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Contents

Case Report

Journal of Emergency Medicine Case Reports

Is Every Involuntary Movement Epileptic?

Ramazan Gurlu, Ozlem Tolu Kendir, Omerhan Baspinar, Nilgun Erkek
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Abstract

Introduction: Paroxysmal nonepileptic events are episodic changes in behavior, sensation, or consciousness that are similar to epileptic seizures but not associated with abnormal ictal brain electrophysiological discharges. Here, a case treated as epileptic seizure was presented in order to draw attention to paroxysmal nonepileptic events in differential diagnosis.

Case: A 4 years old girl sent to our hospital with the diagnose of status epilepticus due to change in her consciousness, contractions and abnormal movements in her body, arms and legs those started after taking 6 spoonfull syrup of Peditus® (Containing 120 mg paracetamol, 50 mg guaifenesin, 6.25 mg pyrilamine maleate, 5 mg phenylephrine hcl in 5 ml scale) and 5 Medikinet® 10 mg capsules (10 mg methylphenidate hydrochloride in 1 capsule). She was conscious and cooperate and has involuntary snake-like movements throughout her body on admission. The patient's movement disturbances thought as methylphenidate-induced choreoathetosis responded to given haloperidol treatment and any sign of poisoning were not observed in the patient's follow up.

Conclusion: Chorea side effects were observed in our patient but not any poisoning symptoms, who received a toxic dose of methylphenidate for her age. This suggests that methylphenidate, a central nervous system stimulant, may have therapeutic, toxic dose limits and side effects profile those associated with individual pharmacogenetic variations. Accurate distinction of chorea from drug-related paroxysmal nonepileptic events will ensure early effective treatment of patients and to protect patients from unnecessary drug risks.

Key words: Status epilepticus, Methylphenidate, Korea

Introduction

Epilepsy has an incidence of 40-50/100000 in the childhood age group and is characterized by spontaneous and repetitive seizures resulting from abnormal and excessive electrical discharge in cortical neurons[1]. The most important first step in the management of childhood epilepsy is to decide whether the event described is an epileptic seizure. Paroxysmal nonepileptic events (PNEs) are episodic changes in behavior, sensation, or consciousness that are similar to epileptic seizures but not associated with abnormal ictal brain electrophysiological discharges. While psychogenic seizures and cardiac events constitute the majority of PNEs; parasomnia (sleep gait, sleep terrors and nightmares), movement disorders, narcolepsy, breath-holding spells, Sandifer syndrome, and behavioral events are other types of PNE events. In the literature, it has been reported that PNE events constitute 23% of the patients followed up with a diagnosis of epilepsy in childhood with long-term video EEG[2]. Here, a case treated as epileptic seizure was presented in order to draw attention to paroxysmal nonepileptic events in differential diagnosis.

The Case

A four-year-old girl was referred to our hospital due to change in consciousness, convulsions, and abnormal movements in her body and limbs. Patient epicrisis revealed that the patient had taken 6 spoonfuls of *Peditus^{R}syrup* (5 ml spoonful containing 120 mg paracetamol, 50 mg guaifenesin, 6.25 mg prilamine maleate, and 5 mg phenylephrine HCl) by mistake ~1 hr before admission and was treated with gastric lavage and activated charcoal at the center of first admission. The patient was hospitalized and followed up, then began to have seizures, received two intermittent infusions of 0.25 mg/kg diazepam IV and 20 mg/kg phenytoin IV. Convulsions could not be stopped, and the patient was referred to our center with the diagnosis of status epilepticus. When patient anamnesis was further investigated, it was learned that the patient had also taken five Medikinet®10 mg altered release capsules (10 mg methylphenidate HCl in 1 capsule). The patient had no self or family history of epilepsy or movement disorder, and neuromotor development was appropriate for her age. At the initial evaluation

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of the patient, the airway was clear, spontaneous breathing and circulation were normal. Body weight was 20 kg (90p), vital signs were as follows: fever, 37.1°C; pulse, 131/min; respiratory rate, 24/min, blood pressure, 113/72 mmHg; conscious, and normal orientation and cooperation. Pupils were isocoric, light reflex was bilateral (+). The patient was able to speak meaningfully and regularly, and had involuntary snake like movements throughout the body (Video-1, Video-2). No pathology was detected in systemic examination of the patient. With the diagnosis of choreatetosis due to methylphenidate poisoning, the patient was administered with intramuscular 0.1 mg/kg haloperidol. Abnormal movements stopped within five minutes. Hemogram, kidney and liver function tests, cardiac enzymes, coagulation tests, and paracetamol levels were within normal limits and ECG was evaluated as normal. Patient was discharged without any problems after 48-hours of follow-up.

Discussion

Diazepam and phenytoin treatments were previously administered to our patient with the suspicion of epileptic seizures because of convulsions and abnormal movements in the patient's body, arms, and legs. Patient was referred with a preliminary diagnosis of status epilepticus because she did not respond to the treatment. Persistent snake-like movements were observed in the whole body, and the patient was able to respond normally to verbal stimuli with proper content and articulation. The gold standard in the diagnosis of epileptic generalized tonic clonic seizures is 24-hour video EEG monitoring. However, at first glance, PNE generalized movement disorders can be separated from generalized tonic clonic seizures with no change in consciousness. Difficulties in assessing the state of consciousness, especially in young children, can be misleading.

Chorea is defined as a short, involuntary, and hyperkinetic movement disorder. Many potential causes, including auto-immune processes, infections, hypoxic or ischemic injuries, mitochondrial diseases, and toxins, can be associated with chorea. Drug-associated choreiform movements have been reported due to methamphetamine overdose as well as poisoning with prescription Central Nervous System (CNS) stimulants, high-dose use for therapeutic purposes, or taking CNS stimulants with a second dopaminergic drug[3].

Methylphenidate, a psychostimulating drug, acts mainly by blocking dopamine and norepinephrine reuptake receptors in the synaptic gap, and can also block serotonin reuptake receptors. This leads to an increase of neurotransmitters in the extraneuronal space and prolongation of their effects[4]. At low doses, methylphenidate used in the treatment of Attention Deficit Hyperactivity Disorder (ADHD) reduces movement and impulsiveness by acting in the prefrontal cortex, increasing cognitive function, including attention and working memory. For 3-5 year-old children, methylphenidate treatment is recommended 5-30 mg/day with gradually increasing the dose[4]. It has been reported that doses up to 40 mg are well tolerated in this age range[5].

2-17% of patients may experience side effects such as insomnia, depression, decreased appetite, weight loss, headache, visual impairment, palpitations, and dizziness during methylphenidate treatment. Movement disorders such as tics, tourette syndrome, tremor, dyskinesia, and chorea are rare side effects[6].

It is suggested that excessive dopaminergic activation in striatum, caudate nucleus, or putaments is largely responsible for the choreiform movements[6].

Poisoning with methylphenidate produces symptoms similar to typical sympathomimetic agent toxicity. Psychiatric or neurological effects of varying degrees (e.g., headache, CNS excitation or depression, abnormal movements or rigidity, changes in mood or behavior, hallucinations, paranoia), cardiovascular effects (e.g., hypertension, tachycardia, chest pain) and sometimes gastrointestinal effects (e.g. vomiting, abdominal pain), or various laboratory abnormalities (e.g. high serum transaminases or creatine kinase or thrombocytopenia) can be seen. In some cases, hyperthermia, arrhythmias, and seizures have been reported[5].

The 4-year-old patient, who was referred to our center with preliminary diagnosis of poisoning and subsequent seizures, had received a single dose of 50 mg of methylphenidate. Except for choreiform movements due to methylphenidate, there were no identified signs or symptoms of intoxication during the 48 hour follow-up.

In the literature, the side effect of chorea associated with the use of methylphenidate in therapeutic doses or rapid dose increase for therapeutic purposes has been reported in children[5,6]. This condition is attributed to the prefrontal cortex alpha-2A adrenergic receptor gene polymorphism, which can increase the effectiveness of the drug, or the CES1 gene variation that delays the metabolism of the drug[7,8]. Our patient who had no history of consanguinity have a brother who was receiving 10 mg of methylphenidate daily due to ADHD and he had no side effects such as movement disorder etc..

Animal studies have shown that guaifenesin and phenylephrine taken by our patient with methylphenidate may exhibit anticonvulsant effects, and there is no data in the literature on the effects of their use with methylphenidate[9].

There are numerous studies recommending the use of haloperidol, a postsynaptic dopamine (D2) receptor blocker in the mesolimbic system, in motion disorders due to methylphenidate and other CNS stimulants[10]. Consistent with the literature, we observed that the symptoms regressed and do not recur with haloperidol use.

Conclusion

Even though the patient had received a toxic dose of methylphenidate for her age, there were no signs of toxicity in our patient. Therapeutic and toxic dose limits of CNS stimulants such as methylphenidate may differ individually depending on pharmacogenetic diversity. On the other hand, chorea should be kept in mind in the differential diagnosis of epileptic seizures from PNE events caused by CNS stimulants. Correct distinction will ensure early effective treatment of patients and protect them from unnecessary drug risks.

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Case Report

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An Unexpected Acute Abdomen Case: Torsion of a Wandering Spleen Treated with Splenopexy

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Abstract

Wandering spleen is a rare condition resulting from the absence or looseness of the hanging ligaments that keep the spleen in its normal anatomical location. It can lead to an acute abdomen with torsion and infarction of the splenic pedicle. In this article, we present a patient who came to the hospital with a grievance of severe abdominal pain and was diagnosed with wandering spleen torsion. The patient was treated with splenopexy. Although it is rare, a wandering spleen should be considered in the differential diagnosis of acute abdomen.

Keywords: Acute Abdomen, Wandering Spleen, Torsion,

Introduction

Wandering spleen is a rare condition where the spleen is mobilized from the left upper abdominal quadrant. It causes an abnormal position of the spleen secondary to a developmental abnormality or acquired looseness of the spleen's ligaments. Nearly one-third of all cases of wandering spleen occur in young girls older than one year. In adults, women of childbearing age are more frequently affected[1]. Diagnosis of the wandering spleen can be difficult because its symptoms are variable. It may present as intermittent abdominal pain, abdominal mass, or acute abdomen[2,3].

Case Presentation

We describe here a young woman with torsion of a wandering spleen that was managed by splenopexy. A 29-year–old female was admitted to the emergency service of our hospital with complaints of widespread abdominal pain and nausea that continued for three days. On physical examination, there was a palpaple mass and defense in the right lower quadrant. Her blood pressure was normal. Laboratory parameters showed hemoglobin 10.5 gm/dL and white blood cells 3.89/mm^3. The platelet count was normal. Blood electrolytes, urea, creatinine, and random blood sugar analyzes were normal too.

On ultrasound images, the spleen was not at its normal site. Instead, it was located in the right lower quadrant and increased in size (130x97x78mm). The splenic parenchymal contrast enhancement was reduced. Furthermore, capsular rim-like enhancements were spleen in the computed tomography sections (Figure 1a), and there was a whirled view of the splenic vessels (whirl sign) (Figure 1b). Intestinal loops were observed in the left upper quadrant of the abdomen (Figure 1c).

With a preliminary diagnosis of the torsion of a wandering spleen, the patient was referred to another center where she was operated, and the spleen was detorsioned and moved to the left upper abdominal quadrant (Figure 1d).

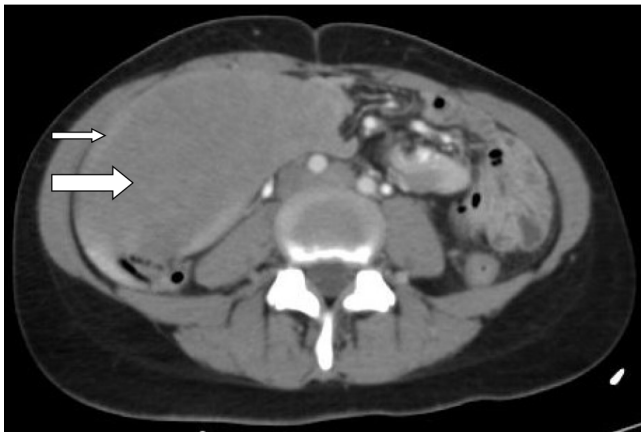

Figure 1a. Contrast-enhanced abdominal CT in axial view: Splenic enhancement like capsular rim (thin arrow) with reduced parenchymal enhancement (thick arrow) located in the right lower quadrant

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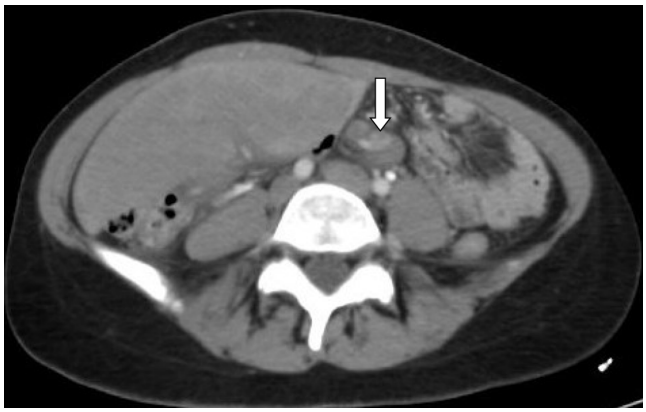

Figure 1b. Contrast-enhanced abdominal CT in axial view showing; the splenic vascular peduncle rotated around itself (whirl sign)

Discussion

Wandering spleen is an unusual clinical condition. Hence, discussions in the literature are limited. Its most common complication (60%) is the torsion of the pedicle which can cause a splenic infarction, sepsis, acute pancreatitis, and gastrointestinal bleeding secondary to portal hypertension or splenic vein thrombosis[4,5]. Other less common complications include intestinal obstruction, gastric volvulus, spontaneous or traumatic spleen rupture[5]. Splenic torsion may be acute or chronic. Acute torsion may mimic peritonitis, acute appendicitis, twisted ovarian cysts, or bowel obstruction[6]. On the other hand, chronic torsion may present as an abdominal mass, which may be located in any quadrant[7]. In this patient, there were signs of a mass in the right lower quadrant with acute abdominal symptoms such as pain, defense, and nausea.

