Case Report

Journal of Emergency Medicine Case Reports

An application to Keep in Mind in Cases of Pulmonary Thromboembolism: Positive Airway Pressure System

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Abstract

Massive pulmonary embolism is a vital emergency pulmonary pathology. Early diagnosis and treatment approach reduce mortality. Thrombolytic applications play an important role in the treatment. However, complications of thrombolytic therapy put a serious strain on physicians. In this case series, we aimed to present the management of carbon dioxide retention with the EzPAP® device in two young massive embolism patients who were given thrombolytic therapy. Our 26 and 36-year-old male patients applied to our emergency department with complaints of flank pain and syncope, respectively. Massive PTE was detected in PCTAs taken due to hypoxia and tachycardia in our patients. When carbon dioxide retention developed in patients who were decided to be given thrombolytic therapy, barotrauma, which could be caused by non-invasive mechanical ventilation, was withdrawn. And it was decided to apply EzPAP® to these patients. In both patients, hypoxia and carbon dioxide retention were treated in a short time without complications. Etiological investigations of the patients are continuing and they were discharged with convalescence. While thrombolytic therapy provides rapid and positive results, it also includes serious life-threatening side effects such as bleeding. EzPAP® can be used as an alternative treatment method for thrombolytic-related bleeding that may develop in the face and respiratory tract due to barotrauma of non-invasive mechanical ventilation.

Keywords:PEzPAP®, carbon dioxide retention, massive pulmonary thromboembolism, thrombolytic therapy

Introduction

Pulmonary thromboembolism (PTE) is an important preventable clinical problem with high mortality and morbidity. Symptoms such as shortness of breath, chest pain, and hemoptysis are not specific to the disease and can be seen in many cardiopulmonary diseases. Since shock and cardiopulmonary arrest may be clinical in massive cases, it is important to go for diagnosis and treatment quickly. PTE develops when pieces of deep vein thrombosis (DVT) in the leg occlude the pulmonary artery and/or its branches. When advanced diagnostic methods are used, deep vein thrombus is detected in 79% of PTE patients. PTE and DVT are also called venous thromboembolism (VTE). Pulmonary thromboembolism is the third most common acute cardiovascular disease after myocardial infarction and stroke. Pulmonary thromboembolism is a frequent complication of hospitalizations and one of the leading causes of preventable hospital deaths[1,2]. VTE is seen in 23-269 per hundred thousand per year. 30-day mortality due to PTE is 1.8%. Prior surgery, immobility, cancer, major trauma and obesity are important risk factors. In low-risk patients, low molecular weight heparin or standard heparin is started and added to oral anticoagulant therapy[3]. Thrombolytic methods are used effectively in the treatment of massive PTE. It is necessary to avoid unnecessary procedures that may cause complications such as invasive, traumatic and barotrauma to patients during thrombolytic therapy[4]. EzPAP® (a noninvasive positive airway pressure device) is a simple, manual device that can be used in cases of acute atelectasis. It creates a continuous positive airway pressure (CPAP) effect. Thus, it aims to prevent good oxygenation and carbon dioxide (CO_2) retention in patients[5-6].

In this case series, we aimed to present the management of carbon dioxide retention with the EzPAP® device in two young massive embolism patients who were given thrombolytic therapy.

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Case-1

A 26-year-old male patient applied to the emergency department with the complaint of left flank pain for 3-4 hours. After, the patient had syncope. Glasgow coma score (GCS): 15, general condition was good, conscious,

