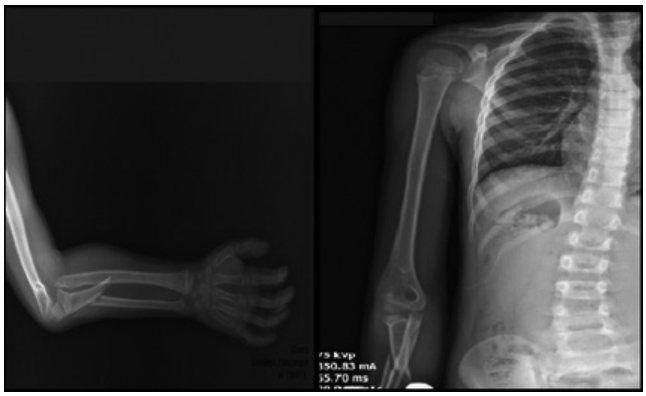

Figure 1. Images before surgery

Childhood Monteggia fracture-dislocations occur as a result of low-energy trauma, whereas in adults, they occur with high-energy trauma[4, 5]. Although Monteggia equivalent injuries are mentioned in the literature, associated ipsilateral radial neck fractures are rare. The mechanism of injury for these types of fractures occurring at two different levels has not been fully resolved. As the post-traumatic force acts on the elbow in the axial direction, a varus rotational force is activated, which may be responsible for the Monteggia lesion. The direction of the rotational force causes different Monteggia fractures[3]. In children, the looser annular ligament makes the Monteggia-equivalent lesions we have presented extremely rare [6, 7]

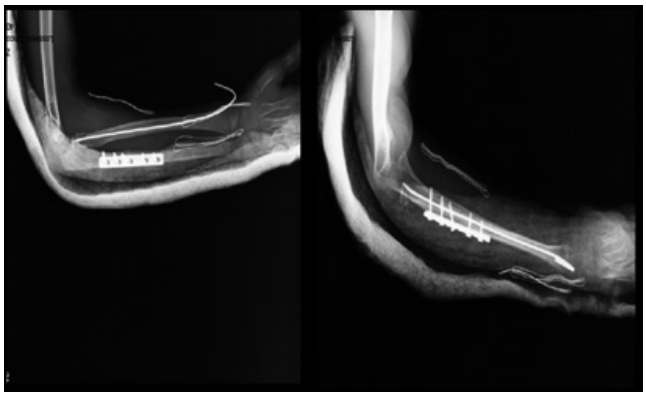

Figure 2. Postoperative images

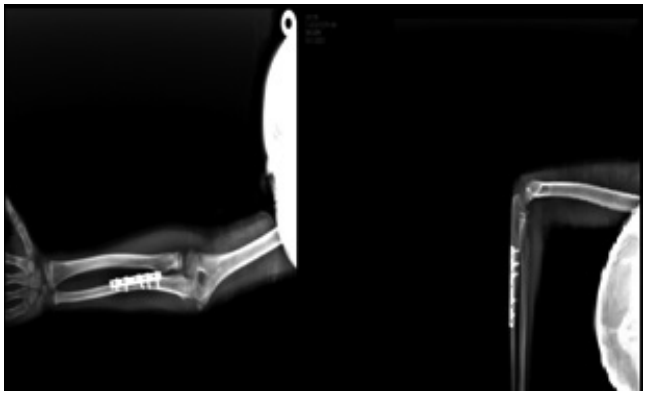

Figure 3. Follow-up images

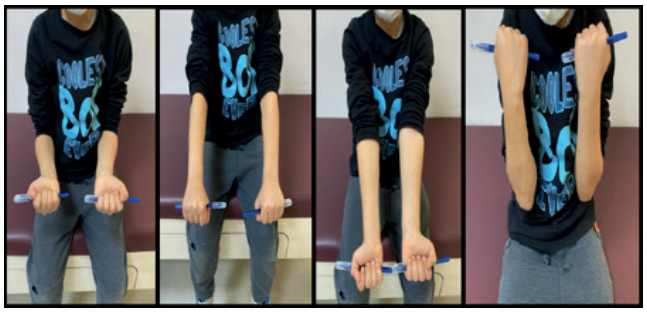

Figure 4. Functional results at the 1-year follow-up

Bado suggested that Monteggia type I and equivalent lesions can be treated by closed reduction with a supination maneuver. In a type I equivalent lesion, if the ulna or proximal radius fracture is unstable, intramedullary fixation should be made with a Kirschner wire[1].

Evans et al observed the injury pattern and suggested that the soft tissue component may be responsible[8]. He emphasized that when a person falls on his elbow in extension, the sudden and strong contraction of the biceps muscle will cause the radial head to come forward. Also, fracture of the ulna occurs as a result of contraction of both the interosseous membrane and the musculus brachialis[9].

In most cases, the radial head is spontaneously reduced after the ulna is anatomically reduced and fixed. Incorrect and inadequate reduction of the ulna can make reduction difficult and cause permanent dislocation of the radial head. If the radial head is dislocated after ulna reduction and fixation, the ligamentous structures of the radial head should be checked or interposition of the posterior interosseous nerve should be considered. In such cases, it can be intervened with a lateral approach[10]. If the fracture structure is short-oblique or transverse, it can be fixed with intramedullary nailing; however, if the fracture structure is long segmented or fragmented, fixation with plate-screw may be required[11, 12]. The looser annular ligament in children makes Monteggia equivalent lesions less common[13]. In the cases presented similarly in the literature, good results were reported in the treatment and follow-up, as in our case.

Conclusion

There are many variations of Monteggia equivalent lesions. In conclusion, the combination of ulna shaft and displaced radius neck fracture is a rare injury. Early diagnosis of these fractures is made by careful examination of the fracture pattern and identification of the injury mechanism. Prompt diagnosis and treatment is imperative to avoid complications. Anatomical fixation of the ulna fracture and stable reduction of the radius head or neck, dislocation or fracture should be provided for good functional outcome. This rare case is presented as a contribution to the literature.

DOI: 10.33706/jemcr.1132878
Journal of Emergency Medicine Case Reports. 2022;13(4): 118-120

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Case Report

Journal of Emergency Medicine Case Reports

Diagnosis of Transient Brain Lesion in the Corpus Callosum Splenium in Emergency Service and Elucidation of Accompanying Conditions

Mehmet Yılmaz[1], Ali Kemal Erenler[2]

[1] Hitit University Erol Olçok Education and Research Hospital, Department of Emergency Medicine, Çorum, Turkey.

[2] Hitit University, School of Medicine, Department of Emergency Medicine, Çorum, Turkey.

Abstract

Corpus Callosum Cytotoxic Lesion (CLOCCs) once rarely seen in the literature has been more often diagnosed in emergency services nowadays with widespread use of cranial magnetic resonance imaging (MRI). CLOCCs is defined as a clinical and radiological spectrum disorder. Patient's neurological symptoms usually improve completely within 1 month after the onset of the disease without any sequel. This is generally associated with cytotoxic edema of the splenium corpus callosum. It is important investigate the primary causes that lead to this condition and start the appropriate treatment according to the real diagnosis. We present a case diagnosed as CLOCCs secondary to pneumonia upon admission to our emergency service.

Keywords: Corpus callosum, splenium, transient brain lesion

Introduction

Splenium is the name given to the posterior segment of corpus callosum (CC). In Greek, it means bandage wrapped around a wound. According to anatomical monitoring studies, splenium fiber composition is heterogeneous: while anterior segment contains thin late myelinating fibers emanating from parietal and medial temporal conjunction area, posterior segment contains thick early myelinating fibers connecting primary/secondary visual fields. Majority of splenium fibers are mixed type and they connect hemispheres to each other homotophically (1). Basic physiological effects of CC are conceptualized as stimulation and inhibition. Specifically, while stimulation means tendency of a region in one hemisphere to activate a symmetrical region, inhibition means opposite. Except agenesis of CC, there is not specific pathology it is included (2).

Corpus Callosum Cytotoxic Lesion (CLOCCs), first introduced by Tada et al in the year 2004 with the name of Reversible Splenium Lesion (MERS) is clinically defined as a clinical and radiological syndrome with mild encephalitis/encephalopathy symptoms (3). This phenomenon, later more broadly named as CLOCCs, was defined as a clinical and radiological spectrum disorder (4). Patient's neurological symptoms usually improve completely within 1 month after the onset of the disease without any sequel. However, studies conducted over time have revealed 3 following features. First, condition is not always mild and might be severe rarely. Second, except viral encephalitis/encephalopathy various diseases and conditions also are defined ad CLOCCs. Third, CLOCCs is not always completely reversible. In the light of these 3 features, CLOCCs illustrated by cranial magnetic resonance imaging (MRI) is considered to be secondary to other disorders (5).