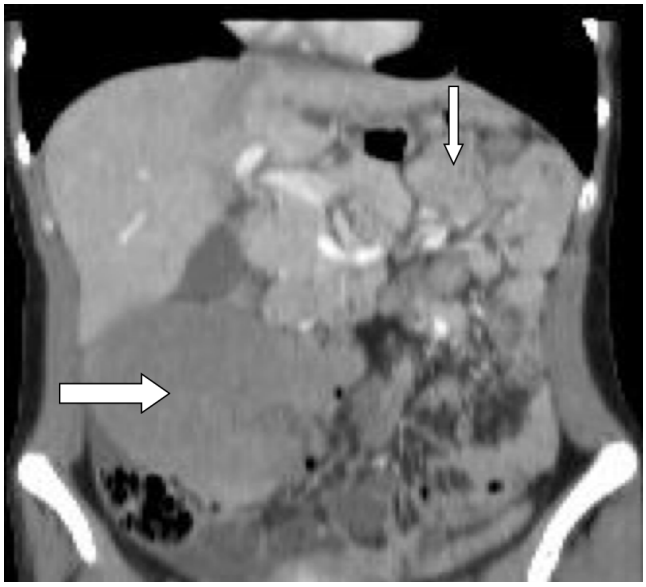

Figure 1c. Coronal reformate image showing the; spleen located in the right lower quadrant (thick arrow) and bowel loops (thin arrow)

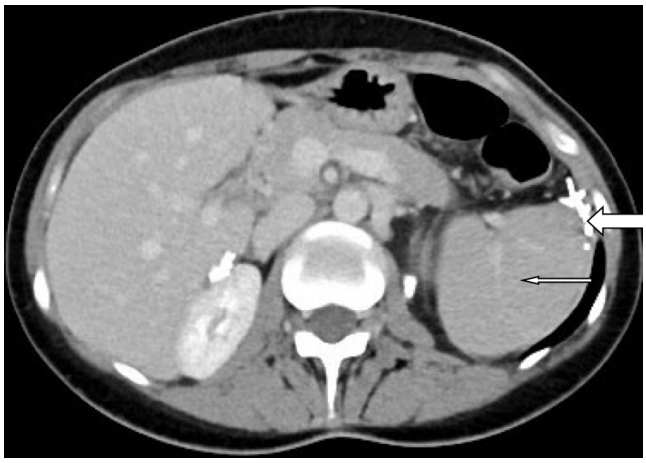

Figure 1d. Spleen (thin arrow) in its normal size and localization and suture materials (thick arrow) after surgery

Radiologists play an essential role in diagnosing the wandering spleen and its complications[8]. The most reliable and least invasive method of making the diagnosis is with ultrasonography, which can demonstrate the characteristics and localization of the spleen. The Doppler ultrasonography provides information about vascular structures, while ectopic position and torsion of the spleen appear on computed tomography and magnetic resonance images. The whirled appearance is a particular sign of torsion of the splenic pedicle[9]. Besides, a loss of parenchymal enhancement and rim enhancement of the splenic capsule are typical findings too In this patient, these findings were present together (Figures 1a, b, and c).

Operative treatment in wandering spleen cases is the definitive option. Splenectomy has been the traditional management of wandering spleen for many years. However, splenopexy may be a suitable method when the splenic vein's recanalization can be exactly proven[10]. Most adult splenic torsion cases in the literature have been treated with splenectomy. On the other hand, this case was admitted to our emergency department relatively early, was diagnosed very quickly, and treated with splenopexy. No complications were found during a one year follow-up.

Conclusion

Wandering spleen is a rare disease. However, increasing awereness and its inclusion in the differential diagnosis enables a rapid diagnosis, prevention of complications, and salvage of the spleen as it was in our case.

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Case Report

Journal of Emergency Medicine Case Reports

Tocilizumab-induced anaphylaxis in two patients with COVID-19-induced cytokine storm

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Abstract

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a zoonotic virus which was first discovered in Wuhan, the People's Republic of China in December 2019 and has led to one of the greatest pandemics of world history in a short period of time[1]. SARS-CoV-2 is a rapidly spreading infectious disease with a high mortality rate. The disease has a moderate and severe course in approximately 20% of the patients and mortality reaches up to 62% among these patients[2]. The majority of the patients develop SARS-CoV-2-induced pneumonia and manifestations of pneumonia rapidly progress to respiratory failure. In severe Covid pneumonia, it has been demonstrated that increased plasma concentrations of cytokines including interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and IL-12 are involved in immune response and in cytokine storm caused by the increase in these cytokines lead to mortality[1, 3]. Tocilizumab (TCZ) is a promising agent that is used for the treatment of cytokine storm. TCZ is an IgG1 class recombinant humanized monoclonal antibody against IL-6 receptor[3]. It has then been used for the treatment of rheumatic diseases. Due to its mechanism of action, TCZ treatment comes to the forefront particularly in cases of severe COVID-19-induced cytokine pneumonia presenting with cytokine storm[3]. However, potential IgE-mediated immunological reactions against this drug, especially anaphylaxis, may deprive these patients of an important treatment option for the treatment of COVID-19-induced cytokine storm. Although TCZ-induced anaphylaxis has been reported as case reports of indicated rheumatic diseases, TCZ-induced anaphylaxis has not yet been reported in patients using TCZ for SARS-CoV-2-induced cytokine storms[4]. In this case series, we aimed to represent cases of anaphylaxis which developed in two different patients using TCZ for SARS-CoV-2-induced cytokine storm.

Key words: anaphylaxis, COVID-19, cytokine storm, drug allergy, Tocilizumab

Introduction

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a zoonotic virus which was first discovered in Wuhan, the People's Republic of China in December 2019 and has led to one of the greatest pandemics of world history in a short period of time[1]. SARS-CoV-2 is a rapidly spreading infectious disease with a high mortality rate. The disease has a moderate and severe course in approximately 20% of the patients and mortality reaches up to 62% among these patients[2]. The majority of the patients develop SARS-CoV-2-induced pneumonia and manifestations of pneumonia rapidly progress to respiratory failure. In severe Covid pneumonia, it has been demonstrated that increased plasma concentrations of cytokines including interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and IL-12 are involved in the inflammatory process and immune response and that the cytokine storm and macrophage activation syndrome caused by the increase in these cytokines lead to mortality[1, 3]. Tocilizumab (TCZ) is a promising agent that is used for the treatment of cytokine storm. TCZ is an IgG1 class recombinant humanized monoclonal antibody against interleukin-6 (IL-6) receptor[3]. It has then been used for the treatment of rheumatic diseases including adult-onset Still's disease and systemic juvenile idiopathic arthritis. Due to its mechanism of action, TCZ treatment comes to the forefront particularly in cases of severe COVID-19-induced cytokine pneumonia presenting with cytokine storm (macrophage activation syndrome)[3]. However, potential IgE-mediated immunological reactions against this drug, especially anaphylaxis, may deprive these patients of an important treatment option for the treatment of COVID-19-induced cytokine storm. Although TCZ-induced anaphylaxis has been reported in the literature as case reports of indicated rheumatic diseases, TCZ-induced anaphylaxis has not yet been reported in patients using TCZ for PCR positive COVID-19-induced cytokine storms[4]. In this case series, we aimed to represent cases of anaphylaxis which developed in two different patients using TCZ for COVID-19-induced cytokine storm.

Case presentation

Patient-1

A 48-year-old male patient admitted to the COVID outpatient clinic with complaints of fatigue, joint pain, and high temperature for 1 week. From his background, it was learned that he had a diagnosis with asthma for approximately 20 years. In laboratory tests of the patient, the SARS-CoV-2 PCR test was

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found to be positive and areas of diffuse ground-glass appearance consistent with COVID pneumonia were detected in his computed tomography (CT) of thorax, after which he was hospitalized (Figure-1). The patient has initiated treatment with hydroxychloroquine (2x200 mg, po, 5 days), favipiravir (loading dose of 2x 800 mg for the first day, 1x800 mg po for consecutive days, a total of 5 days), and anticoagulants (enoxaparin 1x0.6 ml, sc). In addition to the treatment of the patient with asthma, a long-acting beta-2 agonist (Formoterol 12 mcg 2x1, inhalator), inhaled steroids (Budesonide 2x800 mcg, inhalator), ipratropium bromide (4x100 mcg, inhalator) were added. Due to the refractory fever of the patient and detection of an increase in acute phase reactants including C-reactive protein (CRP), ferritin, and fibrinogen, the patient was considered to have COVID-19-induced cytokine storm and macrophage activation syndrome (MAS) and infusion of TCZ at a dose of 8 mg/kg every 12 hours were initiated. Laboratory results of the case are summarized in Table. The patient who described itching has no visible rash about 2 hours after initiation of the infusion. No additional pathologies or changes were observed on cardiac and respiratory system examinations. Pheniramine maleate at a dose of 1x45.5 mg was added to the treatment of the patient who described pruritus. The second infusion of TCZ was determined to be given 24 hours later. On day 2, about 5 minutes after initiation of the second infusion of TCZ, the patient developed shortness of breath, nausea, and a drop in blood pressure, followed by respiratory and cardiac arrest. The infusion was immediately stopped and for anaphylaxis, intramuscular epinephrine at a dose of 1x0.3 mg, IV pheniramine maleate at a dose of 1x45.5 mg, methylprednisolone at a dose of 0.5 mg/kg/day, and ranitidine at a dose of 1x50 mg were administered. The patient became conscious after epinephrine treatment and cardiopulmonary resuscitation and his blood pressure were stabilized without requiring an additional vasopressor agent. As MAS of the patient carried on, initiation of intravenous immunoglobulin (IVIG) treatment was decided. The patient has initiated IVIG treatment at a dose of 0.5 mg/kg/day. No reaction was observed after IVIG treatment. A significant improvement was observed in clinical and laboratory findings of the patient who received IVIG treatment for 5 days.

Patient-2

A 52-year-old female patient admitted to the COVID outpatient clinic with complaints of fatigue, diarrhea, shortness of breath, and high temperature for 3-4 days. In laboratory tests of the patient with no additional disease, the SARS-CoV-2 PCR test was found to be positive and areas of diffuse ground-glass appearance consistent with COVID pneumonia were detected in her computed tomography (CT) of thorax, after which she was hospitalized (Figure-2). The patient has initiated treatment with hydroxychloroquine (2x200 mg, po, 5 days), favipiravir

Table 1. Demographic and laboratory features of the patients

	Patient-1			Patient-2		
Age, years	52			48		
Gender	Male			Female		
Infusion with reaction	2nd infusion			First infusion		
Comorbidities	Asthma			None		
Anaphylaxis-associated symptoms/signs	Urticarial skin rash, pruritus Nausea Dispnea Hypotension			Urticarial skin rash, pruritus Dispnea Hypotension		
	29.05.20	11.06.20	13.06.20	23.07.20	29.07.20	31.07.20
White cell count, $\times 10^9$ /L	10.68	9.90	3.54	6.62	6.76	4.76
Lymphocyte, %	16.4	15.1	13.8	35.3	20.4	18.6
Lymphocyte, mm^3	1.61	1.62	490	1.68	1.26	1.07
CRP, mg/L	10.9	13.14	68.7	77.5	95.9	130
D-dimer, ng/mL	0.4	0.6	4.3	0,4	0.6	1
Ferritin, ng/mL	1225.1	1533.0	598.7	233.0	262.5	372,4
Fibrinogen, g/L	341.5	245.6	405	400	1000	1010
Procalcitonin, ng/mL	0.042	0.078	0.15	0.056	0.536	0.763

CRP: C-reactive protein

(loading dose of 2x 800 mg for the first day, 1x800 mg po for consecutive days, a total of 5 days), and anticoagulants (enoxaparin 1x0.6 ml, sc). Again, in this patient, the patient was considered to have COVID-19-induced cytokine storm and MAS, as during follow-up, she developed refractory fever, an increase in ferritin, CRP, and fibrinogen levels, and a gradual decrease in oxygen saturation (Table). The patient has initiated an infusion of tocilizumab at a dose of 8 mg/kg. Close to the end of the infusion, the patient who developed whole-body redness, diffuse rash, and worsening shortness of breath was considered to have a drug-induced allergy and the infusion was discontinued. The patient whose blood pressure was determined to be 76/43 was given intramuscular epinephrine at a dose of 1x0.3 mg, IV pheniramine maleate at a dose of 1x45.5 mg, methylprednisolone at a dose of 0.5 mg/kg/day, and ranitidine at a dose of 1x50 mg for anaphylaxis, as well as IV dopamine at a dose of 20 mcg/kg/min as an additional vasopressor agent. The patient whose shortness of breath relieved and became normotensive was considered to have TCZ-induced anaphylaxis. No TCZ treatment was planned for the future for the patient.

The patients whose complaints regarding SARS-CoV-2 and who improved in terms of clinical status, laboratory, and radiologically were discharged. Although the patients were referred to the Allergy outpatient clinic for confirmation of TCZ-induced reactions with skin tests, both of the patients refused to undergo skin testing.

Discussion

Anaphylaxis is a life-threatening multisystemic hypersensitivity reaction that suddenly occurs due to mediators released into systemic circulation by mast cells. In adults, drugs are one of the most common causes[5]. In this case series, we represented 2 cases presenting with clinical manifestations of anaphylaxis affecting mucocutaneous, respiratory, gastrointestinal, and cardiovascular systems who used TCZ for treatment of COVID-19-induced cytokine storm and MAS. Tocilizumab is a recombinant humanized anti-IL-6 receptor monoclonal antibody which inhibits IL-6 signal transduction and is usually well tolerated. Although TCZ-induced anaphylaxis has already been reported during treatment of various rheumatic diseases[6, 7], TCZ-induced anaphylaxis has not been reported in patients using TCZ for the treatment of COVID-19-induced cytokine storm and MAS. In regard to this, our cases are the first cases reported in the literature.