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cooperative, oriented. In his vitals, the patient had arterial blood pressure: 93/63 mm/Hg, pulse: 115/minute, fever: 36.2 C°, oxygen saturation 90% at room air, and had no additional complaints. Electrocardiography (ECG) was sinus tachycardia. The patient had a known diagnosis of autism. There was no significant change in the blood of the patient, whose physical examination did not reveal any active pathology, except for the white blood cell count of 11,000, high sensitive troponin I was 660 ng/L and C-reactive protein (CRP): 140 mg/L. Unfortunately, the d-Dimer level could not be measured even though it was repeated due to sample error. In the pulmonary computed tomography angiography (PCTA) report taken in the emergency room, the diameter of the pulmonary trunk increased and thrombus material was observed that caused the filling defect extending from the pulmonary trunk bifurcation to the right and left main pulmonary arteries, and a hampton hump secondary to pulmonary embolism was observed in the left lung lingula and lower lobe posterobasal. In the ECHO cardiogram of the patient, the left ventricular ejection fraction was interpreted as 50%, global mild hypokinesia, minimal mitral insufficiency, 1-2 tricuspid insufficiency, right heart chambers wide, spab: 35 mmHg. The patient was started on alteplase in the form of an intravenous (iv.) infusion in 2 hours. During the administration of thrombolytic therapy, carbon dioxide (CO_2) retention was observed in the patient. We decided to start EzPAP® because the patient was afraid of the barotrauma effect of the thrombolytic due to hypoxia and CO_2 retention. Our initial blood gas values were as follows. pH was 7.24, oxygen saturation 83.8%, pO_2 count was 45, pCO_2 count was 58 and HCO3 was 19. After two hours, pH was 7.40, oxygen saturation 93%, pO_2 count was 47.4, pCO_2 count was 38 and HCO3 was 23.4.The patient, who was stabilized clinically, was transferred to the intensive care unite for treatment and follow-up by pulmonologist. Systemic lupus erythematosus was determined as the etiological cause. The lupus anticoagulant scan came in 75.75 seconds. But the patient, whose etiological investigation continued, was discharged with convalescence.

Case-2

A 36-year-old male patient applied to the emergency department after dispnea and syncope in the bathroom for 5 minutes. GCS: 15, general condition was good, conscious, cooperative, oriented. His vitals were arterial blood pressure: 91/55 mm/Hg, pulse: 127/minute, fever: 36.2°C, oxygen saturation at room air was 78%, and the patient had no additional complaints. ECG was sinus tachycardia. Physical examination did not reveal any active pathology. Glucose value was 452 mg/dL, high sensitive troponin I was 168 ng/L, d-Dimer was 21.27 mg/L and CRP was 112 mg/L. In the PCTA taken in the emergency department, there were filling defects consistent with diffuse PTE in the bilateral main pulmonary artery and its branches. Right heart chambers were enlarged, and there was significant diameter reduction in the descending aorta and abdominal aorta. The patient, who was compatible with massive pulmonary embolism, was started on iv. alteplase infusion in 2 hours as thrombilytic therapy. After the treatment was finished, CO_2 retention was observed in the patient. in venous blood gas, pH was 7.06, pCO_2 was 66.2, HCO_3 was 18.3 and oxygen saturation %88. It was decided to apply EzPAP®, as the patient developed carbon dioxide retention and the application of non-invasive mechanical ventilation simultaneously with after thrombolytic therapy could cause complications. 2 hours after the start of EzPAP®, the venous blood gas values became pH was 7.11, pCO_2 was 57, HCO_3 was 17.9 and oxygen saturation was %95. The patient who benefited from the EzPAP® application was admitted to the intensive care unite for treatment and follow up by pulmonologist. The patient, whose etiological investigation continued, was discharged with convalescence.

Discussion

Pulmonary embolism is one of the life-threatening thoracic emergencies that the emergency physician should consider in patients presenting to the emergency department with symptoms such as chest pain, dyspnea, tachypnea and hemoptysis. In patients diagnosed with massive embolism, close vital hemodynamic monitoring should be performed, necessary treatments should be started in terms of hemodynamic stability, and invasive procedures should be completed before treatment if there is an indication for thrombolytic initiation. In the treatment of PTE, emergency physicians should keep in mind the application of EzPAP® in addition to other oxygen treatments such as nasal cannula, balloon mask and noninvasive mechanical ventilation (NIMV)[7]. The NIMV provides respiratory support with positive pressure continuously or separately during expiration and inspiration. It is especially used in cardiogenic pulmonary edema and chronic obstructive pulmonary disease. Treatment success is demonstrated by the improvement of oxygenation and a decrease in pCO_2 in the first 2 hours. During treatment, it may cause facial erythema due to pressure and mask, nasal congestion, and ulceration and irritation on the mucosal surfaces[8]. Atelectasis developing in the postoperative period leads to hypoxia. Treatment with positive end-expiratory pressure (PEEP) can often be used in this period. Although different devices can be used for this, the EzPAP® system is an alternative method. With 5-8/L oxygen flow, this device provides 35-42% fractionated oxygen. It does this by increasing the oxygen flow fourfold. An increase in lung volume provides positive expiratory pressure during expiration. With less effort, atelectasis lung tissue begins to oxygenate. The superiority of the devices used in the treatment of