In cranial magnetic resonance imaging, it is visualized as hypointense in T1 and FLAIR and hyperintense in T2 sequences (h). In diffusion weighted MRI, CLOCCs displays itself as low diffusion fields. CLOCCs can't be visualized with contrast weighted imaging. Lesions tend to be on the midline and relatively symmetric. Involvement of corpus callosum shows one of 3 typical patterns: 1- a small round or oval lesion in the center of splenium, 2- a lesion that is in the center of splenium but extends from callosal fibers laterally to the adjacent white matter or 3- a lesion that is central to splenium but extends to anterior corpus callosum. These lesions are generally reversible, but not always (6). On the other hand, cranial computed tomography (CT) might be nonspecific. Cytotoxic edema is the most common cause of the etiopathogenesis of such lesions (7).

Pathophysiological hypothesis of CLOCCs which is believed to cause cytotoxic edema through cytokines is as

Corresponding Author: Mehmet Yılmaz **e-mail:** myilmazm@gmail.com

Received: 28.06.2022 • **Accepted:** 16.08.2022

DOI: 10.33706/jemcr.1137059

Cite this article as: Yilmaz M, Erenler AK. Diagnosis of transient brain lesion in the corpus callosum splenium in emergency service and elucidation of accompanying conditions. Journal of Emergency Medicine Case Reports. 2022;13(4): 121-124

DOI: 10.33706/jemcr.1137059
Journal of Emergency Medicine Case Reports. 2022;13(4): 121-124

follows: first tumor necrosis factor alpha, and cytokines like interleukin-1(IL1) and interleukin-6 cause endothelial damage. Also, tumor necrosis factor alpha and IL1 stimulate astrocytes to produce vascular endothelial growth factor, and this in turn weakens tight connections of brain vascular system and impairs blood-brain barrier (8). In addition, IL1 might induce astrocytes to uptake glutamate, and this triggers glutamate-glutamine cycle and then increases extracellular glutamine levels. Intracellular ATP consumption that causes mitochondrial dysfunction and oxidative stress is induced by activated glutamate-glutamine cycle. As a result, influx of excessive extracellular fluid and Na+ into cells is induced and consequently cellular swelling occurs. These all finally lead to cytotoxic edema (9).

Clinical impression is related to underlying pathology rather lesion itself. So, patients might not only present with encephalopathy symptoms but also with symptoms of central nervous system infection, and nonspecific symptoms such as pneumonia, sepsis, nausea, vertigo, fever or symptoms of metabolic disorders (10). Our patient also presented with nausea and confusion.

Case Report

A 57-year-old male patient with congenital speech and hearing impairment and who has been immobile for 2 years were admitted to emergency service with complains of decreased eating and drinking and change in consciousness for 1 week. According to the information taken from his relatives, he also had nausea, vomiting and cough for last few days, but no fever, no epileptic seizure and use of any antiepileptic drug. He had no history of drug use except angiotensin converting enzyme inhibitor for hypertension treatment. His general condition was bad, blood pressure was 110/70mmHg, heart rate was 90 beats/min, respiratory rate was 20 breaths/min, body temperature was 37.7 0 C. He was confused. Glasgow coma scale was 12. Abnormal findings in biochemistry tests of patient whose whole blood count was normal were as follows: Urea: 69 (N:8-48), BUN: 32 (N:4-23), AST: 148 (N:5-50), ALT: 89 (N:5-50), Total Bilirubin: 2.74(N:0.3-1.2), Direct Bilirubin: 2.74(N:0.3-1.2), LDH: 521 (N:5-248), Sodium: 130 (N:136-146), Chlore: 97 (N:101-109). On the PA chest X ray, there was an infiltrative field in right upper zone (Figure 1). On the thorax computerized tomography (CT) taken afterwards, a consolidated field with air bronchogram was observed in the right lung upper lobe posterior segment (Figure 2). Brain CT was normal (Figure 3). On cranial diffusion MRI, an acute diffusion restriction 5mm in diameter was observed in splenium CC (Figure 4a-b). There was no other lesion in cortex and white matter. Whole abdominal ultrasonography (USG) was normal.

In neurology consultation of the patient, CLOCCs was diagnosed, and as there was no history of seizure disorder,

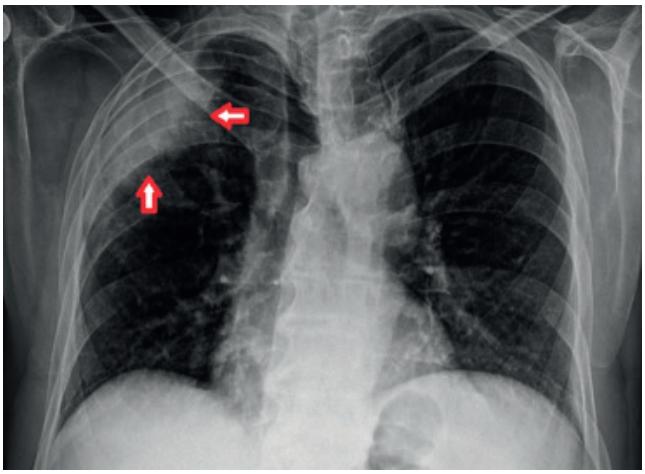

Figure 1. The PA chest X ray, there was an infiltrative field in right upper zone.

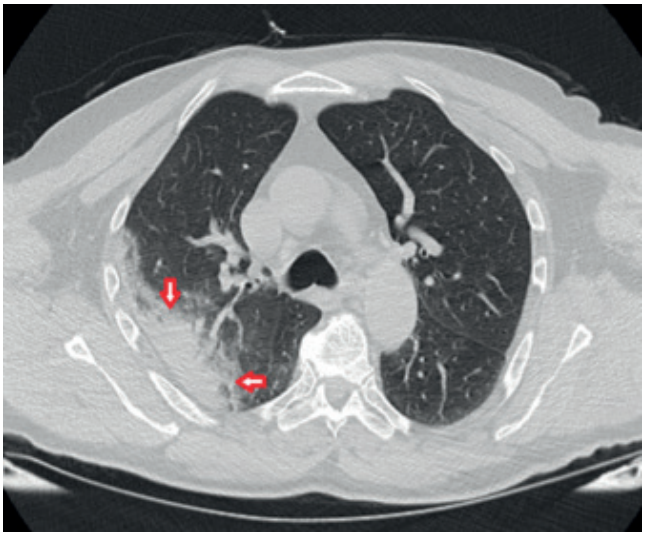

Figure 2. The thorax (CT) taken afterwards, a consolidated field with air bronchogram was observed in the right lung upper lobe posterior segment.

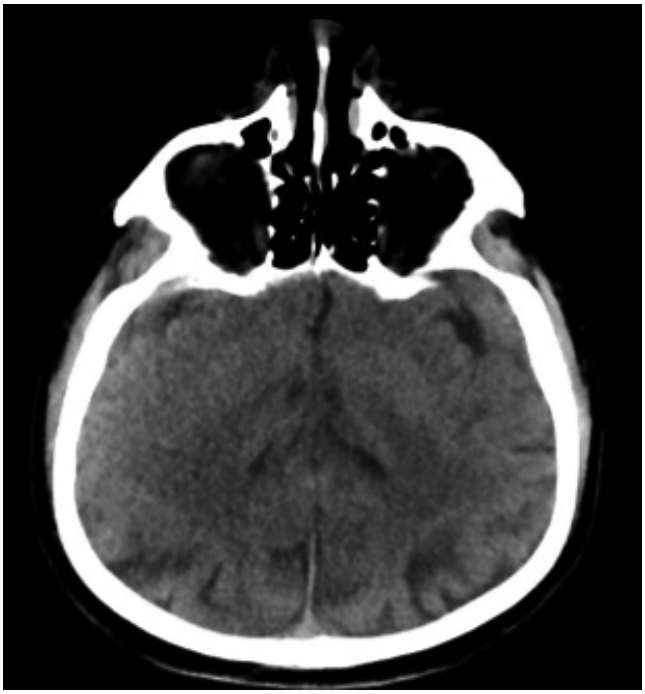

Figure 3. Brain CT was normal.