As in many other drugs, adverse reactions including TCZ-induced urticaria, erythroderma, cutaneous vasculitis, and anaphylaxis have already been reported in patients using TCZ[8]. Park et al. reported that severe infusion-induced reactions developed in 1.9% of patients with rheumatoid arthritis who were using TCZ[6]. Although TCZ-induced anaphylaxis develops after 2nd-5th infusions in the majority

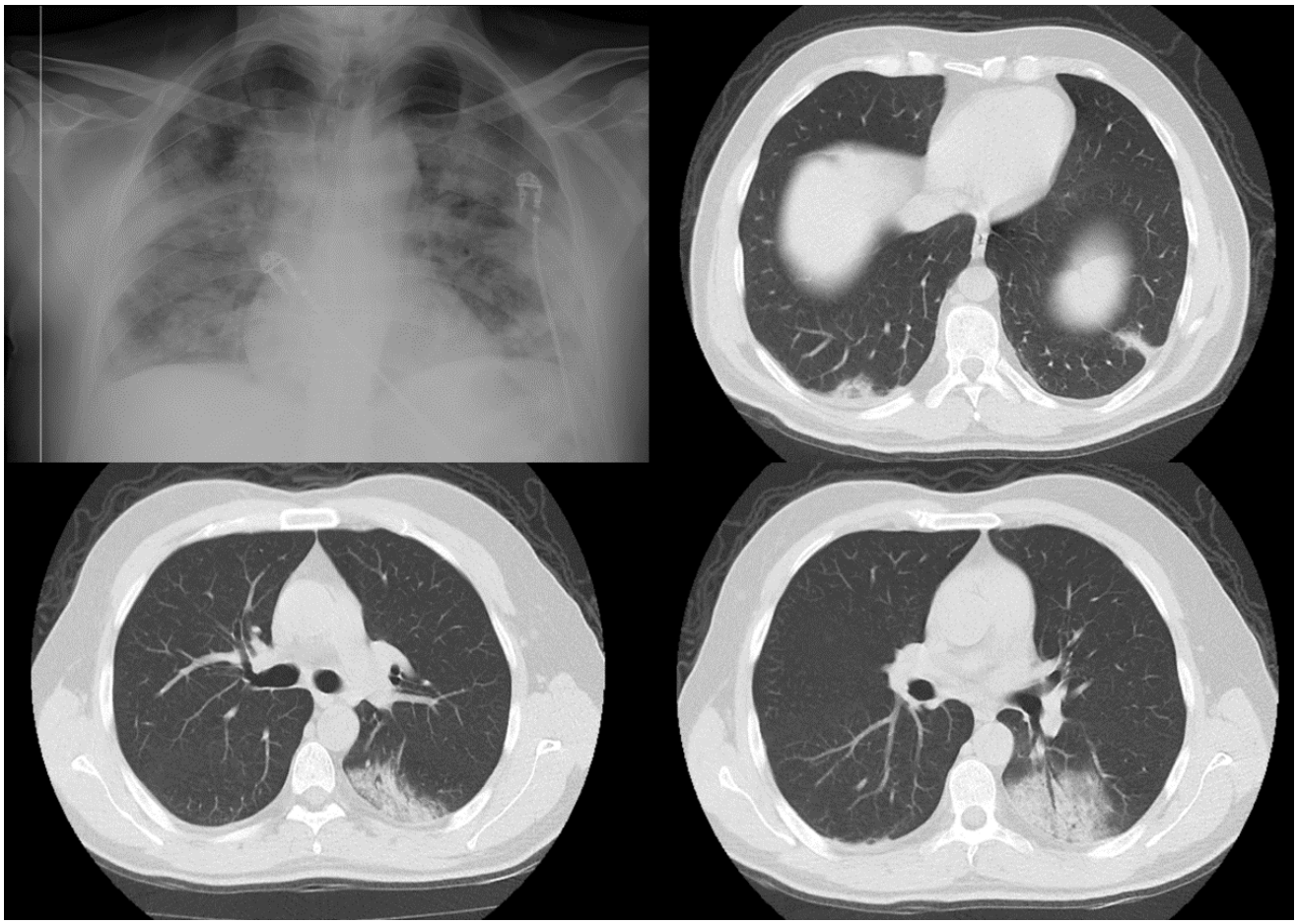

Figure-1: Radiological findings of Patient-1

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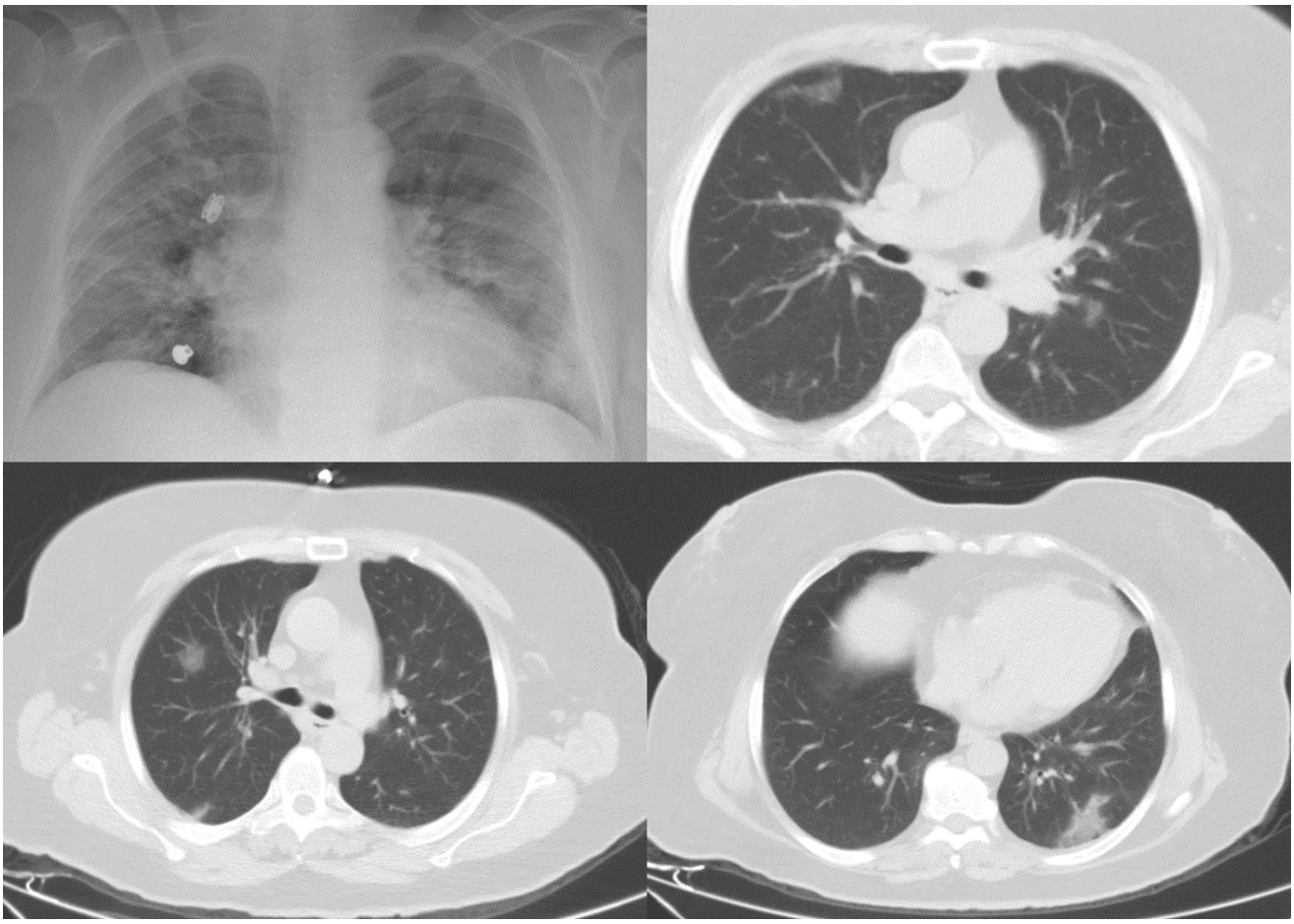

Figure-2: Radiological findings of Patient-2

of the patients, there are cases reporting that anaphylaxis developed during the first infusion of TCZ. While infusion-induced reactions that develop during the first infusion have been primarily associated with cytokine release, infusion-induced reactions that develop during 2nd and further infusions have been associated with type 1 hypersensitivity, complement activation, and anti-drug antibodies[7]. Yasuoka et al. reported younger patients, increased white blood cell count, and acute phase reactants as risk factors for TCZ-induced anaphylaxis[9].

In the case of suspected TCZ-induced anaphylaxis, skin testing such as prick and intradermal tests can be used for direct demonstration of possible drug-specific IgE antibodies. Due to false negativity due to wasting of specific IgEs in blood and intensive degranulation by mast cells, it should be performed 4-6 weeks after the history of anaphylaxis[10]. In our case series, the patients refused to undergo skin testing for the diagnosis of TCZ-induced anaphylaxis. It is a limitation of our study. Treatment of TCZ-induced anaphylaxis in treatment of COVID-19-induced cytokine storm and MAS is not different from treatments of anaphylaxis caused by other reasons and intramuscular administration of adrenaline is the most important and primary treatment option in this patient group as well [10]. Following rapid recognition of anaphylaxis and immediate discontinuation of the drug infusion in both patients, they responded to the intramuscular administration of adrenaline very well and their clinical condition rapidly improved.

In conclusion, TCZ-induced anaphylaxis may develop also in patients using TCZ for the treatment of COVID-19-induced cytokine storm and MAS. More extensive studies are needed in order to reveal risk factors for TCZ-induced anaphylaxis. Clinicians should be alert not only for current comorbidity of COVID-19 infection but also for TCZ-induced anaphylaxis and it should be remembered that epinephrine treatment is the most important and effective treatment option in TCZ-induced anaphylaxis, as in anaphylaxis caused by other reasons.

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Case Report

Journal of Emergency Medicine Case Reports

Extracorporeal Shock-Wave Lithotripsy's Unusual Complication: Retroperitoneal Gas

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Abstract

After extracorporeal shock-wave lithotripsy (ESWL) in renal stone disease, complications such as hematuria, pain, infection, and less often complications such as pancreatitis, myocardial infarction can be observed. We aimed to present retroperitoneal gas findings, an unusual complication post-ESWL detected on Computed Tomography. Lumbar vertebral injury due to ESWL should be kept in mind in the presence of retroperitoneal gas.

Keywords: Emergency radiology, Computed tomography, Nephrolithiasis, Extracorporeal shock wave therapy , Lithotripsy.

Introduction

Extracorporeal shock-wave lithotripsy (ESWL) has been used in the treatment of renal stone disease since the early 80's[(1)]. Its easy application, noninvasiveness and high success rate in renal stone treatment increase the frequency of use in renal and ureteral stone treatment[(2)]. Stone localization and stone burden are important for patient selection in ESWL. ESWL has contraindications such as pregnancy, uncontrolled urinary tract infections, coagulopathy, renal artery and aortic aneurysm, severe obesity and skeletal system abnormalities. Complications such as pain, hematuria, hypertension and less often pancreatitis, pneumonitis may occur in the early period after ESWL[(3,4)]. In this article, it is aimed to present the retroperitoneal gas, an unusual complication of ESWL, detected in the computed tomography (CT) images of the patient who applied to the emergency department with left lower quadrant pain after ESWL.

Case Report

A 70-year-old female patient was admitted to the emergency room with the complaints of increasing nausea and left lower quadrant pain after ESWL performed 1 week ago. In her medical history, the patient reported that the complaint of bloody urine that developed after ESWL regressed, her complaint of dysuria was ongoing, and she had no complaints of low back pain. Vital signs of the patient were stable, and physical examination revealed no findings other than tenderness in the left lower quadrant. In the laboratory examination of the patient, white blood cell count was found to be 12.15 K / uL, creatinine 1.33 mg / dl, CRP 209 mg / L and leukocyte positivity in urine analysis. In the ultrasonographic examination; hydroureteronephrosis, minimal perirenal fluid and stone fragments were seen in the left kidney. In addition to ultrasound findings, linear density increases in perirenal fat tissue and minimal perirenal fluid were observed in unenhanced abdominal CT examination. On unenhanced CT images, gas densities were observed in the retroperitoneal area in the left paraaortic, prevertebral region and medial to the psoas muscle (Figure 1). In order to rule out the differential diagnosis of gastrointestinal system perforation or fistulization, after oral contrast material administration, abdominal CT was repeated and contrast agent extralumination was not observed. Compared to the patient's abdominal CT in 'Picture Archiving and Communication Systems' (PACS) 2 years ago; It was observed that gas densities were newly emerged in this examination, and degenerative vacuum phenomenon was found in the intervertebral disc of the L2-L3 level in the previous examination (Figure 2). Irregularity was observed in the interverterbal disc anterior section of L2-L3 level in the CT after ESWL, and it was found that the vacuum phenomenon observed at this level in the previous examination was not observed in this examination (Figure 3). Gas densities in the retroperitoneal area were evaluated as intervertebral disc / vertebral corpus injury that developed after ESWL. Treatment was initiated for the diagnosis of pyelonephritis developing after ESWL, and it was decided to follow-up retroperitoneal gas densities. We obtained written and oral informed consent of the patient for the publication of her case and any accompanying images.