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this hypoxemia due to atelectasis is not fully evident[9,10]. Both of our patients had massive pulmonary embolism syncope and deep hypoxia before coming to the emergency department. We started oxygen support with nasal cannula for our patients. However, CO_2 retention accompanying hypoxia also developed in the patients. In the meantime, our patients after thrombolytic therapy needed respiratory support with NIMV. We decided to apply EzPAP® because of the risk of barotrauma complications. This device has shown successful results in post-operative hypoxia and CO_2 retention. Saturation, increase in oxygen and decrease in CO_2 values were easily achieved. Patient compliance and ease of application also provide an important advantage[10,11].

In our case series, attention was drawn to the convenience provided by EzPAP® in clinical follow-up after thrombolytic therapy. Respiratory support was provided in two of our cases in EzPAP® treatment, and improvement in blood gas values 2 hours later and clinical improvement were observed. This treatment method gave us an advantage to avoid the complication of barotrauma (especially subcutaneous or mucosal bleeding, airway obstruction due to hematoma) after thrombolytic treatment. Anxiety in hypoxia patients may cause compliance problems at the point of application of NIMV. These 2 patients, to whom we applied EzPAP®, adapted easily to the device.

Conclusion

Considering that such patients may need NIMV; Simpler device applications such as EzPAP® that provide positive expiratory pressure and can be used to reduce carbon dioxide retention can be used. In this way, the patient is not affected by the complication of NIMV application, which may cause barotrauma during thrombolytic therapy.

Declaration of competing interest: *The authors have no outside support information, conflicts or financial interest to disclose. Informed consent from the patient has been obtained.*

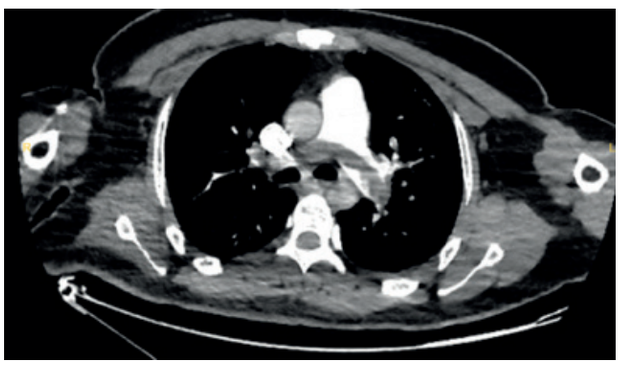

Figure 1. Presence of thrombus in the bilateral main pulmonary artery of Case 1

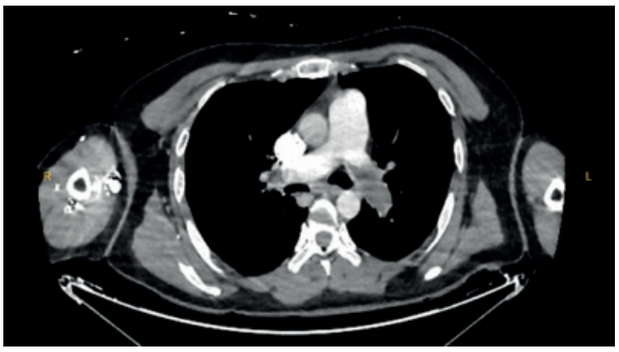

Figure 2. Filling defect in bilateral main pulmonary artery and branches of Case 2.

Figure 3. Positive Airway Pressure System; EzPAP®.

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Is Hiccups a Precursor to a Stroke?