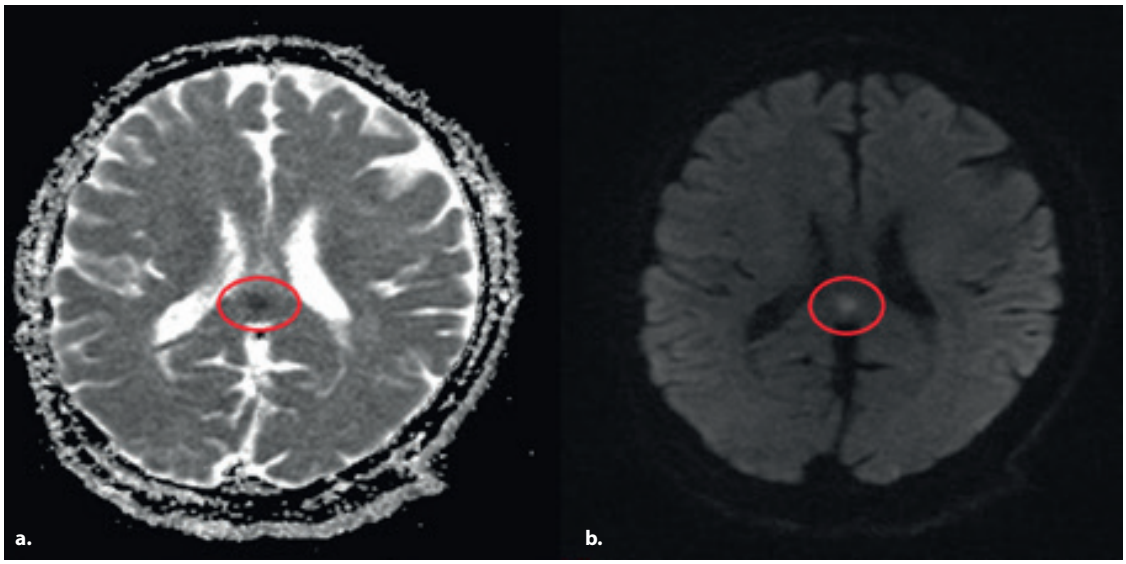

Figure 4a-b. Cranial diffusion MRI, an acute diffusion restriction 5mm in diameter was observed in splenium CC.

or use of antiepileptic drug, neurological pathology was not considered present. It was recommended to investigate metabolic and infectious causes. As whole abdominal USG requested for liver function test disorder was normal, the Department of Gastroenterology was consulted: a Magnetic Resonance Cholangiopancreatography (MRCP) was planned and outpatient clinical control was recommended. After the Department of Pulmonology consultation, patient was admitted to the pulmonology clinic for the treatment of pneumonia.

Discussion

Especially in patients with mixed and nonspecific symptoms that are difficult to diagnose, when CLOCCs is seen one should be alert and underlying primary reasons should be investigated. As Eren et al suggested in their study, CLOCCs might be associated with epilepsy and antiepileptic drugs (43.36%) (7). Although it is the most common cause, our patient had no history of epileptic seizure and no use of antiepileptic drugs. CLOCCs might also be associated with other neurological disorders such as multiple sclerosis (MS), hydrocephalus, subarachnoid bleeding, and ischemic stroke. However, in our patient Brain CT was normal, and history or clinic of MS was not present. In addition, in diffusion MRI there was no other ischemic area. The second most common cause is infective with 33.63% (5). As stated in the study of Şimşek et al, it might be related to central nervous system infections such as meningitis, and encephalitis or immune deficiency (10). Physical examination of our patient did not reveal any finding like fever, neck stiffness or headache. Apart from central nervous system, viral or bacterial infections (influenza virus, rotavirus, measles virus, adenovirus, human parvovirus B19, cytomegalovirus and Mycoplasma pneumonia, Legionella pneumophila, Streptococcus pneumonia and malaria parasites) also might trigger CLOCCs as in our case. Thoracal images of our patient were compatible with bacterial lobar pneumonia. And also, other less common causes (10.62%), metabolism related conditions (5.31%) and high altitude (7.80%) might also lead to CLOCCs (5). Despite further investigations for our patient's liver function test disorder, no liver pathology was detected; MRCP taken after his admission was reported as normal.

Conclusion

Corpus Callosum Cytotoxic Lesion (CLOCCs) once rarely seen in the literature has been more often diagnosed in emergency services nowadays with widespread use of cranial magnetic resonance imaging (MRI). Emergency physician should diagnose this lesion and start the investigation of primary causes and make appropriate consultations for exact diagnosis related to patient's clinic without delay.

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Case Report

Journal of Emergency Medicine Case Reports

Bilateral Elbow Dislocation Without Fracture After Minor Trauma in a Healthy Adult Man

Nafis Vural[1], Murat Duyan[2]

[1] Department of Emergency Medicine, Konya Ereğli State Hospital, Konya, Turkey.

[2] Department of Emergency Medicine, Antalya Training and Research Hospital, Antalya, Turkey.

Abstract

The elbow is the second most frequently dislocated major joint in adults. Elbow dislocation is a common traumatic injury, occurring in approximately 20% of all joint dislocations. Falling onto an outstretched hand is the most prevalent cause of elbow dislocation. Simultaneous bilateral elbow dislocation is a rare presentation, and reports of similar injuries are very limited. A 21-year-old male patient was brought to the emergency department after landing on both hands with flexed elbows from a height of one meter while doing home decoration work. The posterolateral dislocations was detected in both elbows without a fracture on the plain radiographs. The patient's elbows were reduced under procedural sedation analgesia in the emergency department. The patient's splints were removed in the orthopedics outpatient clinic after a 2-week follow-up, and he had no complications. It should be noted that serious bilateral dislocations can occur even in minor traumas like this. Procedural sedation not only prevents the delay of the procedure and the occurrence of complications, especially during the treatment of such multiple dislocations, but also increases patient comfort.

Keywords: bilateral elbow dislocation, elbow dislocation without fracture, minör trauma, work accident

Introduction

The elbow is the second most frequently dislocated major joint in adults. Elbow dislocation affects 6 out of every 100,000 people in their lifetime (1). Elbow dislocation is a common traumatic injury, occurring in approximately 20% of all joint dislocations (2). Falling onto an outstretched hand is the most prevalent cause of elbow dislocation (3). Posterior or posterolateral dislocations constitute 90% of elbow dislocations (1). Sporting activities account for approximately 40% of all elbow dislocations (4). A study conducted in The United States showed that elbow dislocations were most common in football, wrestling, and basketball for male, while gymnastics and skating were the most common for female (5)with use of the National Electronic Injury Surveillance System (NEISS.

Simultaneous bilateral elbow dislocation is a rare presentation, and reports of similar injuries are very limited. In this article, we presented a case of bilateral elbow dislocation without fracture caused by low-energy trauma at work.

Case Report

A 21-year-old male patient was brought to the emergency department after landing on both hands with flexed elbows from a height of one meter while doing home decoration work. The patient complained of pain in both elbows and limitation of joint movements. The patient had no history of dislocation or joint laxity and no known chronic medical disease. The patient's clinical examination showed swelling in both elbows, tenderness on palpation, deformity, and severe limitation in joint range. The bilateral radial pulse was clear and equal. While the patient had no neurological deficit in the right upper extremity, there was a slight decrease in sensation in the fifth finger in the left upper extremity in accordance with the distribution of the ulnar nerve. Other systemic examinations revealed no other pathological findings. The posterolateral dislocations was detected in both elbows without a fracture on the plain radiographs of the patient (figure 1).

Firstly, procedural sedation analgesia (75 mcg of fentanyl followed by 40 mg of propofol) was administered. After the patient was relaxed and sedated, the following

Corresponding Author: Nafis Vural **e-mail:** 42nafisvural@gmail.com
Received: 01.07.2022 • **Revision:** 27.11.2022 • **Accepted:** 29.11.2022
DOI: 10.33706/jemcr.1139501

Cite this article as: Vural N, Duyan M. Recurrent temporomandibular joint dislocation due to antiemetic induced acute dystonia: a case report. Journal of Emergency Medicine Case Reports. 2022;13(4): 125-127

DOI: 10.33706/jemcr.1139501
Journal of Emergency Medicine Case Reports. 2022;13(4): 125-127

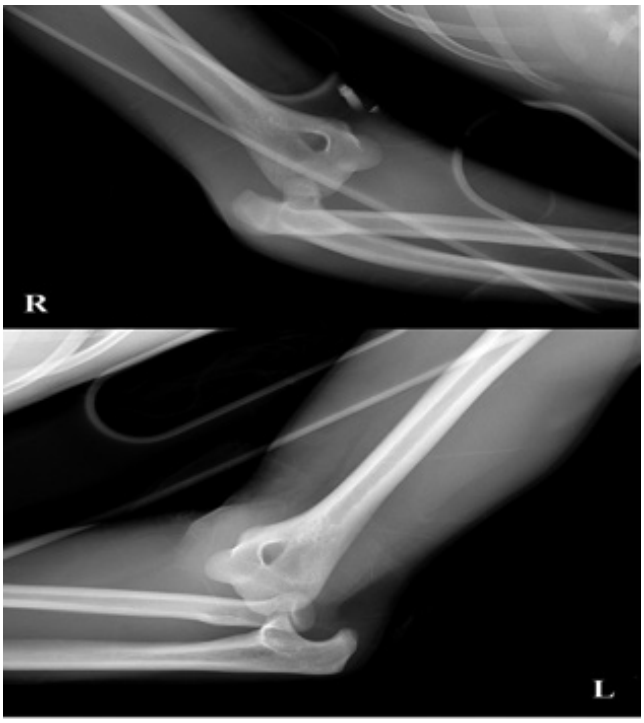

Figure 1. Radiograph of both elbows of the patient before reduction (bilateral posterolateral dislocation)

steps were followed to maneuver with the emergency and orthopedic physician: The patient's elbow was brought to traction, slightly rotated, and closed reduction was completed by flexing it after a gentle manipulation. Then, both elbows were stabilized with a splint at 90 degrees of flexion. The patient was re-applied to radiography, and the reduction was found to be successful (figure 2). Moreover,