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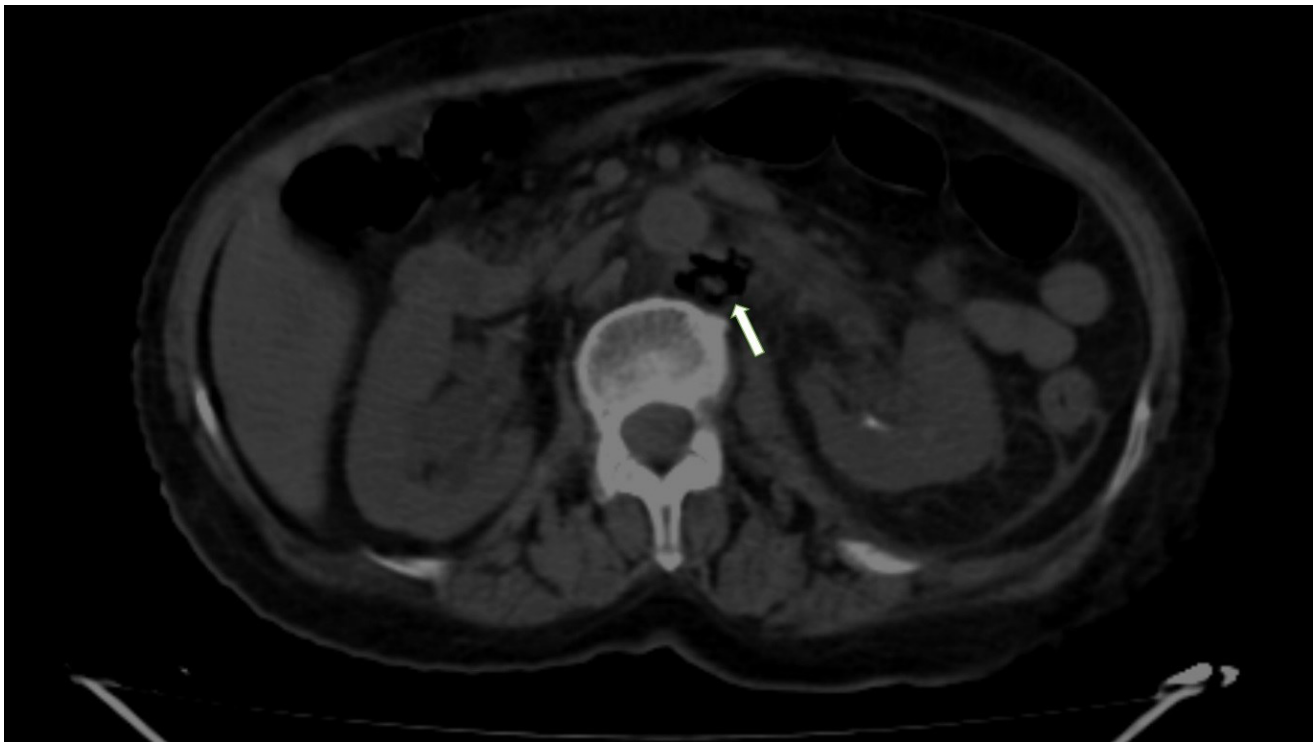

Figure 1.

Discussion

It has been reported that the effectiveness of ESWL treatment and the risk of possible complications in renal stone disease are related to the ESWL shock rate. Accordingly, it has been reported that 60 shock / minute rate in ESWL reduces the risk of kidney damage compared to 120 shocks / minute rate and provides an improvement up to 16% in the treatment of small stones[3,5]. Apart from the frequency and number of shocks given in ESWL, the location and mineral density of the stone are also associated with post-ESWL complications and treatment success. An increase in the risk of developing renal hematoma in obese patients and a decrease in the success rate of the treatment can be expected due to the increase of the distance between the lithotriptor and the stone in the effectiveness of ESWL in obese patients. It has been reported that the distance between the skin and the stone is less than 10-11 cm is an independent predictor of ESWL treatment[6]. Providing an unobstructed path for shock wave during ESWL will reduce the incidence of organ complications in the kidney or ureter neighborhood. Real-time imaging is important to enable shocks to reach the stone, and it has been reported that increased fluoroscopy time increases the effectiveness of stone treatment, despite the risk of radiation[7]. Although there are no similar cases in the English literature, it was reported that retroperitoneal air was detected in 6 patients who underwent epidural anesthesia in the control radiographs obtained after ESWL[8]. There is no interventional procedure in this case before or after ESWL.

Conclusion

As a result, especially in elderly patients with lumbar degeneration, lumbar vertebral injury due to ESWL should be kept in mind in the presence of retroperitoneal gas.

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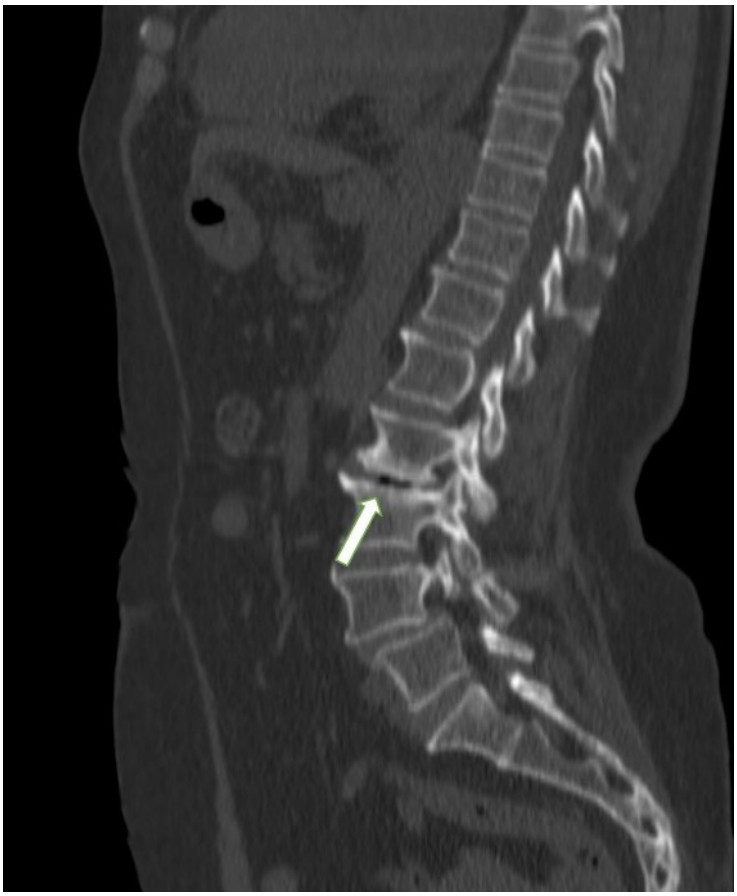

Figure 2.

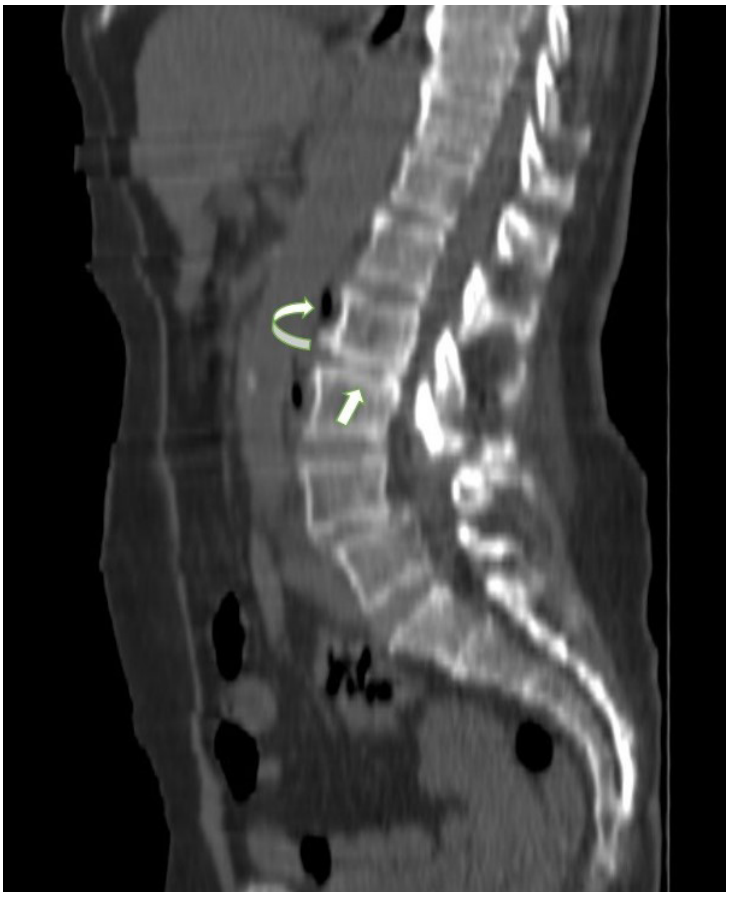

Figure 3.

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Figure Legends

1. Left paraaortic gas density (arrow) in the retroperitoneal area, linear densities in the left perirenal adipose tissue, stone fragment in the left kidney in unenhanced computed tomography image(axial view).
2. In the sagittal reformatted computed tomography image of the case 2 years ago, gas density due to degenerative vacuum phenomenon is observed in the L2-L3 level intervertebral disc.
3. In the new sagittal reformatted computed tomography image of the case, there is no gas density in the L2-L3 level intervertebral disc and gas densities are observed in the prevertertebral area.

Case Report

Journal of Emergency Medicine Case Reports

Delayed Neuropsychiatric Syndrome Following Carbon Monoxide Poisoning: Report of a Rare Case With Response to Treatment

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Abstract

Carbon monoxide (CO) is a colorless, odorless, and non-irritant gas that is lighter than air. Carbon monoxide intoxication is the leading factor of deaths due to toxications. Besides, it is an important health problem due to its morbidities related to the delayed neurological and/or psychiatric syndromes that occur after the acute recovery period. Besides behavioral problems, mood and personality changes, parkinsonism, motor deficits, and dementia syndrome are the most common clinical pictures. Compared with hyperbaric oxygen, which has been proven effective in the acute period in CO intoxication, there is no treatment with definitely proven efficacy for the delayed neuropsychiatric picture. In this paper, a case of CO intoxication followed with a delayed neuropsychiatric syndrome is presented in the light of the literature.

Keywords: carbon monoxide, poisoning, delayed neuropsychiatric syndrome, methylprednisolone, memantine

Introduction

Carbon monoxide (CO) is a colorless, odorless, lighter than air and non-irritating gas. Carbon monoxide poisoning usually presents with a severe clinical course and may result in severe neurological sequelae and even death[1]. Rarely, neurological and/or psychiatric syndromes may be encountered following the recovery period after acute CO poisoning. The delayed neurological or neuropsychiatric syndrome usually emerges within one to four weeks following acute poisoning. The typical radiological feature of this syndrome is mostly symmetrical, demyelinating lesions that are localized to the subcortical white matter[2]. The prevalence of the neuroimaging lesions is closely related to the severity of the clinical picture and the poor prognosis[3].

In this report, a case in whom a delayed neuropsychiatric syndrome had developed following CO poisoning is presented to be discussed in terms of its pathophysiological and clinical features and treatment.

Case Presentation

A 69-year-old female patient was admitted to the emergency department of our hospital with a complaint of nervousness, weakness, and fatigue in the last two weeks. The weakness in both arms and legs had worsened for the last week. She could not walk without support and perform self-care. She was discharged from another hospital with no sequelae after hyperbaric oxygen therapy, where she applied for CO poisoning about six weeks ago. During the neurological examination of the patient, she was conscious, albeit partially disoriented for place and time. We found mild dysarthria and quadriparesis. The standardized mini-mental test score was 20/30. The brain tomography was normal. Cranial magnetic resonance imaging revealed diffuse demyelinating lesions without contrast enhancement. The lesions which were located at the periventricular white matter and centrum semiovale were mildly hypointense in the T1W sequence and hyperintense in T2W and FLAIR sequences (Figure 1). The widespread slow-wave activity was observed in the electroencephalogram (Figure 2). The rest of the laboratory and imaging tests, performed for differential diagnosis were normal. The patient, who was suspected to have late period demyelinating central nervous system involvement, was hospitalized in our clinic. We have decided to manage the clinical picture with intravenous methylprednisolone 1 gram daily for 10 days, memantine 20 mg daily, and physiotherapy. Behavioral and cognitive problems started to improve immediately under these treatments. The patient was discharged in the 4th week of follow-up when she could be independently mobilized and perform daily routine activities.

Discussion

Carbon monoxide is an odorless, colorless, non-irritating gas formed as a result of hydrocarbon combustion. Carbon

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monoxide poisoning is the leading cause of death due to toxins[1]. It binds to hemoglobin with a higher affinity than oxygen and converts to carboxyhemoglobin (COHb), disrupting oxygen transport. Carbon monoxide can cause derangement in many systems but particularly affects the central nervous and cardiovascular systems because of their highest consumption rate of oxygen. In severe CO poisoning, cardiovascular and metabolic complications such as seizures, coma, myocardial ischemia, arrhythmia, pulmonary edema, lactic acidosis, and irreversible neurological deficits can be seen[2]. Acute myocardial ischemia is the most important clinical presentation determining long-term mortality[3].

The mechanism of late pathological changes due to CO poisoning is not clear. White matter lesions may arise based on cerebral vascular damage, cerebral parenchymal cytotoxic edema, and hypersensitivity reaction as a result of the combined effects of acidosis, hypoxia, and hypotension. Direct toxic effects as a result of various intracellular proteins that bind CO is also a contemplated mechanism. It is among the theories that lipid peroxidation caused by toxic oxygen molecules may be another cause[4]. Besides, it was proposed that exposure to hyperoxia as a result of reperfusion during the recovery period after acute CO poisoning may increase the existing oxidative damage[2].

Although the first one to four weeks after acute CO poisoning is the most common time for the development of a delayed neuropsychiatric syndrome, cases may be seen ranging from 3 to 240 days in the literature[5]. Morbidity depends on early neurocognitive impairment a great deal. Delayed neuropsychiatric syndrome and cognitive sequelae, which reach a serious extent, are extremely rare, and it has been reported that sequelae findings may persist for 1 year or longer (6). Our patient had applied to the neuropsychiatry clinic approximately four weeks after the end of the hyperbaric oxygen treatment, and no other etiology that would cause widespread white matter demyelination was detected.

The incidence of the delayed neuropsychiatric syndrome was reported to be 2.75% in a case-serial study including 2360 cases[7]. However, it has been emphasized that the frequency may reach 10% in recent studies[8]. Carbon monoxide poisoning causes nonspecific symptoms such as headache, visual disturbances, dizziness, nausea, and vomiting in the acute period. Mental status changes ranging from mild confusion to coma often accompanies. Cognitive disorders, dementia, mood abnormalities, and personality changes can be seen in the late period following CO poisoning. In many cases in the literature, the primary neurological symptom had been parkinsonism in addition to cognitive loss[9]. In our case, it was known that during the acute CO poisoning period, there were no neurological or cardiac symptoms other than headache and confusion. However in the late period, there were quadriparesis, dysarthria, and agitation in addition to cognitive impairment.