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Abstract

Hiccups that last more than 48 hours are known as persistent hiccups. A 38-year-old man presented to the emergency with a 3-day history of persistent hiccups, and after partial treatment and normal test results, he was discharged from the emergency. After 22 hrs, he was brought to the emergency again with a complaint of stuttering and was admitted with a chance of stroke. A 50% stenosis development in the right carotid artery was reported, and the MRI results were normal. After 20 hrs of admission, the patient's stutter was thoroughly treated, and he was discharged with the diagnosis of TIA. Persistent hiccups can be a precursor to a stroke.

Keywords: Persistent hiccups, stroke, TIA

Introduction

Hiccups are a form of continuous and involuntary jerks of diaphragmatic muscles, with spasmodic contractions occurring along with early glottic closure disrupting the inspiration.[1] Hiccups are commonly transient and classified depending on the duration. Hiccups are identified as persistent or chronic when they last more than 48 hours.[2] Regardless of the fact, that persistent hiccups are considered a rare medical condition, they can have a detrimental impact on an individual's life.[3]

Though hiccups may potentially be regarded as being caused by metabolic gastrointestinal abnormalities, they might be an indication of the lesions located in the brainstem. [4],[1, 2] Herein, we present a case of a 38-year-old man with persistent hiccups.

Case Report

A 38-year-old man presented to the emergency with a 3-day history of persistent hiccups. He visited a general physician twice, but the hiccups were refractory to several medications. Even traditional treatments and homemade remedies were ineffective. Subsequently, his situation didn't improve.

The patient was a heavy smoker (~40 cigarettes per day) with a history of coronary artery angiography of 4 years earlier, which showed a 50% stenosis in a coronary artery. He complained of nausea, epigastric pain, and continuous hiccups. In the physical examination, the patient was alert and oriented. Other than mild dehydration and epigastric tenderness, there were no other abnormalities.

There were no pathologic findings in echocardiography (ECG). Cardiac troponin and other routine laboratory tests were normal. After a prescription of ampule Chlorpromazine 50 mg and Haloperidol 2.5 mg, the patient was temporarily treated. Considering clinical, laboratory, and imaging findings being normal, he was discharged from the ER.

After 22 hours, the patient was brought to the emergency again, complaining of stuttering. In the examination, the patient was stuttering and had difficulty spelling some words, and his muscle force was normal. Afterward, he was admitted to the neurology ward. In Echocardiography (ECG), Ejection Fraction (EF) = 50% and mild MR (Mitral Regurgitation),

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and in Color Doppler Sonography, hypoechoic plaque in the right carotid bulb, with an expansion into the internal carotid and a stenosis development <50% was reported.

In Magnetic Resonance Imaging (MRI) in the ER, there was no other finding other than bilateral sinusitis in ethmoidal sinuses.

The patient's stutter was thoroughly treated after 20 hours of admission. Eventually, he was diagnosed with a Transient Ischemic Attack (TIA) and discharged home without any symptoms.

Discussion

Hiccups are continuous and myoclonic contractions of diaphragmatic muscles, with an early closure of the glottis disrupting the air exchange. Hiccups based on the duration are categorized as transient, which is the most common, and persistent or chronic, which last more than 48 hours. Transient hiccups are mostly caused by factors like alcohol, tobacco, eating too many spicy foods, or overeating in general.

Although the etiology of persistent hiccups is not particular, however, gastrointestinal abnormalities are likely to cause persistent hiccups. On the other hand, persistent hiccups can be elicited by lesions located in the central nervous system.

The posterior cranial fossa is the largest and the most profound cranial of three cranial fossas, which incorporates the cerebellum and the brainstem. The brainstem is the part of the brain, which controls body functions like breathing. We think our patient was having persistent hiccups as a symptom of a Transient Ischemic Attack, which we believe occurred in the arteries of the hiccups center, presumably located in the brainstem near the inspiratory center.

Transient Ischemic Attack (TIA) has similar symptoms to a stroke, but it is temporary. A TIA is also called a mini-stroke, which can be an indication of a future stroke. Almost 30% of the people who have had TIA are likely to have a stroke. In addition, hiccups, particularly persistent hiccups, might be a symptom of a TIA. Hence, persistent hiccups can be a precursor to a stroke.