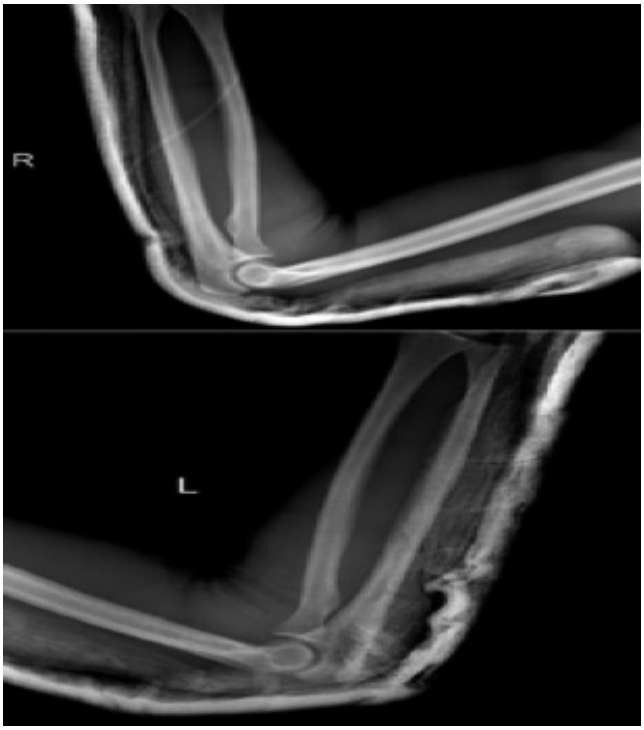

Figure 2. Radiograph of both elbows of the patient after reduction (successful reduction)

joint computed tomography (CT) was performed to evaluate for intra-articular or minor nondisplaced fracture, and no fracture was observed. After the patient had been observed in the emergency department for a while and had recovered from the sedation effect, a control examination was performed, and no neurovascular deficit was found. The patient was discharged with recommendations for elevation, cold application, analgesic, and orthopedic outpatient control, since his general condition was good and he had no fractures. The patient's splints were removed in the orthopedics outpatient clinic after a 2-week follow-up, and he had no complications.

Discussion

Elbow dislocations can be classified as simple or complex. A simple dislocation involves injury to the capsular or ligamentous structures only. A complex dislocation involves fractures of the surrounding bone structure. These fractures usually occur in the radial head, coronoid process, olecranon, distal humerus, and medial or lateral epicondyle of the humerus (6). Although there was bilateral elbow dislocation in our case, there was no displaced or nondisplaced fracture. During an elbow dislocation, soft-tissue structures can also be harmed. Soft tissue deterioration that starts laterally and progresses anteriorly and posteriorly to the medial side with increasing degrees of subluxation is known as the "circle of Horii" (7)"mendeley":{"formattedCitation":"(7.

Cases of bilateral elbow dislocation have more powerful energy mechanisms than cases of unilateral elbow dislocation (8). Mechanisms such as falling off a cross-country bike, falling from a broken ladder, and sports injuries have been documented in the literatüre (9). A gymnast with hyperlaxity is also among the reported cases (10). Furthermore, the mechanism in our case is not falling on an outstretched hand, unlike most elbow dislocations, but falling forward while both elbows are flexed. Consequently, our patient is a rare case in terms of both low energy and mechanism.

The second-decade group is responsible for approximately half (43.5%) of elbow dislocations. Dislocations are also more common in males than in females (3). In this regard, our case is consistent with the literature.

Closed reduction is usually the first line of treatment and is generally performed in the emergency department. Intravenous sedation is generally recommended in the emergency department for adequate relaxation during reduction. Sedation has been shown to reduce the length of stay in the emergency department and improve patient satisfaction (3). Re-evaluation of the neurovascular system is advised to ensure that the reduction has not resulted in any arterial or nerve damage (6).

Patients could continue nonsurgical treatment after adequate reduction with a stable joint, usually with a

posterior long-arm splint at 90 degrees of flexion. After two weeks, if the joint is stable, the splint can be removed, and physical therapy is initiated using range-of-motion exercises to prevent loss of terminal extension (3). It is often beneficial to begin a supervised range of motion in order to avoid prolonged immobilization (1).

Conclusion

In the medical literature, bilateral elbow dislocation is a rare condition. This case is even rarer due to the mechanism of occurrence. Emergency department physicians must be experienced in the management of elbow dislocations. It should be noted that serious bilateral dislocations can occur even in minor traumas like this. In addition, procedural sedation not only prevents procedure delays and complications, especially when treating multiple dislocations, but it also improves patient comfort.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Postoperative Complication of Acute Appendicitis: Portal Vein Thrombosis Required Small Intestine Resection

Tolga Kalayci[1]

[1] Department of General Surgery, Erzurum Regional Education and Research Hospital, Turkey.

Abstract

This case report aims to present the diagnosis and treatment process of portal vein thrombosis, which occurred one week after laparoscopic appendectomy and required small bowel resection. A thirty-eight-year-old man was admitted with abdominal pain in the periumbilical and epigastric regions. He had a history of appendectomy and occlusive cerebrovascular disease. In the physical examination of the abdomen, tenderness was detected in the epigastric region on deep palpation. Leucocytosis, increased levels of alanine transaminase level, aspartate transaminase, gamma-glutamyl transferase, lactate dehydrogenase, c-reactive protein, and D-dimer were detected in laboratory analyses. A computed tomography scan revealed total thrombus in the portal vein, oedema in the segment of approximately 10 cm in the distal ileum, and free fluid in the pelvic region. Enoxaparin sodium was started. During follow-up, widespread defence and rebound in all quadrants of the abdomen occurred. 20 cm ileal resection with end ileostomy was performed. Enoxaparin sodium treatment was continued. On the 6th day of the service follow-up, the patient had left leg pain, and a subacute thrombus was detected in the common femoral, superficial femoral and deep femoral veins on doppler ultrasonography. Edoxaban tosylate 60 mg tablet every 24 hours started as an anti-coagulant treatment, and the patient was discharged without complications on the 18th day of hospitalisation.

Keywords: Appendectomies, enoxaparine, portal vein, thromboses.

Introduction

Acute appendicitis (AA) is the most common cause of acute abdomen in patients of all ages presenting to the emergency department (1). Portal vein thrombosis (PVT) is a rare but important cause of abdominal pain that should be quickly diagnosed and treated (2). It has been reported that the lifetime risk of developing PVT is 1% (3). Intra-abdominal infections, liver diseases, hypercoagulability, and abdominal surgery predispose to PVT (4). PVT usually occurs during clinical signs of acute appendicitis, rarely at the onset of inflammation or after appendectomy in perforated cases as a severe complication.

Early diagnosis of PVT is essential in preventing complications such as gastrointestinal bleeding and mesenteric ischemia. The specificity and sensitivity of ultrasonography (USG), usually chosen for diagnosis, are between 80-100% (5). Computed tomography (CT) shows intraluminal material and helps reveal the possible cause of thrombosis or complications such as perforation and bowel ischemia (6). Anticoagulant therapy, surgical thrombectomy, endovascular thrombectomy and thrombolytic therapy options are treatment methods in patients with PVT. In patients with PVT, it has been reported that the thrombus is recanalised in more than 80% of patients with anticoagulant therapy (7). However, imaging tools indicate diagnostic surgeries (laparoscopy/laparotomy) in cases with severe abdominal pain and suspected ischemia/necrosis.

This case report aims to present the diagnosis and treatment process of portal vein thrombosis, which occurred one week after laparoscopic appendectomy and required small bowel resection.

Case Report

A thirty-eight-year-old man was admitted to the tertiary health centre emergency department with abdominal pain lasting about two days in May 2022. From the beginning, the pain was in the periumbilical and epigastric region and did not show displacement. He underwent a laparoscopic appendectomy one week ago. In addition, he had a history of occlusive cerebrovascular disease about ten years ago, and he was followed up with warfarin about six years ago. The patient with a familial disorder has never had a genetic test before.