Hyperbaric oxygen administration is the accepted and applied treatment in the acute phase of CO poisoning. However, there is still no commonly applied and accepted method in the treatment of delayed neuropsychiatric syndromes. Re-application of hyperbaric oxygen administration was evaluated in a meta-analysis study, but its effect could not be demonstrated[10]. Therefore, corticosteroid applications have come to the fore intending to reduce inflammatory and oxidative stress. This approach has resulted in a favorable response in a small portion of the patients. In a few patients, steroids have been used successfully in combination with

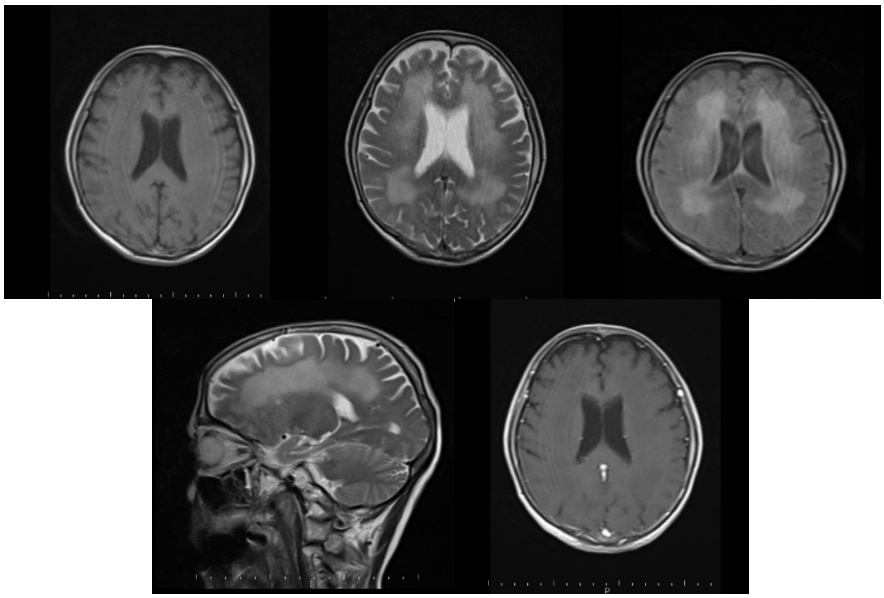

Figure 1. Diffuse demyelinating lesions without contrast enhancement at periventricular white matter and centrum semiovale mildly hypointense in the T1W sequence and hyperintense in T2W and FLAIR sequences.

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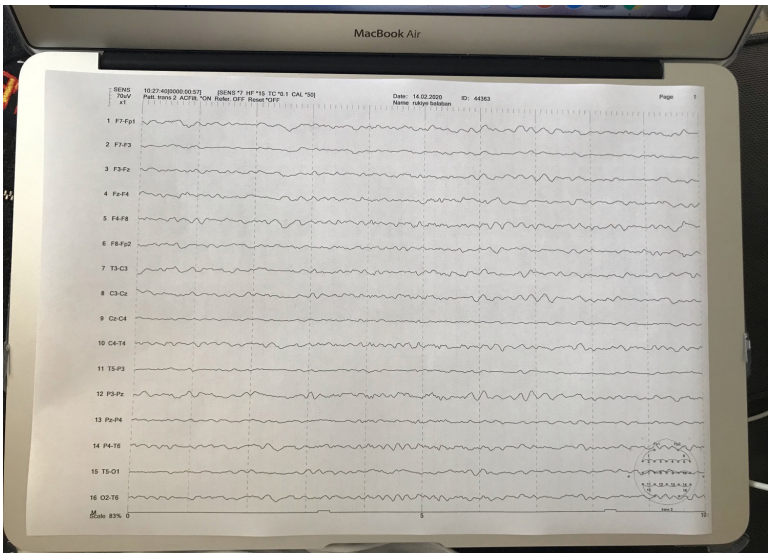

Figure 2. The widespread slow-wave activity in the electroencephalogram.

memantine as in our case. Parkinsonism is a common motor deficit and hence, a levodopa trial is proposed. However, the results of levodopa are frustrating[11].

Conclusion

Acute CO intoxication is known to be life-threatening, with rare but serious complications. Consequently, CO poisoning should be considered and questioned by emergency department personnel in patients who admit signs of the delayed neuropsychiatric syndrome. This approach avoids the risk of morbidity and death in such cases.

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Case Report

Journal of Emergency Medicine Case Reports

"Frog-Legged Trauma Patient". Bilateral Anterior Hip Dislocation

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Abstract

Anterior hip dislocation is rare (10-15% occurrence) compared with posterior dislocation. Bilateral simultaneous anterior hip dislocation is even extremely rare. Most of the previously reported case were either a unilateral or combination of anterior and posterior dislocation. We present a case of bilateral anterior hip dislocation post motor vehicle accident. Patient brought in the Emergency Department in "frog-legged" posture, unable to straighten the lower limbs. Even the Pre-Hospital Care (PHC) team had a tough time to load the patient onto the ambulance stretcher. On examination, both lower limbs were held in abduction, external rotation and in flexion position. Neurovascular assessments were intact. Pelvic x-ray shows bilateral anterior hip dislocation, right iliac bone fracture and left greater trochanteric femur fracture. Closed manual reduction of both hip were performed under procedural sedation analgesia. CECT pelvic done later confirmed right iliac bone fracture extending to and comminuted fracture of the left greater trochanteric. Bilateral avascular necrosis of both femoral head was also noted. Traumatic hip dislocations are a true orthopedic emergency. Early reduction (within 6 hours) is required as soon as appropriate x-rays have been obtained to exclude associated injuries since avascular necrosis of the femoral head increases in direct proportion to delay in reduction. If this fails, closed reduction under general anesthesia is indicated.

Keywords: bilateral, anterior, hip dislocation

Introduction

Traumatic dislocations of the hip are common nowadays with the rising incidence of motor vehicle accident (MVA) involving motorcycles (Akinyoola & Abiodun 2005). Anterior hip dislocation is rare (10-15% occurrence) compared with posterior dislocation. Bilateral simultaneous anterior hip dislocation is even extremely rare. Previously reported cases were either a unilateral or combination of anterior and posterior dislocation. We present a case of bilateral anterior hip dislocation following MVA.

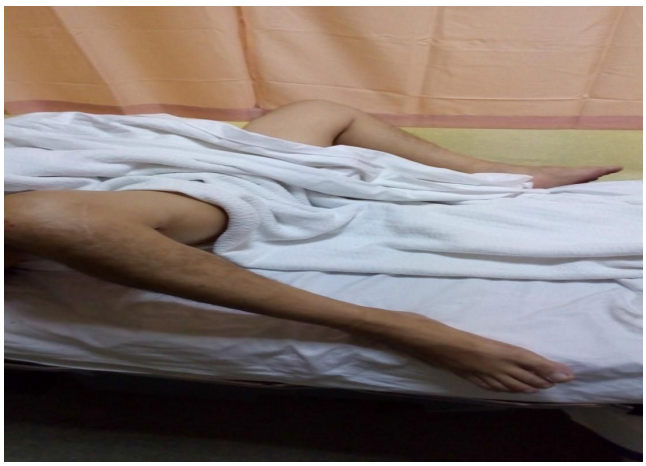

Figure 1: Patient presented with bilateral hip in abduction, external rotation and in flexion position.

Case Presentation

A 45-year-old man was involved in MVA; head on collision between motorcycle versus car from opposite direction. Patient brought in the Emergency Department after 2hours post MVA in "frog-legged" posture, unable to straighten the lower limbs (**Figure 1**). Even the Pre-Hospital Care (PHC) team had a tough time to load the patient onto the ambulance stretcher. PHC team reported no sign of head injury or any external bleeding at the scene. For a brief moment we are captivated by the odd posture; nevertheless, we proceeded with trauma resuscitation. Primary survey was performed and life-threatening conditions were ruled out. Patient had full GCS, stable vital signs and only complaint of pain over both hips (pain score 8/10). Examination of the bilateral hip: both lower limbs were held in abduction, external rotation and in flexion position. Neurovascular assessments were intact. Pelvic x-ray shows bilateral anterior hip dislocation, right iliac bone fracture and left greater trochanteric femur fracture (**Figure 2**). After 5 hours post MVA closed manual reduction (CMR) of both hip were performed under procedural sedation analgesia using Allis' manoeuvre with the addition of lateral traction to the proximal thigh as the femoral head is displaced medially and the patient

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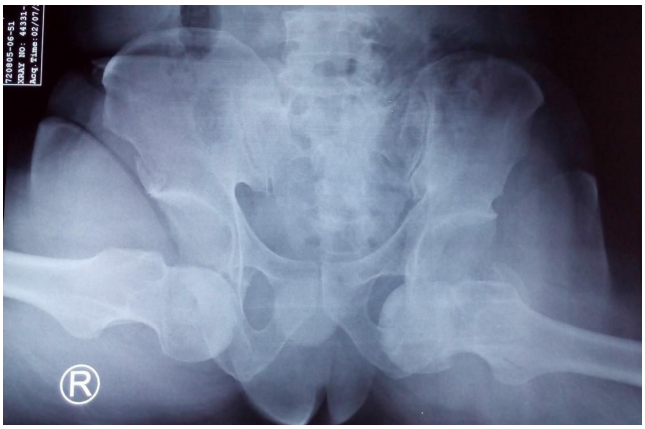

Figure 2: AP pelvic x-ray shows bilateral inferior anterior hip dislocation (Type II or Obturator type).

placed in bilateral Hamilton-Russell traction. Patient admitted to orthopedic ward after post reduction pelvic x-ray shows reduction of both hips (**Figure 3**). Patient have been advised for wheelchair immobilization for 6 weeks. A CECT pelvic done later confirmed right iliac bone fracture extending to acetabular involving the roof, posterior wall and comminuted fracture of the left greater trochanteric. Bilateral avascular necrosis (AVN) of both femoral head was also noted. Patient have been follow up after 4weeks post trauma, and patient claim that he able to ambulate with minimal pain over the both hips.

Discussion

Anterior hip dislocation is considered rare due to the anterior capsule of the hip is stronger and is further reinforced by iliofemoral ligament (Akinyoola & Abiodun 2005). The hip is a spheroidal type of joint with a good congruence between the femoral head and the acetabulum and fortify by a thick articular capsule and tough ligaments (Radulescu R. et al. 2013). All these features make the hip joint very stable. Mechanism of injury must involve extreme abduction causing the femoral head to be pushed out through a tear in the anterior capsule as commonly seen in MVA and a blow to the back while squatting. There are several case reports of anterior hip dislocation involving contact sports as well e.g. rugby, American football and soccer.

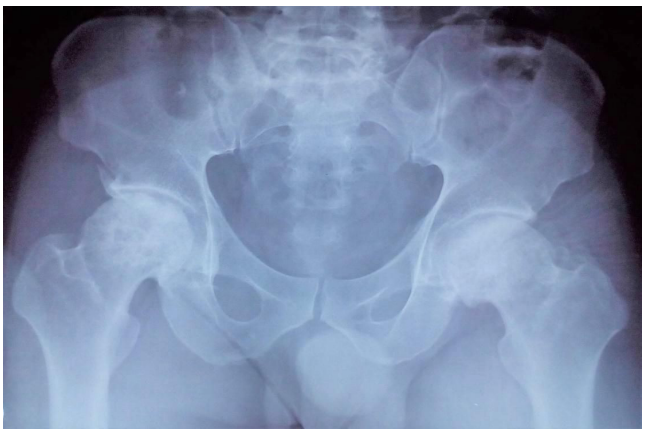

Figure 3: Post reduction pelvic x-ray shows concentric and congruent reduction of both hips.

There are 2 subtypes of anterior hip dislocation namely anterior superior dislocations (Type I or pubic type) whereby the limb is slightly abducted, externally rotated and extended. Another subtype namely anterior inferior dislocations (Type II or obturator type), the limb is abducted, externally rotated and flexed. According to Horner S. et all (2012), inferior dislocation is the most common type of anterior dislocation, comprising more than 92% of anterior dislocations. Our patient sustained Type II anterior hip dislocation. The anterior dislocations are further described by the Epstein classification:

Type I - Superior dislocations
- IA: no associated fractures
- IB: associated fracture or impaction of the femoral head
- IC: associated fracture of the acetabulum

Type II - Inferior dislocations
- IIA: no associated fractures
- IIB: associated fracture or impaction of the femoral head
- IIC: associated fracture of the acetabulum

In our case, the reduction was done within 5 hours after the injury as waiting for the orthopaedic team decision to reduce both hip as it was associated with right iliac bone fracture and left greater trochanteric fracture. In this patient, the risk to develop AVN increases as patient have concomitant pelvic fractures (Sraj & Lakkis 2007). The cause of AVN is thought to be multifactorial; firstly, during dislocation, the vascular network emerging from the trochanteric area is injured together with the joint capsule and the round ligament artery together with the ligament. Other reasons demonstrated a functional disruption of cephalic circulation by a spasm of the large artery or of the cervical branches, with no organic lesion itself. If we take into discussion this mechanism, the early reduction of the dislocated hip able to reduce the risk of AVN by 10-40% (Radulescu R. et al. 2013). Early complications of anterior dislocations involved femoral artery, vein and nerve injury. Late complications (in addition to osteoarthritis and AVN of the femoral head) includes pulmonary embolism as a result from femoral artery/vein thrombosis. It has been recommended that follow up x-ray study at 3–6-month intervals for at least 2 years need to be done to evaluate for AVN (Honner & Taylor 2012).