Regarding the patient's initial presentation and their main complaint of hiccups, the final diagnosis of an ischemic stroke in the Posterior Cranial Fossa, in further studies of elderly patients presenting to hospital with persistent hiccups that are resistant to regular treatments, one of the main diagnoses that need to be considered is Cerebrovascular event.

Conclusion

TIA has similar symptoms to a stroke. Almost 1 in 3 patients with a TIA tends to have a stroke. Our patient had persistent hiccups and was diagnosed with a TIA. Taking that into account, persistent hiccups as a symptom of a TIA can be an indicator of an ischemic stroke.

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Rhino-Orbital Mucormycosis After Covid-19 In the Emergency Department

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Abstract

Rhino-orbital mucormycosis (ROM) is an acute and fulminant infection. The number of ROM cases developing after coronavirus disease 2019 (COVID-19) is increasing. A 62-year-old male patient was admitted to the emergency department in northern Syria complaining of new-onset vision loss, swelling, and severe swelling of the left eye. It was noted that a 3-day course of prednisolone 250 mg was given to treat COVID-19. We found that he had a history of diabetes mellitus (DM) for 10 years. Physical examination revealed ptosis, proptosis, and ocular movement restriction in all directions in the left eye. All other systemic examinations were normal. A cranial and orbital magnetic resonance imaging scan performed after hospitalization for further evaluation and treatment showed an increase in the density of the sphenoid, ethmoid, and frontal sinus walls in the left orbit. Despite antifungal and surgical treatment, the patient died on day 14. The use of steroids in treatment and the presence of concomitant DM are the main predisposing factors. The prognosis of this disease, which has a high mortality and morbidity, is adversely affected in geographic regions where health care is inadequate.

Keywords: COVID-19, emergency service, mucormycosis, rhino-orbital mucormycosis

Introduction

Rhino-orbital mucormycosis (ROM) is an acute and fulminant infection caused by Mucoraceae fungi and occurs in immunocompromised and diabetic patients[1]. While the paranasal sinuses and nose are the primary sites of inoculation, these aggressive fungi can cause life-threatening infections in the orbit and brain through direct or hematogenous spread. Coronavirus disease 2019 (COVID-19 disease) affects T lymphocytes and causes immunosuppression lymphopenia. In addition, the presence of concomitant diseases such as diabetes mellitus (DM), the use of steroid therapy, and the use of other immunomodulatory drugs cause immunosuppression that predisposes these patients to opportunistic fungal infections[2,3]. The coexistence of COVID-19 with invasive ROM is rarely reported in the literature, and diabetes mellitus is often cited as the major predisposing factor. In this case report, a rare case of ROM was reported that developed in a diabetic patient who received high-dose steroid therapy due to COVID-19 treatment.

Case Report

A 62-year-old male patient was admitted to the emergency department in northern Syria complaining of new-onset vision loss, pain, and severe swelling of the left eye. It was noted that he had received 10 days of inpatient treatment for COVID-19 thirty days ago. As part of the treatment, favipiravir, chloroquine, acetaminophen, and 3 days of prednisolone 250 mg were administered. The patient reported that he had had DM for 10 years. The patient's vital signs were stable on admission. Physical examination revealed ptosis, proptosis, and ocular movement restriction in all directions in the left eye (Image 1). All other systemic examinations were normal. A cranial and orbital magnetic resonance imaging scan performed after hospitalization for further evaluation and treatment showed an increase in the density of the sphenoid, ethmoid, and frontal sinus walls in the left orbit (Image 2). A biopsy which had taken from the patient showed Rhizopus spp. IV Amphotericin B was started for the treatment of ROM. Surgical treatment included exenteration of the left eye and sinus drainage. The patient died on day 14 due to multiorgan dysfunction.