On physical examination on admission, his vital findings were as follows: blood pressure: 118/52 mm Hg, pulse rate: 107 beats per minute, oxygen saturation on room air: 97%, and body temperature: 37.3o Celsius. In the physical

Corresponding Author: Tolga Kalayci **e-mail:** dr.tolgakalayci@gmail.com
Received: 06.07.2022 • **Revision:** 17.07.2022 • **Accepted:** 03.08.2022
DOI: 10.33706/jemcr.1141241

Cite this article as: Kalayci T. A rare postoperative complication of acute appendicitis: portal vein thrombosis required small intestine resection. Journal of Emergency Medicine Case Reports. 2022;13(4): 128-131

examination of the abdomen, tenderness was detected in the epigastric region on deep palpation. Leucocytosis (12.70x103/mm3), increased levels of alanine transaminase level (118 U/L), aspartate transaminase (83 U/L), gamma-glutamyl transferase (252 IU/L), lactate dehydrogenase (255 U/L), c-reactive protein (17 mg/L), and D-dimer (4548 ng/mL) were detected at laboratory analyses. Other laboratory parameters were unremarkable, including the lactate level on blood gas (1.6 mmol/L). On the USG evaluation of the abdomen, only minimal fluid was found in the pelvic cavity. CT with intravenous contrast showed increased heterogeneous linear density in the mesenteric fat planes with free fluid in the peri-intestinal area (postoperative changes). The patient was hospitalised in the service. Oral intake stopped, and intravenous fluid replacement started. Ceftriaxone 1 gr vial every 12 hours and metronidazole 500 mg/100 mL every 8 hours started for prophylaxis. On the first day of the follow-up, control USG was obtained, and free fluid with a depth of 50 mm was observed in the pelvic area. In control CT, there was a total thrombus in the portal vein (Figure 1), an oedematous bowel loop in the distal ileum (Figure 2), and free fluid in the pelvic region.

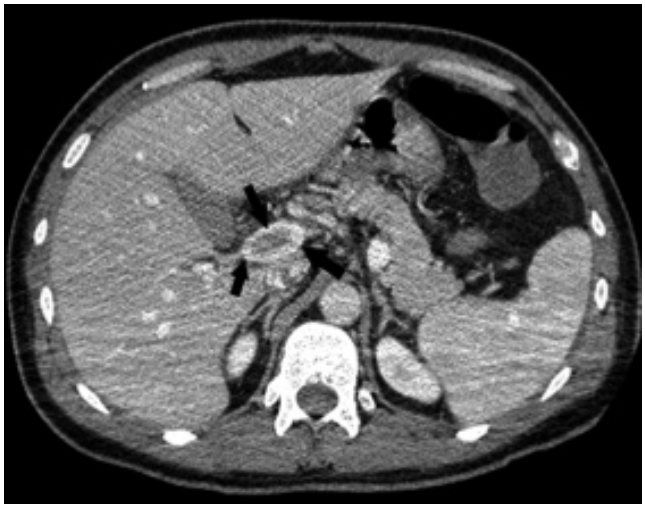

Figure 1. On CT scan, total thrombus in the portal vein is indicated with black arrows.

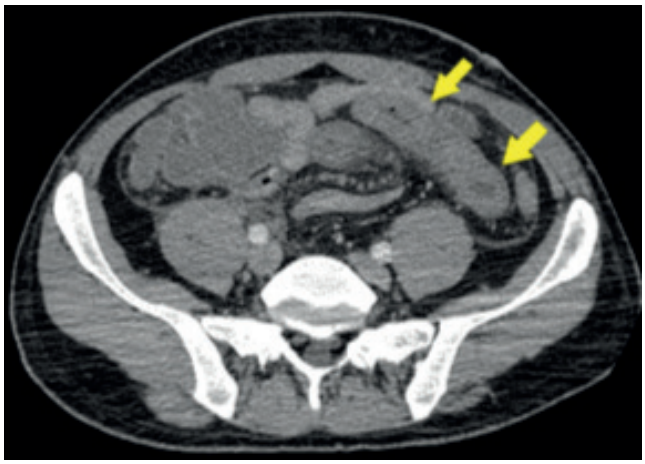

Figure 2. On the CT scan, the oedematous bowel loop in the distal ileum is shown with yellow arrows.

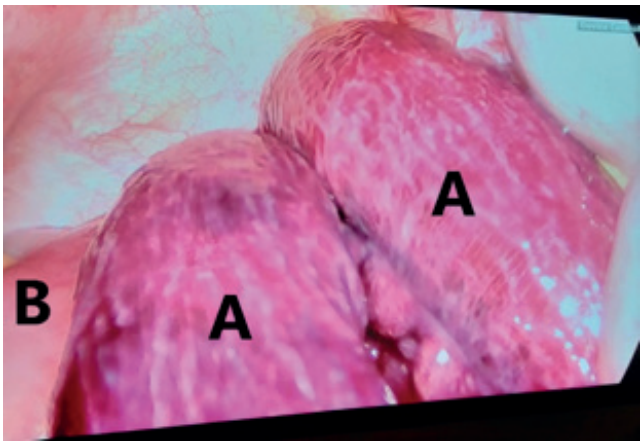

Figure 3. During laparoscopy, there was an oedematous intestinal loop with necrosis in the distal ileum (A: oedematous bowel loop with insufficient blood supply; B: oedematous bowel loop with regular blood supply).

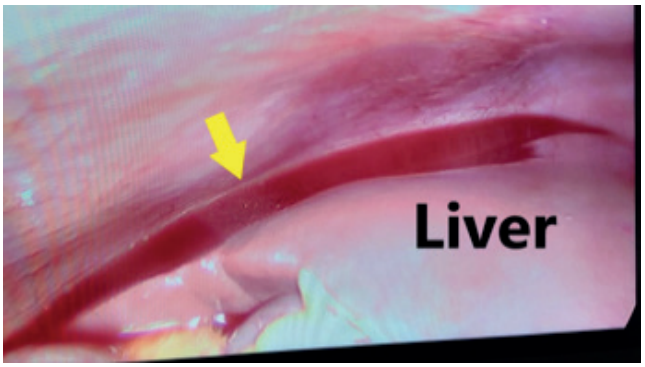

Figure 4. The yellow arrow shows haemorrhagic fluid on the perihepatic area during laparoscopy.

Enoxaparin sodium 6000 U subcutaneously every 12 hours was started. On the 3rd day of the patient's follow-up, there was widespread defence and rebound in all quadrants of the abdomen in the abdominal examination. Emergency laparoscopy was planned. On exploration, there was oedema in the intestinal loops and necrotic intestinal loop in the distal ileum with haemorrhagic fluid in all abdominal cavities (Figures 3 and 4), and laparotomy with midline incision was performed. There was a 5 cm necrotic area, 15 cm ischemic area, and a demarcation line. 20 cm resection and end ileostomy were performed (Figure 5). The patient was taken to the intensive care unit during the postoperative period, and enoxaparin sodium treatment was continued on the 1st postoperative day. Small bowel contents came from the ostomy on the 2nd postoperative day, and the patient was transferred to the service on the 3rd day. On the 6th day of the service follow-up, the patient had left leg pain, and a subacute thrombus was detected in the common femoral, superficial femoral and deep femoral veins on doppler USG. Edoxaban tosylate 60 mg tablet every 24 hours started as an anti-coagulant treatment. The patient was discharged without complications on the 18th day of hospitalisation. Genetic examination of the patient had homozygous polymorphism of both MTHFR A1298C and Plasminogen activator inhibitor (PAI) 4G/4G.

DOI: 10.33706/jemcr.1141241
Journal of Emergency Medicine Case Reports. 2022;13(4): 128-131

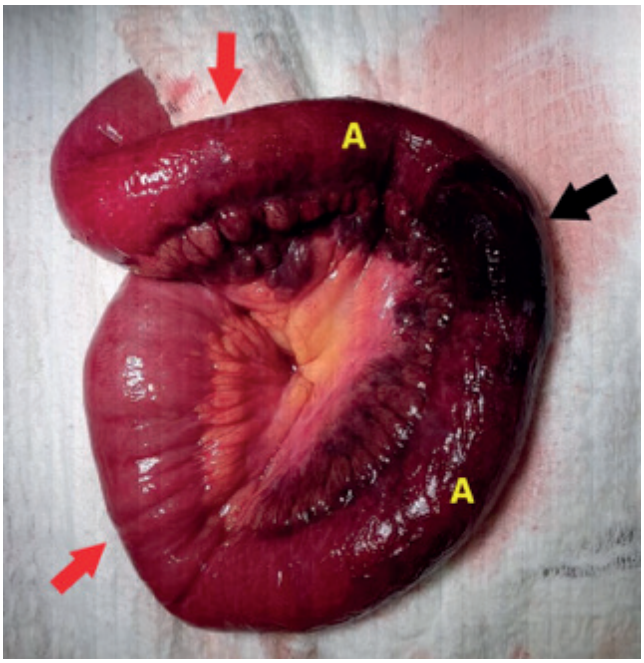

Figure 5. Intraoperative image of the resection material (A: pre-ischemic areas, red arrows show the demarcation lines, and black arrow shows necrotic intestinal loop).