Conclusion

Traumatic hip dislocations are a true orthopedic emergency. Early reduction (within 6 hours) is required as soon as appropriate x-rays have been obtained to exclude associated injuries since AVN of the femoral head increases in direct proportion

to delay in reduction. The diagnosis should be suspected based on the mechanism of injury and characteristic clinical presentation. The risk of AVN, which is seen in 0-5% if the hip is reduced in less than 6 hours after the injury compared in 50% if the hip is reduced more than 6 hours after the injury (Sultan et al. 2012). If this fails, CMR under general anesthesia is indicated. Neurovascular assessment should be performed and documented prior and post any reduction attempts.

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Case Report

Journal of Emergency Medicine Case Reports

COVID-19 at First Glance, Pulmonary Tuberculosis With a Glance in Depth

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Abstract

Introduction: Here we report a case who presented with fever, dry cough, sore throat and myalgia, mimicking COVID-19 and diagnosed as Pulmonary Tuberculosis. Case Report: A 31-year-old female patient was presented to the COVID-19 outpatient clinic with 7-day history of fever,dry cough,night sweats,chills,sore throat,and myalgia. Laboratory results revealed lymphopenia, C-reactive protein, erythrocyte sedimentation rate and D-dimer elevation compitable with COVID-19. She was hospitalized with diagnosis of "probable COVID-19". SARS-CoV-2 multiplex PCR were resulted negative. Chest CT revealed cavitary lesion located in the upper lobe of the right lung with uncertain borders, consolidation in the right upper lobe primarily in favor of TB infection. She had three samples of sputum acid-fast bacillus smear that came back positive. TB PCR also resulted positive. Conclusion: Although the most common presentation of COVID-19 seems to be pneumonia, there is no unique clinical feature that reliably differentiates COVID-19 from other upper / lower airway viral or bacterial infections.

Keywords: Pulmonary Tuberculosis, COVID-19, SARS-CoV-2

Introduction

"Coronavirus Disease 2019" (COVID-19), caused by the virus, originally called 2019-nCoV, later named "SARS-CoV-2" was declared as a pandemic by the World Health Organization (WHO) in March 2020[1]. Although the most common presentation of infection seems to be pneumonia, there is no unique clinical feature that reliably differentiates COVID-19 from other upper / lower airway viral infections. The most commonly documented reason for hospitalization is new onset cough and respiratory distress which are also main symptoms of pulmonary *Tuberculosis* (TB)[2].

In 2018, an estimated 10 million people fell ill with TB worldwide[3]. In Turkey the incidence of TB decreased from 29.4 per hundred thousand in 2005 to 14.1 per hundred thousand in 2018[4]. However TB should always be in the differential diagnosis of patients presenting with fever, cough, night sweats, and fatigue.

In the midst of pandemic, COVID-19 ranks the first in differential diagnosis of all patients with fever and respiratory symptoms. Physicians awareness is the cornerstone in differential diagnosis of different diseases presenting with similar symptoms.

Here we report a case who presented with fever, dry cough, sore throat and myalgia, mimicking COVID-19 and diagnosed as TB.

Case Report

A 31-year-old female patient presented to the COVID-19 outpatient clinic with 7-day history of fever, dry cough, night sweats, chills, sore throat, and myalgia. She had no travel history or no contact with anyone who had traveled anywhere or had COVID-19. Her body temperature was 38,5°C. The rest of physical examination was unremarkable. Chest radiography showed prominent bronchovascular branches and increased localized reticulonodular density in the right upper lobe **(Figure 1).**

Laboratory results were as follows: hemoglobin 10,7 gr/dL (11.7-15.5), white blood count 6.6 x10^3/µL (4.1-11.2 x10^3/µL) with 0.92 x10^3/µL (1.2-3.6 x10^3/µL) lymphocytes count. C-reactive protein (CRP) was 7.59 mg/dl (normal; <0.1), erythrocyte sedimentation rate (ESR) was 80 mm/h (0–25 mm/h). D-dimer was 1.81 mg/L (0-0.55) and ferritin was 47.5 µg/L (11-307).

She was hospitalized with diagnosis of "probable COVID-19 or TB" and isolated in a single room, with standard, contact and droplet precautions. Nasopharyngeal swab specimens were obtained for respiratory viral, bacterial and SARS-CoV-2 multiplex Real-Time Polymerase Chain Reaction (RT-PCR) test that all were resulted negative the next day. In the detailed history of the patient after she was hospitalized, it was learned that she has been coughing for 3 weeks and she unintentionally lost 2 kilograms during the same period. She had no sputum or hemoptysis. She noticed an increase in night sweats. Low dose chest computed to-

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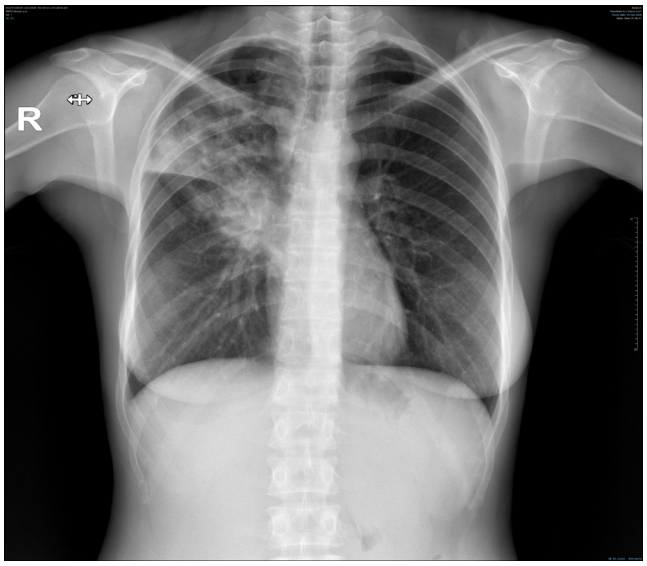

Figure 1: Postero-Anterior Chest Graph: Prominent bronchovascular branches and increased localized reticulonodular density in the right upper lobe

mography (CT) revealed cavitary lesion located in the upper lobe of the right lung with uncertain borders, consolidation in the right upper lobe primarily in favor of TB infection **(Figure 2).**

A bronchoalveolar lavage (BAL) sample was taken from the right upper lobe by fiberoptic bronchoscopy (FOB). She had three samples of sputum acid-fast bacillus (AFB) smear that came back positive. She had a skin purified protein derivative (PPD) test which showed active induration of 15 mm. TB PCR also resulted positive. The patient started on four-drug regimen; isoniaside, rifampin, ethambutol and pyrazinamide.

On the 7th day of treatment, her fever resolved. The general condition of the patient improved. Laboratory values started to improve. The patient was discharged by a follow-up plan.

Discussion

The COVID-19 pandemic is an awe-inspiring situation. Although data regarding clinical presentations related to this new virus and the list of common symptoms is rapidly growing, fever and respiratory symptoms hold theirs' own.

TB and COVID-19 baseline clinics are similar. They are both transmitted mainly by airdrops with close contacts. They have similar symptoms such as fever, cough which could lead to delays in the timely diagnosis in the midst of pandemic. In fact TB is a chronic disease with cough of 2 weeks and COVID-19 has a rapid onset[5], but it can be challenging to take a detailed history of symptoms with masks, glasses and PPE as it happened in our case.

COVID-19 and TB also have similar laboratory findings. Lymphopenia accompanied by mild thrombocytopenia is among the most common abnormal findings, attracting the attention of COVID-19 patients. The total white blood cell count or neutrophil counts are found to vary[1]. While lymphopenia and thrombocytopenia are rare in the course of TB, mostly normocytic anemia, leukocytosis, thrombocytosis, and elevated liver enzymes are seen as a result of increased cytokine activity[6]. Although anemia may be seen in COVID-19 patients at admission, it has been reported that this may be associated with chronic disease anemia due to underlying comorbidities rather than the pathophysiology of COVID-19[7]. ESR has been shown to be a prognostic factor in both COVID-19 and TB[7,8]. As expected, in inflammatory processes CRP and ferritin also increase during both COVID-19 and TB. While the procalcitonin (PCT) level is usually within the normal range at the initial diagnosis of COVID-19, it has been shown to increase in those with bacterial co-infection and intensive care patients[7]. PCT is also not generally elevated in pulmonary TB patients however when serum PCT is not within the normal range, it is a poor prognostic marker[9]. Increased levels of lactate dehydrogenase (LDH), alanine aminotransferase (ALT), aspartate ami-

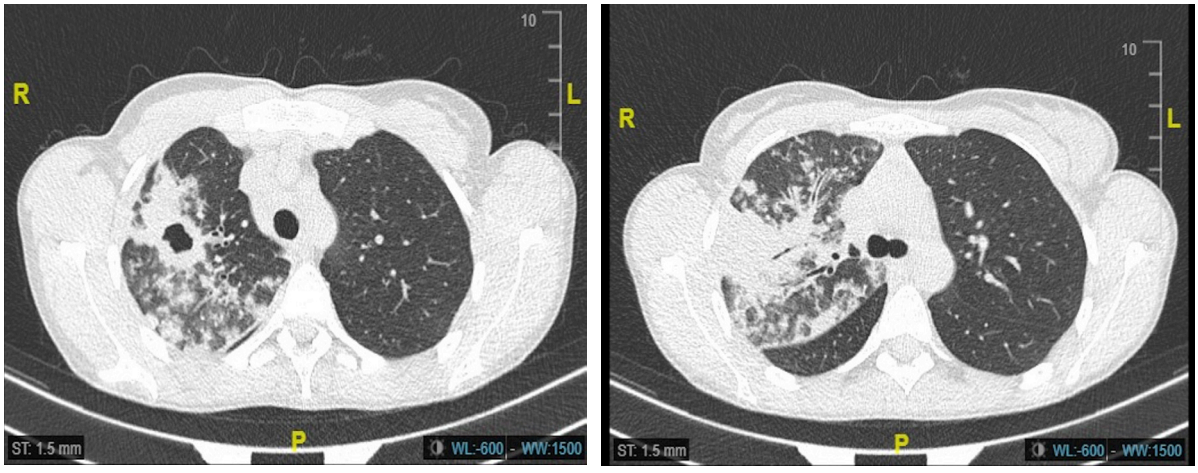

Figure 2: Chest CT: Cavitary lesion in the right lung with uncertain borders of the upper lobe, consolidation in the upper lobe anterior segment and distal-inferior cavitary lesion, patchy ground glasses in the middle lobe, patchy opacities that tend to merge in the upper lobe superior segment and adjacent major fissure area

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notransferase (AST) and decreased levels of albumin are among the most common abnormal biochemical laboratory findings in COVID-19 patients, and elevated D-dimer support the occurrence of coagulopathy as an important indicator of disease progression[7]. In our case, all these similar findings first suggested the diagnosis of COVID-19. However, TB should still be first in the differential diagnosis in patients presenting with anemia, elevated ESR, and CRP.

Pulmonary TB is caused by the bacterium *Mycobacterium tuberculosis* complex. The bacterium is easily spread from an infected person to someone else. Bacteria can be contracted by breathing in air droplets from a cough or sneeze of an infected person. The resulting lung infection is called primary TB. Most people recover from primary TB infection without further evidence of the disease. The infection may stay inactive (dormant) for years. In some people, it reactivates. Symptoms of pulmonary TB are cough, difficulty in breathing, excessive sweating (particularly at night), fever, fatigue, weight loss[10]. According to guidelines, the diagnosis of pulmonary TB should include smear and culture of sputum samples for ARB, as well as nucleic acid amplification testing[11].

Diagnosis of COVID-19 is challenging for a couple of reasons: Firstly preferred diagnostic test for COVID-19 is a RT-PCR test that detects SARS-CoV-2 from the sample taken from the upper respiratory tract[12]. But RT-PCR sensitivity reported to be 63% for nasal swabs[13]. Secondly; diagnostic value of chest X-ray in COVID-19 pneumonia ranges from 30-60%. CT plays an important role in the early stages of infection but it can not be easily available in all settings.

COVID-19 and TB are both infectious diseases that primarly involve the respiratory tract. They have very similar symptoms and laboratory findings which could lead to delays in the timely diagnosis in the midst of pandemic. COVID-19 is grabbing the headlines, but other diseases - including TB - haven't stopped yet. Physicians' awareness is the cornerstone of the precise diagnosis of diseases with similar diagnostic tests, presenting with similar symptoms.

*Written consent has taken from our patient.

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Case Report

Journal of Emergency Medicine Case Reports

Middle Cerebral Artery Infarction Associated with Sildenafil Citrate (Viagra) Use

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Abstract

İntroduction: Sildenafil is a phosphodiesterase 5 enzyme inhibitor used in the treatment of erectile dysfunction and pulmonary hypertension. The use of sildenafil with nitrates is well known to cause myocardial ischemia. However, patients with a history of hypotension, arrhythmia, previous myocardial infarction, or stroke should be given sildenafil carefully. A few cases have been reported in the literature related to ischemic stroke and transient ischemic attack due to sildenafil use.

Case report: We report a case of middle cerebral artery (MCA) infarction after two tablets sildenafil in a 57-year-old male patient with no underlying disease. In the diffusion MRI (magnetic resonance imaging) imaging of the patient, acute diffusion limitation was observed in the left frontal lobe and parietal lobe starting from the centrum semiovale level and also in the MCA supply area affecting the medial section of the temporal lobe. We believe that this infarction is caused by the development of severe hypotension due to alcohol use with sildenafil.

Conclusion: Not only availability of hypotension, history of MI and stroke, health care providers should also give attention prescribing sildenafil in the case of cigarette and alcohol use.

Key words: Sildenafil, ischemic stroke, hypotension

Introduction

Sildenafil citrate (Viagra; US Pharmaceutical Group, New York, NY, USA) is the drug of choice for most men with erectile dysfunction. The use of sildenafil with nitrates is well known to cause myocardial ischemia. However, patients with a history of hypotension, arrhythmia, previousmyocardial infarction, or stroke should be given sildenafil carefully. In both placebo-controlled and open-label studies, sildenafil was not associated with an increased risk of stroke[1]. These studies, however, excluded patients who had had myocardial infarction, lifethreatening

arrhythmia, or stroke in the previous 6 months—a population at very high risk for subsequent stroke. In a patient with no risk factor for a cerebrovascular event, we present MCA infarction after sildenafil and alcohol use.