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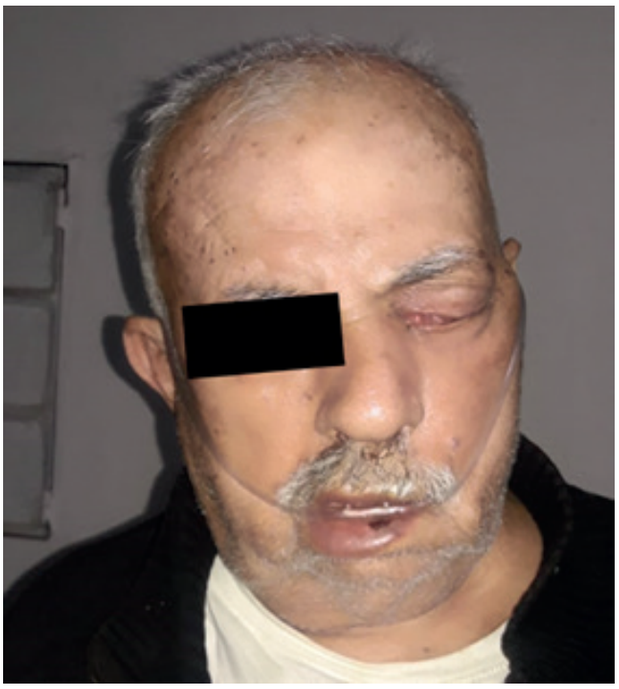

Image 1. Photo of the patient: Face of the patient shows exophthalmos and ptosis in the left eye, nasal deviation to the right, left maxillofacial swelling and edema and right deviation of the vestibulum orris.

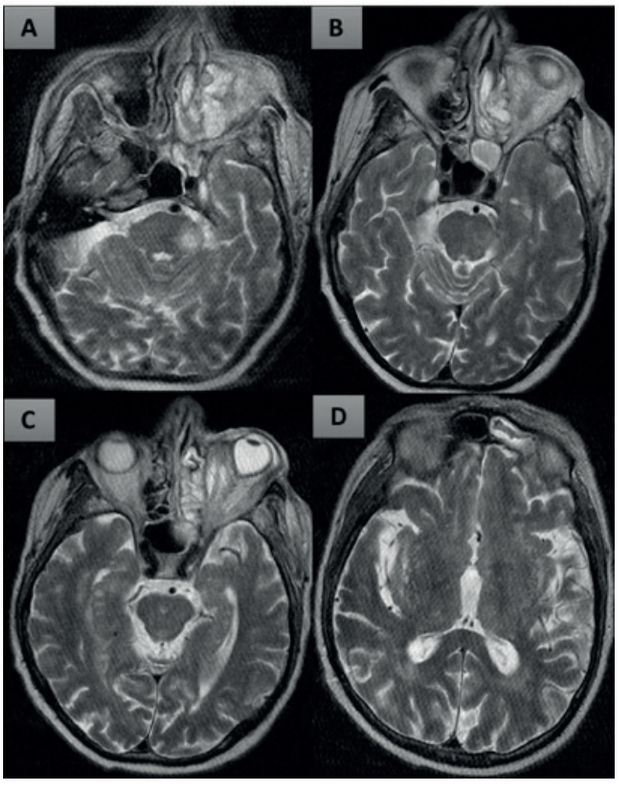

Image 2. Magnetic resonance images of the patient: As a result of mucormycosis; increased density in the left maxillary sinus, deviation of the nasal septum (A), increased density in area of the left parasphenoid and medial paraorbital (B), increased density in the left ethmoid sinus and exophthalmos in the left orbit (C), increased density in the left frontal sinus (D).

Discussion

ROM is a rare disease that is difficult to diagnose and has high mortality and morbidity. Immediate early diagnosis and treatment is lifesaving[4,5]. Although a multidisciplinary approach is of great importance in improving prognosis, treatment approaches may vary depending on the conditions of the health care system. The current treatment modalities include pharmacological treatment following surgical intervention. But the current condition of the health care system in northern Syria from the perspective of resource scarcity and availability of current treatments makes the treatment of rare and deadly diseases more challenging.