Discussion

Appendectomy is the most commonly applied surgical procedure in emergency conditions at all ages. Among the causes of abdominal pain, portal vein thrombosis (PVT) is rare and often overlooked (8). PVT can be seen in cases of abdominal inflammation such as appendicitis, diverticulitis, inflammatory bowel diseases, pancreatitis, cholecystitis, hepatic abscess and cholangitis, liver cirrhosis, malignancies and hypercoagulability (9). Aetiology includes liver cirrhosis in 24-32% of patients, malignancies in 21-24%, myeloproliferative diseases and coagulation disorders in 10-12% (5). No etiologic cause was found in 8-15% of the patients (10).

The presenting symptoms in PVT depend on the degree of thrombus (partial/total). Although acute thrombus cases are clinically asymptomatic, symptoms such as abdominal pain, distention, diarrhoea, nausea, vomiting and bleeding occur as the thrombus duration increases. PVT does not have a specific laboratory finding and is usually expected without liver disease. However, leucocytosis, a decrease in prothrombin time and other coagulation parameters increase in D-dimer level may occur (11). In addition, etiological causes should be investigated in patients with a confirmed diagnosis of PVT. Prothrombotic events, polycythemia vera, factor V Leiden mutation, prothrombin gene mutation, antithrombin III, and protein C/S levels should be investigated. In patients with a disease prone to thrombosis, such severe PVT can be observed even after an appendectomy, which is frequently performed in general surgery practice, and the diagnosis of PVT should be kept in mind by both emergency physicians and general surgeons in this patient population. In our case, the initial symptom was abdominal pain in the periumbilical and epigastric regions that continued for about two days, and tenderness was detected in the epigastric region on deep palpation. Laboratory analyses revealed increased levels of alanine transaminase, aspartate transaminase, gamma-glutamyl transferase, lactate dehydrogenase, c-reactive protein, and D-dimer with leucocytosis. In addition, the patient had homozygous polymorphism of both MTHFR A1298C and Plasminogen activator inhibitor (PAI) 4G/4G at the genetic examination.

In cases where the diagnosis cannot be made, mesenteric ischemia, liver abscess, septic shock, and pulmonary embolism may develop. Therefore, early diagnosis of PVT is essential to reduce morbidity and mortality. Doppler USG, CT, and magnetic resonance (MR) angiography are diagnostic methods that help detect PVT early. USG is the first method used in diagnosis because it is cheaper and non-invasive. Endo-USG can give more detail about small PVT than routine USG. CT shows intraluminal material. It helps reveal the possible cause of thrombosis or complications such as perforation and bowel ischemia (6), while MR angiography can provide information about thrombus localisation and blood flow (2). In our case, no thrombus was seen at USG and CT on admission, but total portal vein thrombosis was seen at the control CT scan.

PVT treatment aims to prevent the progression of thrombosis, ensure the patency of the portal vein and prevent the development of serious complications. Traditional treatment options are antibiotic therapy, anticoagulant therapy, surgery and the endovascular thrombolytic method. Antibiotic therapy is essential for infection control. Antibiotic therapy, recommended for 4-6 weeks, should initially be an empirical treatment for the common microorganisms and then continue with the appropriate antibiotic according to the blood culture results. Today, anticoagulant therapy is the best way to prevent thrombus progression and provide portal vein recanalisation. Initiation of anticoagulant treatment as soon as the diagnosis is made is one of the most critical factors affecting the healing process. While the rate of recanalisation is 69% in those who start anticoagulant therapy in the first week, this rate drops to 25% with the initiation of treatment in the second week (11). However, it has been reported that 10% of the patients are resistant to anticoagulant therapy, and relapse occurs in 6-40% of those who terminate the treatment early (12). It is recommended to continue oral anticoagulant therapy for at least 3-6 months and evaluate thrombus resolution with intermittent MR angiography or CT (13). In the cases where there is non-responsiveness to medical treatment and diagnoses of persistent abdominal pain, peritonitis, intestinal ischemia, and necrosis, diagnostic laparotomy should be made immediately (14). After our patient was diagnosed with

PVT, subcutaneous anticoagulant therapy with enoxaparin sodium was started, but diagnostic laparoscopy was planned because an acute abdomen developed under anticoagulant treatment. Laparotomic bowel resection was performed due to the necrotic bowel loop observed in laparoscopy.

Conclusion

Portal vein thrombosis (PVT) is a rare but important cause of abdominal pain that should be quickly diagnosed and treated. PVT usually occurs during clinical signs of acute appendicitis, rarely at the onset of inflammation or after appendectomy in perforated cases as a severe complication. However, it can also be seen as a complication of appendectomy. Anticoagulant therapy should be started as soon as possible in patients without persistent abdominal pain, peritonitis, intestinal ischemia and necrosis. On the other hand, diagnostic surgeries (laparoscopy/laparotomy) should be considered in the first-line treatment in patients with the indicated symptoms and signs.

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Case Report

Journal of Emergency Medicine Case Reports

Pseudosubarachnoid Hemorrhage on MRI: A Potential Pitfall

Ozan Karatag[1], Ali Kilinc[1], Bilge Gultac[1], Ibrahim Oztoprak[1]
[1]Department of Radiology, Canakkale Onsekiz Mart University, School of Medicine Canakkale, Turkey

Abstract

Fluid attenuated inversion recovery (FLAIR) is one of the most effective magnetic resonance imaging (MRI) sequences in the diagnosis of subarachnoid hemorrhage (SAH). However, sometimes false positive or false negative results can occur. One of the reasons that can lead to erroneous interpretation is artifacts. Especially when metallic artifact occurs, hyperintensity may be observed in the subarachnoid space, similar to SAH. Although FLAIR hyperintensities in the sulci can be detected in many serious diseases, they are not always pathological. Artifact related hyperintensities, especially in cases with severe headache, may be mistakenly evaluated as SAH by a clinician or radiologist who is not well-experienced in MRI. However, it is extremely important to recognise these artifact related hyperintensities, to make a correct diagnosis and to prevent unnecessary interventions. In order to achieve this, the evaluation of all radiological images, especially SWI and GRE, is critical. Both radiologists and clinicians evaluating neuroradiological examinations should be knowledgeable about this subject and show maximum attention.

In this report, we present the radiological images of 4 cases of pseudosubarachnoid hemorrhage, one of which was caused by conductive EEG gel and the other three due to braces artifacts, who were admitted to the hospital with headache.

Keywords: Magnetic resonance imaging, subarachnoid hemorrhage, subarachnoid space, cerebrospinal fluid, artifacts.

Introduction

Today, fluid attenuated inversion recovery (FLAIR) sequence has become an indispensable part of routine cranial magnetic resonance imaging (MRI) examination[1-3]. In this sequence, while the cerebrospinal fluid (CSF) signal is suppressed with the inversion recovery pulse, heavy T2 images are obtained by applying a long echo time[1-4]. Compared to other conventional sequences, the FLAIR technique is superior in detecting lesions in the subarachnoid space and brain parenchyma, especially near the brain-CSF interface[1].

CSF relaxation time changes when there is pathology in the subarachnoid space. Therefore, in FLAIR sequence, complete suppression of CSF with inversion recovery pulse is interrupted and hyperintensity occurs in the subarachnoid space. Commonly encountered conditions that cause hyperintensity in the CSF/subarachnoid space in FLAIR sequence are subarachnoid hemorrhage (SAH), meningitis, leptomeningeal metastasis, stroke, and status epilepticus.1-4 Also, it has been reported that CSF/subarachnoid space hyperintensity may be present in FLAIR images in cases where propofol, supplemental oxygen, or previous iodine/gadolinium-containing contrast material was applied[2,3,5]. Similar hyperintensities can be observed due to head movements, vascular and CSF pulsation, and metallic body-induced artifacts[1].

In this case series, we aimed to present the radiological findings of 4 artifact-induced subarachnoid hyperintensity cases resembling SAH on FLAIR images, and to discuss the pathologies that may cause similar appearance in the light of literature data.