Case Report

A 57-year-old, right-handed male patient came to the emergency room with a speech disorder, dropping of mouth, and inability to recognize his relatives for 2 days. He had no history of hypertension, diabetes, hyperlipidemia, heart disease or stroke. The patient had smoked 15 packs / year and used alcohol rarely.

The patient received two tablets sildenafil with alcohol 2 days ago. The relatives of the patient who think of alcohol as a cause of ill-consciousness did not bring the patient to the hospital on the first day. Since the patient did not improve his consciousness, his relatives brought him to the emergency room. In the physical examination, the patient was lethargic, did not have orientation and cooperation, or nuchal stiffness, his pupils were anisochoric, and myosis on his left eye was present. He had motor and sensory aphasia. The patient had hemiplegia on the right side.

His cranial tomography showed significant loss of density at the level of the left temporoparietal lobe.

MRI of the brain with diffusion-weighted imaging performed in the left frontoparietotemporal region, diffusion

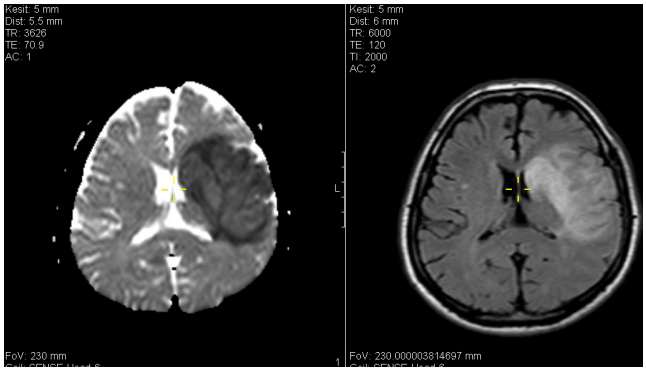

Figure 1: Cranial MRI image

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limitation was observed in the MCA supply area, consistent with acute infarction. In the evaluation of FLAIR images, the increase in signal area was noted (Fig. 1).

CT angiography ther was a plaque in the left Common Carotid Artery distal segment causing 50% stenosis. The left Internal Carotid Artery (ICA) was occluded, plaque was present at the right ICA proximal leading to critical stenosis. The left ICA shows retrograde filling and no contrast enhancement was observed at the MCA level. The patient was not given a tPA (Tissue Plasminogen Activator) and thrombectomy could not be performed because the patient admitted 24 hours later.

TPA and thrombectomy could not be performed because the patient admitted 24 hours later. Clexane 2* 60 mg, aspirin, plavix treatments were started.

His thyroid functions, creatine kinase (CK) and CK-myocardial band (MB) levels, Protein C and S were in normal range. Folate (5,5µ/L), B12 (152 µ/L) . Total cholesterol: 201 mg / dL Triglyceride: 321 mg / dL HDL: 33 mg / dL LDL: 104 mg / dL.

EKG showed normal sinus rhythm and a transthoracic echocardiogram did not reveal thrombus, vegetations, or other source of embolus, EF was measured as 55-60%. Valve pathology was not detected. After 6 days, brain magnetic resonance imaging revealed acute diffusion restriction on the left frontal lobe and parietal lobe starting from the centrum semiovale level, as well as the MCA supply area affecting the medial segment of the temporal lobe. Approximately 15 days of motor weakness and motor aphasia persisted and there was no improvement in right hemiplegia. The blood pressure was between 120/80 and 140/90 mmHg to date.

Discussion

Sildenafil is a phosphodiesterase inhibitor used in the treatment of sexual dysfunction and pulmonary hypertension. The mechanism of action is the inhibition of cyclic guanosine monophosphate (cGMP) -specific phosphodiesterase type 5 (PDE5). Increased cGMP increases the effect of nitric oxide (NO)[2]. Since there are relatively high levels of PDE5 in the human corpus cavernosum and in vascular, visceral and tracheal smooth muscles, NO leads to vasodilatation not only in the corpus cavernosum but also in systemic vessels. Systolic blood pressure decreases by 8-10 mmHg in patients due to systemic vasodilatation[3-4]. Sildenafil causes hyperemic nasal congestion, redness, headache and reduces pulmonary blood flow. Ischemic stroke due to sildenafil use is reported only a few times in the literature[5]. The etiology of vascular ischemia after sildenafil use is still uncertain. Several reports assume that hypotension, cardioembolism, or hypercoagulation is responsible. Although sildenafil has no direct effects on platelet function, it modestly potentates the inhibitory effect of the NO donor sodium nitroprusside on ADP-induced platelet aggregation ex vivo. According to this mechanism of action, adverse bleeding episodes are the major concern so, it is unlikely that sildenafil induces a hypercoagulable state[6-8]. When sildenafil is used with alcohol, the vasodilator effect increases and the tension decreases more. In our patient with symptomatic cerebrovascular disease, we think that the use of sildenafil together with alcohol can significantly reduce blood pressure and cause stroke. Another possible mechanism may be related to an increase in sympathetic activity.

Sildenafil-induced arterial vasodilatation has been shown to produce an increased pressure gradient on the left ventricular outflow tract, and pre-existing cardiomyopathy may be prone to produce atrial fibrillation[8]. It is believed that cardiovascular complications may occur with or before sexual activity[9-10].

Because sexual activity, sildenafil, drug and alcohol interactions or underlying cardiovascular disease are all miscible, it will be more difficult to determine the exact cause of the mechanism of action. It is known that sildenafil should be administered with caution to patients with hypotension, previous myocardial infarction, stroke or arrhythmia. Our experience with unilateral MCA region infarction due to sildenafil use suggests that sildenafil should be prescribed with caution in patients without risk factors.

Conclusion

Not only availability of hypotension, history of MI and stroke, health care providers should also give attention prescribing sildenafil in the case of cigarette and alcohol use

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Case Report

Journal of Emergency Medicine Case Reports

Nailbed Debridement of the Finger Degloving Injury: Two Case Reports

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Abstract

Degloving injury is a serious trauma of the fingers. It is difficult to establish an adequate extent of debridement at the time of initial surgery. Therefore, additional surgery for necrotic areas related to circulatory failure is required in many cases. In this study, we performed replantation for degloving injury of 3 fingers in 2 patients, and obtained findings regarding nailbed debridement on initial surgery. Considering nailbed hemodynamics, when performing replantation for degloving injury, in which the nailbed remains, being adhered to the distal phalanx, but not to an amputated finger, debridement of the nailbed and distal phalanx region to which the nailbed is adhered to on initial surgery may facilitate the avoidance of unnecessary surgery, shortening the treatment period and leading to early rehabilitation.

Keywords: degloving injury, replantation, debridement

Introduction

Degloving injury is the most serious among various types of finger trauma[1, 2]. The bone and skin/soft tissue are markedly damaged in many cases, and it is difficult to establish an adequate extent of debridement on initial surgery. Therefore, several sessions of surgery, such as additional debridement for necrotic tissue related to the progression of circulatory failure, are required in many cases. In this study, we performed replantation for degloving injury in 2 patients and obtained findings regarding nailbed treatment on initial surgery.

Case

[Case 1] A 39-year-old male.

Degloving injury of the right thumb and index finger. During work, the right thumb and index finger were mis-involved in the roller of a printing machine, and injured (Figure 1). Replantation was performed. In both the thumb and index finger, end-to-end anastomosis of a single ulnar digital artery and dorsal cutaneous vein was performed, respectively. Furthermore, finger nerves on the ulnar side of the thumb and radial side of the index finger were sutured. Drainage from the thumb nail margin was observed 19 days after surgery, and nail removal was conducted. Subsequently, survival of the nailbed of the index finger was achieved, but the nailbed of the thumb became necrotic, and the distal phalanx was exposed. The distal phalanx of the thumb exposed to the nailbed area was resected 36 days after surgery, and amputation stump plasty was performed.

[Case 2] A 21-year-old male.

Degloving injury of the right index finger. His right index finger was involved in a roller for paper tube preparation, leading to degloving injury. The exfoliated skin was connected with the center, but there was no circulation. The radial digital artery remained on the exfoliated skin side, and this was anastomosed with the central digital artery. The ulnar digital artery remained on the exfoliated skin side was anastomosed with the central digital artery by use of venous transplantation.

Necrosis of the nailbed appeared 5 days after surgery. Furthermore, the distal phalanx was exposed from the same site 11 days after surgery. Debridement of the necrotic nailbed and distal phalanx was performed 21 days after surgery, and reconstruction with an abdominal wall flap was conducted.

Discussion

Degloving injury refers to serious trauma of the fingers. For initial treatment, replantation, amputation, free skin grafting, negative pressure wound therapy, or grafting of remote flaps, such as abdominal or inguinal flaps, or free flaps is selected. In any case, adequate debridement of the tissue in which recovery is not expected must be performed.

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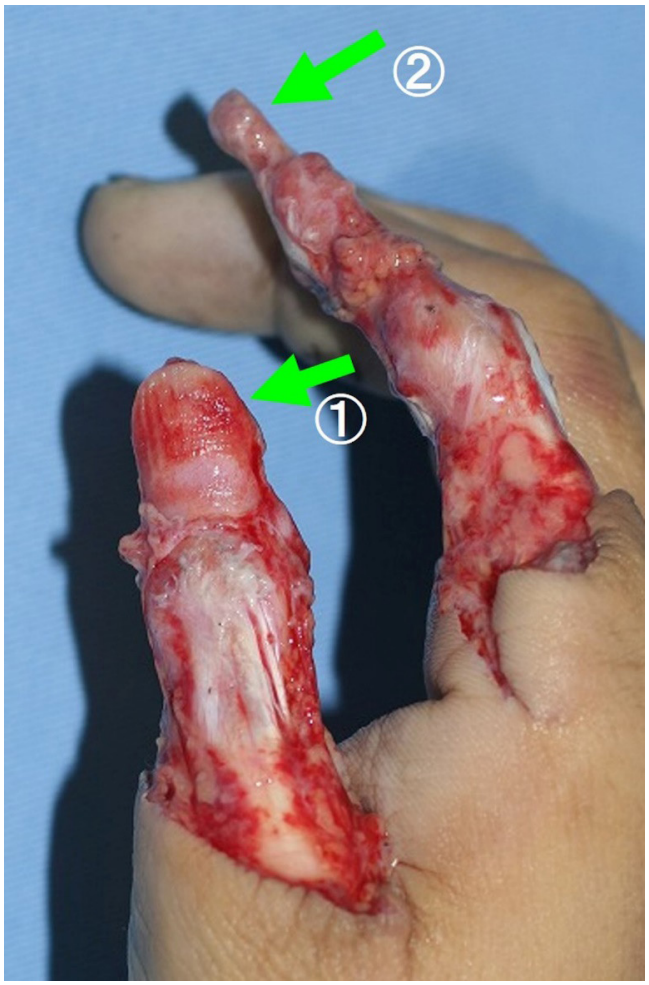

Figure 1: Before surgery, the nailbed was macroscopically confirmed in the Arrow-area. In the Arrow-area, the cortex of the distal phalanx was exposed, and the nailbed could not be confirmed.

We retrospectively examined photographs and X-ray films regarding necrosis of the nailbed after surgery in 2 of 3 fingers with degloving injury. In Case 1, necrosis of the thumb nailbed occurred, but graft survival of the index finger was achieved. We reconfirmed photographs/plain X-ray films at the time of injury (Figure 2)., as well as plain X-ray films 20 days after surgery (Figure 3). In the nailbed of the thumb, a shadow of soft tissue was observed on plain X-ray. Macroscopically, the nailbed was adhered to the distal phalanx, whereas there was no shadow of soft tissue in the area corresponding to the nailbed of the index finger. When confirming plain X-ray films 20 days after surgery, avulsion fracture was observed; therefore, the nailbed of the index finger may have remained in the soft tissue exfoliated with avulsion fracture. This may have contributed to graft survival of the nailbed of the index finger, in which circulation from the soft tissue after revascularization may have been present, and necrosis of the thumb nailbed adhered to the distal phalanx. In Case 2, the nailbed remained at the distal phalanx of the index finger, as demonstrated for the thumb in Case 1. Necrosis of the nailbed occurred 21 days after surgery, requiring additional debridement.

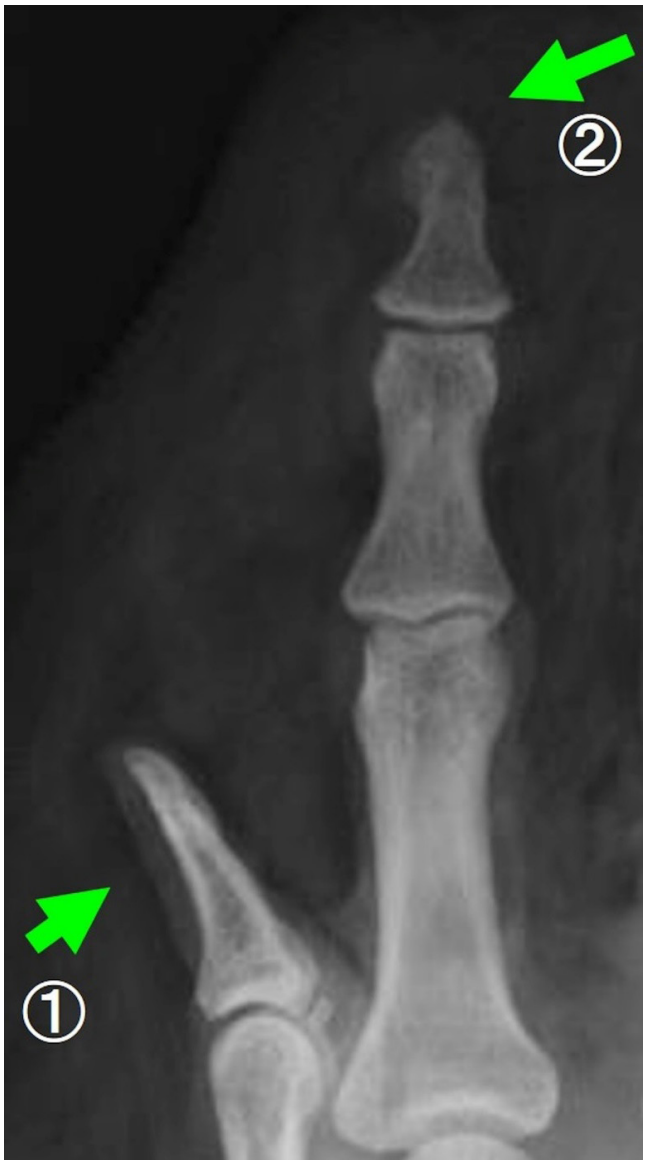

Figure 2: Plain X-ray on the day of injury in Case 1. In the Arrow-area, a shadow of soft tissue suggestive of the nailbed was observed. There was no shadow in the Arrow.