Case Reports

Case 1

A 13-year-old female with a diagnosis of cerebral palsy and refractory epilepsy was admitted to the pediatric neurology department with a generalized tonic-clonic seizure characterized by locking in the jaw and deviation in the eyes, which had occurred every hour for the past 3 days. In her clinical examination, motor and mental retardation and

Corresponding Author: Ozan Karatag **e-mail:** okaratag17@gmail.com
Received: 10.08.2022 • **Revision:** 22.11.2022 • **Accepted:** 24.11.2022
DOI: 10.33706/jemcr.1160208

Cite this article as: Karatag O, Kilinc A, Gultac B, Oztoprak I. Pseudosubarachnoid hemorrhage on mri: a potential pitfall. Journal of Emergency Medicine Case Reports. 2022;13(4): 132-136

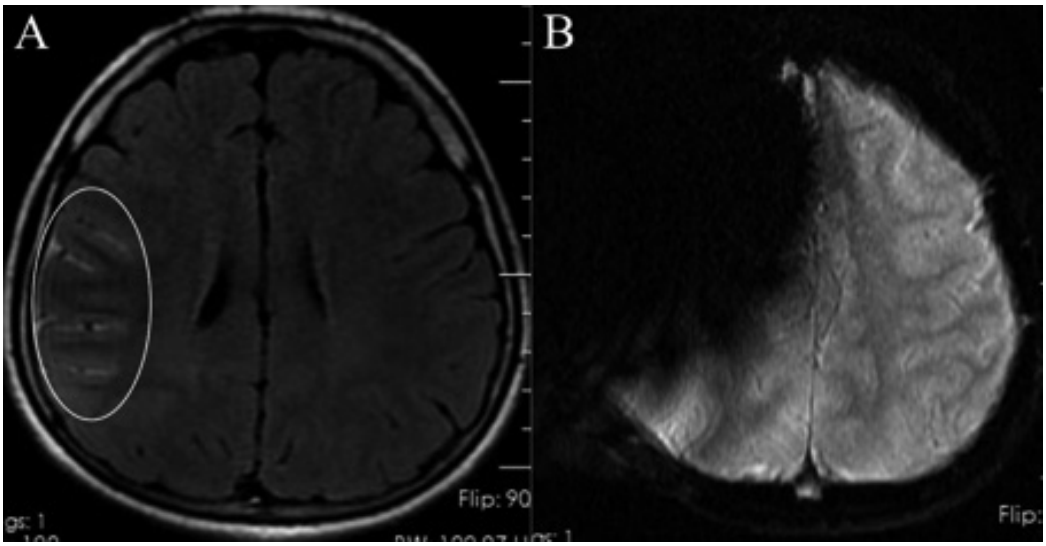

Figure 1. Pseudo-SAH appearance secondary to conductive EEG gel. A, On MRI, FLAIR image shows sulcal hyperintensities in the right frontoparietal region (ellipse). B, Extensive magnetic susceptibility artifact is present in the right frontal region on SWI images.

hypotonia in the lower extremities were detected. She was referred to the radiology department for cranial computed tomography (CT) and MRI examination. CT examination was within normal limits. On MRI, sulcal FLAIR hyperintensities in the right cerebral hemisphere, and extensive susceptibility artifacts in the right frontal area on susceptibility weighted imaging (SWI) were observed (Figure 1). When the patient was retrospectively analyzed in detail, it was learned that an electroencephalography (EEG) was performed before the MRI scan. Sulcal hyperintensities in FLAIR and large susceptibility artifact in SWI were thought to be related to the electrolyte content in the conductive EEG gel.

Case 2

A 15-year-old female was admitted to the pediatric neurology outpatient clinic with headache in the bilateral orbita and frontal region. The patient's physical examination findings were normal. On MRI, FLAIR images revealed hyperintensities in the frontal horns of the lateral ventricle, suprasellar and prepontine cisterns, and frontal sulci on both sides. Signal loss due to susceptibility artifact covering most of the anterior half of the cranium was also observed in SWI images. When the national medical archive information of the case was searched, it was seen that the cranial CT images obtained a few days ago in another center with the same complaints were normal (Figure 2). Sulcal hyperintensities observed in FLAIR images were interpreted as braces-related artifact.

Case 3

A 15-year-old female applied to the pediatric neurology outpatient clinic with occasional numbness in the tongue. The patient's physical examination findings were normal. On MRI, FLAIR hyperintensities were observed in the bilateral frontal sulci and pontocerebellar cisterns. In this case, it was considered that the appearance was related to the braces, due to the artifact extending from the maxillofacial area to the anterior cranium and the intense susceptibility artifact on SWI images (Figure 3).

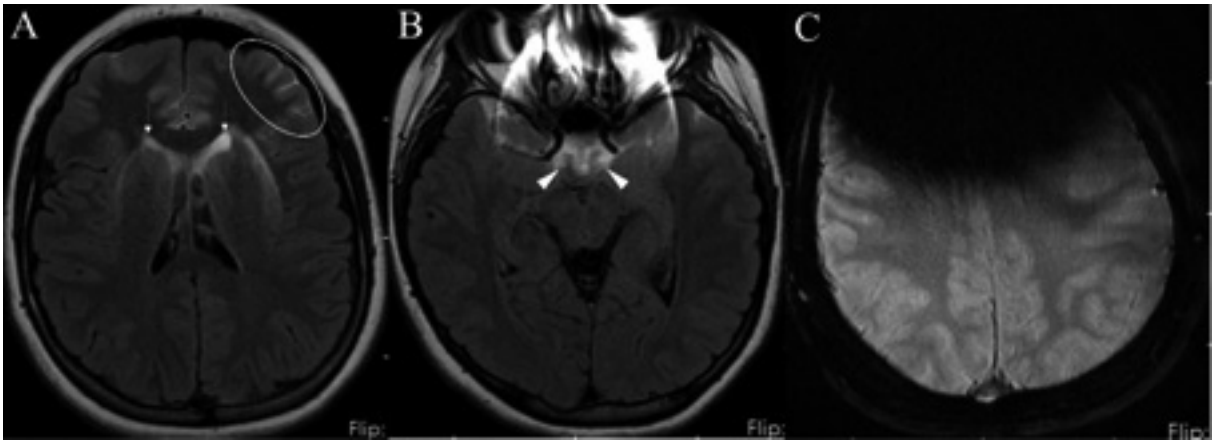

Figure 2. Pseudo-SAH appearance due to braces. A, FLAIR hyperintensities are visible in the left frontal sulci (ellipse) and bilateral lateral ventricular frontal horns (arrows). B, FLAIR hyperintensity at the suprasellar cistern (arrow heads). C, Anterior cranial wide magnetic susceptibility artifact is present on SWI image.

DOI: 10.33706/jemcr.1160208
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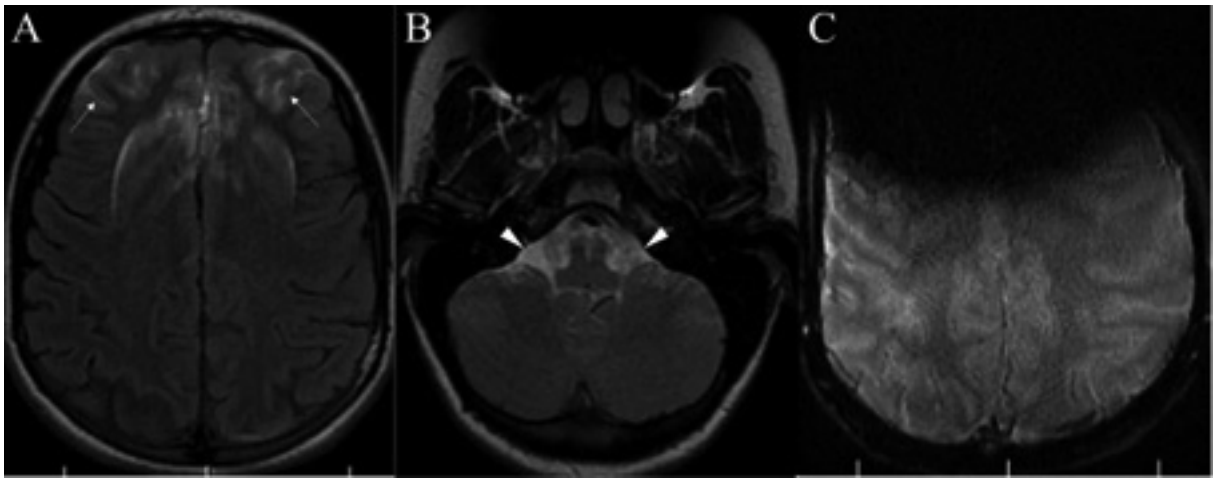

Figure 3. Pseudo-SAH appearance due to braces. A and B, Bifrontal sulcal (arrows) and pontocerebellar cistern (arrow heads) hyperintensities on FLAIR images. C, There is anterior cranial wide magnetic susceptibility artifact on SWI image.

Case 4

An 18-year-old male was admitted to the neurology outpatient clinic due to headache. The patient's physical examination was normal. Kranial MRI examination revealed sulcal hyperintensities that were more prominent in the bilateral frontobasal regions in FLAIR images, while intense susceptibility artifact was present in SWI images (Figure 4). This appearance was also considered to occur due to braces artifact.

Discussion

It has been reported by many researchers that FLAIR imaging is highly sensitive, but not specific, in the detection of many pathologies, especially involving the subarachnoid space[3]. Although in the past years it was accepted that the radiological diagnosis of SAH was easier with CT than with MRI, today FLAIR sequence is known as one of the most sensitive imaging methods for SAH[4]. In SAH, because blood and CSF are mixed, and the oxygen level in CSF is high, the hemoglobin concentration in the hemorrhagic content progresses more slowly. Moreover, SAH is usually of arterial origin; thus hemoglobin is primarily present as oxygenated hemoglobin. In acute SAH, hyperintensity is observed in the subarachnoid space on FLAIR images due to the higher protein content of the bloody CSF. The diagnosis of subacute and chronic SAH is also easier with FLAIR than with CT and other conventional MRI sequences. In addition, this sequence is particularly useful in the diagnosis of SAH in the posterior fossa, which is difficult to evaluate on CT due to beam-hardening artifact[1,4,6].

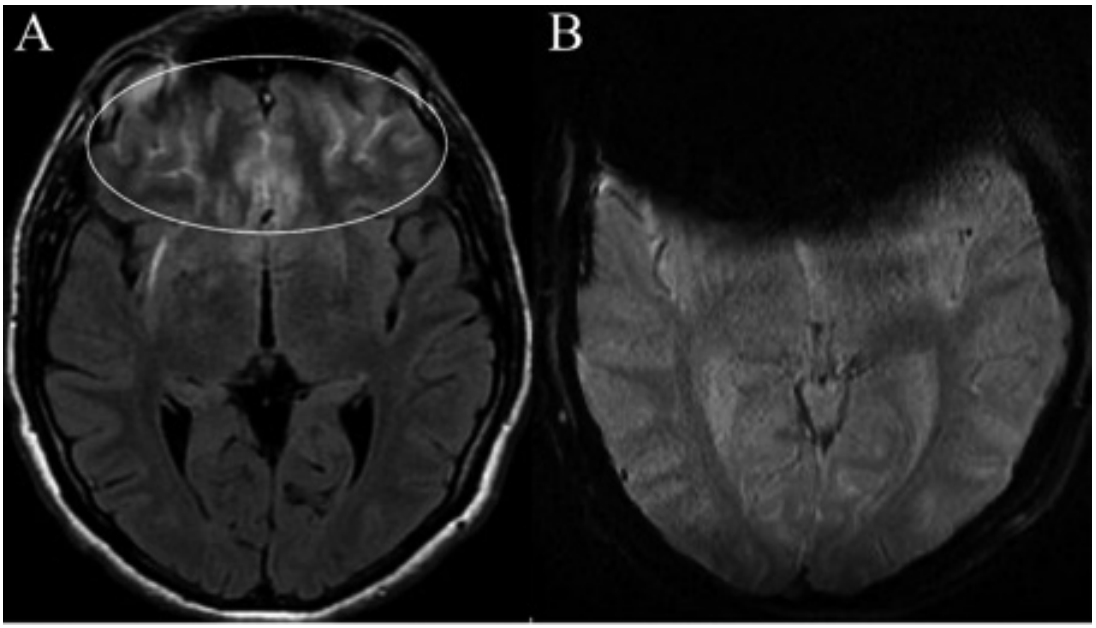

Figure 4. Pseudo-SAH appearance due to braces. A, In FLAIR image, hyperintensities in the bifrontal sulci (ellipse) are seen. B, There is anterior cranial wide magnetic susceptibility artifact in SWI images.

Non-traumatic SAH due to rupture of saccular aneurysm is usually observed at the level of the basal cisternae. In addition, one of the rare causes of SAH close to this region is perimesencephalic hemorrhage. On the other hand, spontaneous cortical SAH, which is mostly observed at the level of convexity, may occur due to vascular and non-vascular causes. These include dural and cortical cerebral vein thrombosis, vascular malformations, reversible cerebral vasoconstriction syndrome, vasculitides, Moyamoya disease, severe atherosclerotic carotid disease, amyloid angiopathy as vascular causes, and cerebral neoplasia and abscess as non-vascular causes. The clinical manifestations of cortical SAH differ from the classic thunderclap headache described in aneurysmal SAH. In these patients, findings such as migraine with aura and epileptic seizures may be observed, in addition to focal deficits that may suggest transient ischemic attack (TIA)[7]. In our cases, similar to the cortical SAH findings mentioned in the literature, sulcal hyperintensities compatible with pseudosubarachnoid hemorrhage were present, and headache in two of the cases, seizure in one and TIA-like clinical findings in one were consistent with the literature data.

Apart from SAH, many other pathological conditions such as meningitis, leptomeningeal metastasis, dural venous thrombosis, stroke, status epilepticus may similarly cause hyperintensity in the subarachnoid space[3,4,6,7]. While the increase in protein and cellularity in the CSF content is responsible for this hyperintensity in meningitis and leptomeningeal metastases, vascular slow flow, congestion, occlusion and thromboembolism are responsible in acute stroke and venous thrombosis[1].

In addition, it has been reported that hyperintensity may occur in the subarachnoid space in patients receiving supplemental oxygen during MRI, intravenous anesthetic agent such as propofol, and previously administered intravenous contrast material containing iodine or Gadolinium[3,4,7].

Artifacts are another important cause of hyperintense CSF in FLAIR images[4]. The displacement of the suppressed CSF due to motion artifacts and metal-induced magnetic susceptibility artifacts may occur[4,5]. While FLAIR hyperintensity due to CSF pulsation is observed mostly at the basal, prepontine and pontocerebellar cisternae levels and in the areas close to the foramen in the ventricles, it is less common in the convexity parts of the cerebral hemispheres where the CSF flow is slow[1]. The reason here is that the pulsatile CSF flow displaces the suppressed CSF protons and causes them to be replaced by unsuppressed protons[5]. Artifacts due to vascular pulsation, on the other hand, appear along the phase coding direction, mostly in the form of ghosting artifacts, having a similar shape, size and location to the vascular structure[1]. Similar artifacts may also be observed due to the patient's head movements.1 As a result of this movement, FLAIR hyperintensity may occur within the subarachnoid space and ventricles when unsuppressed or insufficiently suppressed CSF protons enter the imaging area[5]. Metallic artifact occurs due to insufficient suppression of CSF by inversion pulse due to the inhomogeneous magnetic field caused by the metal in the section[4]. As a result, it leads to a hyperintense appearance in FLAIR in the subarachnoid space[1]. In order to understand whether the sulcal hyperintensities observed in the FLAIR sequence are caused by artifacts, the intense susceptibility artifact in SWI or gradient echo (GRE) sequences that are currently applied in routine cranial MRI practice can be examined. In all of our cases, intense signal loss due to susceptibility artifact was observed in a much larger area than FLAIR sulcal hyperintensities in SWI images.

In conclusion, hyperintensities that can be seen in the subarachnoid space, CSF or sulci on FLAIR images may be an indicator of many serious pathologies, especially SAH. However, similar findings may also occur due to some artifacts. Therefore, both radiologists and clinicians evaluating cranial MRI images should be aware of this aspect. In order to understand whether the hyperintensities of the sulcal/subarachnoid/CSF on FLAIR images in the cranial MRI examinations of the patients who applied to the emergency department or the outpatient clinic with various complaints are due to artifacts, all consecutive sections should be carefully evaluated, and SWI or GRE sequences, if present, should be examined. In this way, the appearance of artifact-induced pseudosubarachnoid hemorrhage is not mistakenly interpreted as pathological, and unnecessary further investigations and interventions are prevented.

Authors' Note : *Ozan Karatag, Ali Kilinc, Bilge Gultac and Ibrahim Oztoprak contributed equally to the manuscript. Written informed consent for patient information and images to be published was provided by the patient. Ozan Karatag, Ali Kilinc, Bilge Gultac and Ibrahim Oztoprak contributed to study concept and design, acquisition of data, data analysis and interpretation, drafting, and revision of manuscript for intellectual content. Ibrahim Oztoprak contributed to revision of manuscript for intellectual content. Study funded by the authors.*

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DOI: 10.33706/jemcr.1160208
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