Nailbed circulation involves the course of lateral vessels branching from the digital artery to the periphery along the lateral margin of the distal phalanx, intra-nailbed anastomosis/arch formation/microvascular network formation with dorsal vessels running through the lateral intraosseous membrane and lateral margin of the distal phalanx to the dorsal side, and anastomosis with the palmar vascular network at the end of the microvascular network[3-5]. Circulation to bones exposed due to degloving injury depends on the medullary cavity, periosteum, and peripheral soft tissue. Even when revascularization for an exfoliated tissue is performed, bone atrophy occurs through bone resorption in most cases. As demonstrated in Cases 1 (thumb), 2 (nailbed remaining in the distal phalanx), proximal and distal nailbed circulation ruptures despite reconstruction by vascular anastomosis and covering with a tissue with circulation. Furthermore, there is

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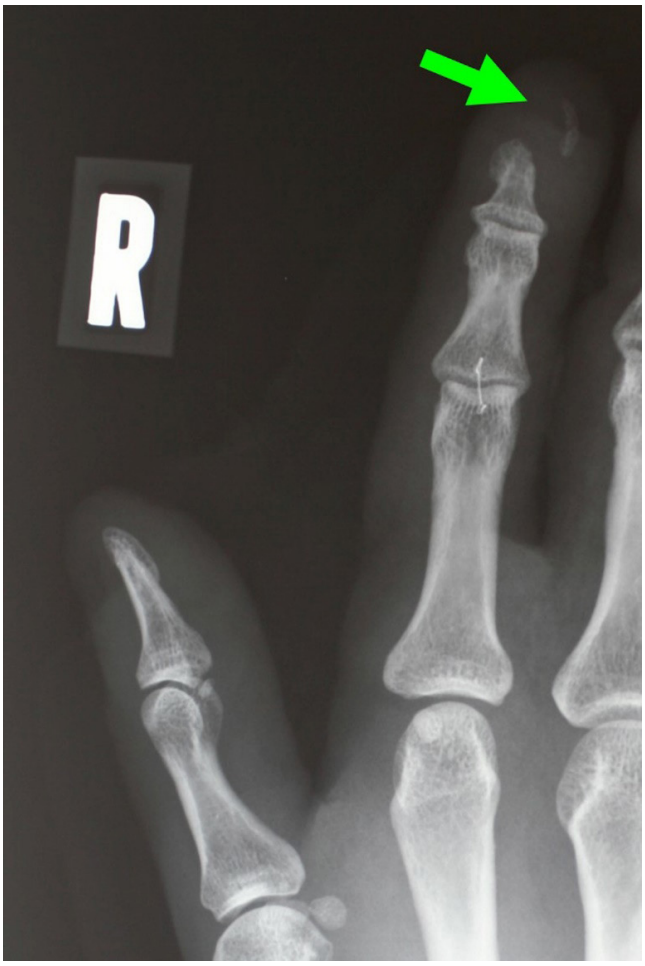

Figure 3: Plain X-ray 20 days after initial surgery. At the end of the index finger (Arrow), avulsion fracture was noted.

no contact point of the nailbed with the skin after revascularization, differing from the distal phalanx in contact with the skin after revascularization; therefore, prompt circulation may not be achieved. For this reason, concerning replantation for degloving injury in which the nailbed remains in the bone at the time of injury, debridement of the nailbed and distal phalanx to which the nailbed is adhered to on initial surgery may facilitate the avoidance of unnecessary surgery.

There are various opinions regarding the indication of surgery for degloving injury or methods. When performing replantation of an amputated finger for degloving injury, whether or not this procedure should be indicated must be examined, considering functional problems of the finger over a long course, the admission period, hospital visits, and several sessions of additional surgery.

Conclusion

Degloving injury is a serious trauma of the fingers. It is difficult to establish an adequate extent of debridement on initial surgery. Concerning degloving injury in which the nailbed remains in the bone, debridement of the nailbed and distal phalanx to which the nailbed is adhered to on initial surgery may facilitate the avoidance of unnecessary surgery, shortening the treatment period and leading to early rehabilitation.

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Case Report

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Intoxication Caused by Paraphenylenediamine After Henna Ingestion

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Abstract

Introduction: Henna, has been applied in eastern cultures for many years, as a hair dye and tattoo. Paraphenylenediamine (PPD) is a kind of aromatic amine added to henna. The formulation formed when PPD added known to be highly toxic.

Case report: Here, a 16-year-old patient admitted to an emergency clinic with severe angioedema, rhabdomyolysis, and acute renal failure caused by PPD after henna ingestion for suicidal purposes, was presented. An emergency tracheotomy was performed to the patient because of severe respiratory distress, and the patient was followed up by connecting to the mechanical ventilator in intensive care.

Conclusion: PPD poisoning is a life-threatening condition, and there is no specific antidote for PPD intoxication. Early intervention to the patient is essential because it is life-saving.

Key Words: Acute renal failure, Angioneurotic edema, Henna dye, Para-phenylenediamine, Rhabdomyolysis, Tracheotomy

Introduction

Henna, an extract of the Lawsonia plant, has been used in many societies for many years to dye hair, hands, and feet. It is popular in some East African countries, the Middle East, and India. Paraphenylenediamine (PPD) is an aromatic amine commonly used in various industrial products, and it usually is not found in nature[1]. PPD is added to intensify the henna color and speed up the dyeing process. Topical application of PPD sensitive people to dermatitis; may cause an increase in lacrimation, persistent blepharoconjunctivitis, and permanent blindness in local eye contact[1-3]. In 1924, the first case of PPD poisoning was reported in a hairdresser[4]. Ingestion of PPD results in severe facial, neck, tongue, and laryngeal edema with respiratory distress, which often requires urgent tracheostomy[2]. Henna intake containing PPD has a high mortality rate [up to 31%] caused by rhabdomyolysis and kidney failure[5]. The authors reported systemic poisoning with PPD, leading to angioedema resulting in tracheostomy and acute renal failure.

Case Report

A sixteen-year-old female patient was admitted to the Nyala Sudan-Turkey Training and Research Hospital emergency clinic complaining of not breathing. It was learned that the patient had ingested henna in an attempt at suicide two hours before. Diffuse edema was seen in the patient's eyes and around her face and neck. Her face and neck were swollen, and the airway was obstructed. On examination, the patient was agitated and had severe respiratory distress. The patient was cyanotic and vital signs; the pulse was 130 / min, blood pressure 160/95 mmHg, and the breathing rate was 35 / min. The oxygen saturation level of the patient was at 85% during follow-up and continued to decrease. We performed an emergency tracheotomy to keep the patient's airway open (Figure 1). Taking the intensive care unit, we connected her to a mechanical ventilator. Gastric lavage was performed by gently applying a nasogastric tube, and toxic stomach contents were removed.

The patient's head and neck edema increased by six to eight hours after starting to follow in the intensive care unit. Henna, which caused acute kidney failure due to the PPD it contains, caused the chocolate brown color urine outflow (Figure 2). The patient's potassium, Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), Creatinine Kinase (CK), and creatinine values increased (Table 1).

The patient was managed with intravenous corticosteroid [methylprednisolone 1 mg/kg], rehydration and alkaline diuretic therapy for two days. On the third day, the edema of the patient's face and neck began to decrease distinctly. The dark urine color turned light, and urine output started to increase. Serum creatinine values continued to decrease without the need for dialysis (Table 1). The patient was taken off the mechanical ventilator at the end of the third day, and then she

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Table 1. Biochemical values of the patient

	Creatinine (IU/L*) (mean 0-1.3)	CK** (IU/L*) (mean 0-145)	Potassium (mEg/l‡,) (mean 3.5-5.5)	ALT† (IU/L*) (mean 0-45)	AST†† (IU/L*) (mean 0-35)
1st day	0,9	1453	5.7	1657	1350
2nd day	3.4	2237	5.9	1309	1232
3rd day	7.8	1807	5.3	705	549
5th day	6.7	761	4.9	578	345
7th day	4.4	323	5.0	234	141
10th day	1.1	165	4.4	41	15

**IU/L: International unit/Liter, **CK; Creatinine Kinase, ‡mEg/l: Milliequivalent per Liter, †ALT; Alanine aminotransferase, ††AST; Aspartate aminotransferase*

was taken to the clinic from the intensive care unit on the fifth day. On the seventh day, the patient was decannulated, and the tracheostomy site was closed. After mobilizing, she continued to recover more quickly, and all metabolic values returned to normal. The patient started oral intake of liquid foods and was discharged from the hospital on the 11th day.

Discussion

PPD is an allergen that can cause contact dermatitis, erythematous urticarial papules, and eczema in susceptible individuals after contact or application and the frequency of PPD allergies in the general population has increased over the years[3,6]. PPD is a highly reactive substance and has a half-life of a few hours on human skin. Skin exposed to PPD has been shown to cause extensive transcriptomic modifications, including tight connectivity and down-regulation of stratum corneum proteins, even in the nonexistence of clinical manifestations[7]. The most significant problem arises when PPD is taken orally, whether accidentally or for attempted suicide or murder. Systemic effects include angioedema, especially in the face and throat, kidney and rhabdomyolysis, and heart toxicity. While accidental intake is seen in children, among adolescents and women, as in this case, it is often drunk for suicide purposes[8].

Figure 1. The patient has severe angioedema during the initial admission to the emergency clinic

While the most common cause of tracheotomy in children used to be infections [epiglottitis, laryngitis], today, it has changed to prolonged intubation. Bezgin et al. reported that the most common tracheotomy indications in children were neurological deficits and cardiopulmonary disease[9]. As in our case, airway obstruction due to acute toxicity is much rarer. PPD begins to form diffuse angioneurotic edema in the oral mucosa, tongue, face, head, and neck regions within two hours of oral intake. Rapidly progressive tissue destruction and edema reach life-threatening levels in a short time. If emergency airway safety cannot be achieved, death will be caused by asphyxia. When our case entered the emergency department in the second hour, her level of edema required an urgent tracheotomy. Laryngeal edema and pulmonary congestion were detected in a postmortem autopsy of another suicide case[8].

Chugh et al first described kidney failure due to PPD poisoning in 1982[2]. The development of kidney failure is related to the dose taken. The beginning of oliguria after oral intake is a known sign that kidney failure will develop. Chocolate- or coffee-colored urine of the patients is a characteristic finding of PPD intoxication. All patients develop rhabdomyolysis, and approximately 80% of patients develop acute renal failure. Dialysis may be needed in the management of patients[6]. Dialysis is for support purposes only, as the toxin is not dialyzable. Kidney failure is thought to be due to myoglobinuria and the primary toxic effects of

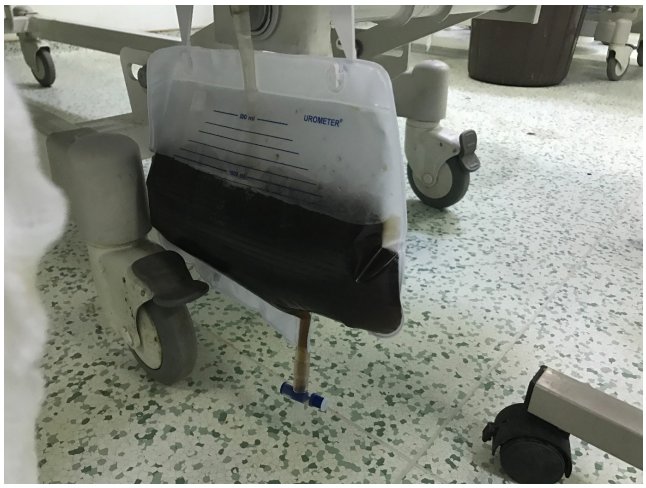

Figure 2. Typical chocolate brown urine

PPD. Acute tubular necrosis was found in a kidney biopsy[10]. Renal failure causes death in patients with airway safety effects. Electrolyte imbalances due to kidney failure lead to cardiac arrhythmias. This situation increases hospitalization time, morbidity, and mortality. Since there is no specific antidote for PPD intoxication, hydration and alkaline diuretic therapy should be started immediately upon diagnosis to protect the kidneys[2,6].

It is most important to diagnose a patient early and keep the airway open by endotracheal intubation or tracheotomy[6]. Intensive supportive treatment should be carried out. Afterward, ventilation should be provided with a mechanical ventilator in intensive care conditions. Gastric lavage should be undertaken with a gentle nasogastric tube application. It is recommended to use steroids on the first day to reduce edema in the head and neck area. Progression of kidney failure can be prevented with urgent and abundant fluid infusion therapy. To determine whether dialysis is needed, electrolyte values should be closely monitored. Alkaline diuretic therapy can be given to offset the metabolic acidosis that occurs. The use of diuretics should be started with hydration and discontinued after the first 24 hours. The use of vasopressor agents may be required[2].

Conclusion

PPD poisoning is a life-threatening condition. The condition is associated with a high risk of multisystem involvement, tracheostomy requirement, prolonged hospital stays, morbidity, and cardiac arrhythmias. The severity of symptoms is directly related to the dose of PPD taken. It is crucial to start supportive therapy immediately since there is no specific medicine known for PPD intoxication. Urgent supportive therapy can be life-saving in cooperation with early recognition, rapid referral, and appropriate specialties. We presented this case because it is rare in our country, and we wanted to draw the attention of the clinicians to this urgency.

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