DM and corticosteroid therapy are the most common risk factors in ROM cases that develop after COVID-19 [6]. At the same time, medical treatments, hospitalization in the intensive care unit, and treatment with broad-spectrum antibiotics predispose COVID-19 patients to opportunistic fungal infections, which occur in 8% of patients[7,8]. DM remains the most important risk factor in ROM after COVID-19 [9,6]. Besides, dexamethasone, hydrocortisone, and prednisolone are reported to be used in most cases that developed ROM. Although steroids are proven to be beneficial in COVID-19, cautious use is recommended because they cause hyperglycemia and monocytic and neutrophil dysfunction. The dose and duration of steroid treatment which may cause ROM remains uncertain in patients with Covid-19.

According to the European Mycosis Working Group guidelines, treatment success depends on the applicability of surgical techniques and the availability of antifungal therapies[4]. For the maximization of the survival rates a multidisciplinary team consisting of medical, surgical, radiological and laboratory specialities, and rapid diagnostic and therapeutic interventions are required. According to this guideline, the use of systemic liposomal amphotericin B along with early surgical debridement with clean margins is recommended as first-line therapy. The challenging decision of exenteration of the occupied orbit is recommended to remove the fungal load and to prevent cerebral involvement. In addition, intravenous isavuconazole or delayed-release tablet posaconazole are recommended but not Amphotericin B deoxycholate because of its toxicity. In assessing the postwar situation in northern Syria, we believe that the limited availability of the drugs, the lack of multidisciplinary approach and technical equipment for biopsy and surgical treatment negatively affect the prognosis.

Early diagnosis and treatment are very important for the prognosis of the disease[10]. In addition to physical examination findings, radiological imaging and knowledge of risk factors can be helpful for early diagnosis. Biopsy and detection of fungi in CSF or sputum are required for a definitive diagnosis. Evidence of nodular thickening or destruction of the bony walls of the paranasal sinuses and

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the absence of an air-fluid level on imaging may be a sign of invasive opportunistic infection.

Conclusion

The number of ROM cases is increasing after COVID-19. The use of steroids in treatment and the presence of concomitant DM are the main predisposing factors. The prognosis of this disease, which has high mortality and morbidity, is adversely affected in geographic regions where health care is inadequate.

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Case Report

Journal of Emergency Medicine Case Reports

A Fatal Case of Culture-Negative Late Prosthetic Mitral Valve Endocarditis

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Abstract

Prosthesis valve dehiscence is one of the most serious complications of prosthetic valves. Despite the improvements in medical and surgical treatments, prosthesis valve dehiscence has high mortality. Due to fatality rates, the early diagnosis and treatment of these patients is required. In this case report, we presented a patient with severe mitral deficiency and acute heart failure following mechanical mitral prosthesis valve dehiscence secondary to culture-negative endocarditis.

Keywords: Culture-negative endocarditis, prosthetic valve endocarditis, dehiscence

Introduction

Prosthetic valve endocarditis (PVE) is an uncommon and dangerous consequence of valve replacement that is linked with a high rate of significant morbidity and fatality, as compared to native valve endocarditis (NVE). There are two major problems: determining the diagnosis and curing PVE. PVE diagnosis is difficult, and it frequently necessitates the use of many imaging techniques in addition to routine microbiological tests[1].

Prosthetic valve endocarditis is responsible for 10-30% of infective endocarditis (IE) and is estimated to occur with an annual incidence of 0.3-1.2% and is infrequently compounded by prosthetic valve (PV) dehiscence[2,3]. Additionally, culture-negative endocarditis is thought to account for 2-30% of all IE cases[4].

Here, we describe a patient with severe mitral deficiency and acute heart failure following mechanical mitral prosthesis valve dehiscence secondary to culture-negative endocarditis.

Case Report

A 50-year-old female immunocompetent patient admitted to the emergency department with complaints of palpitation, fever and weakness for one month.

Initial vital signs on admission day were heart rate was 120 beats/min (Figure-1), respiratory rate was 26 breaths/min, oxygen saturation at 85% on room air. Although the patient stated that she had a fever, she did not have a fever on admission day. In her cardiac auscultation, a metallic heart sound and systolic murmur were heard at the cardiac apex. Physical examination revealed no other signs of other endocarditis stigmas (Splinter hemorrhage, Osler nodule, Janeway lesions, etc.). The laboratory examinations at the admission revealed a white blood cell count (WBC): 10700/mm^3 (54.6% neutrophils), a hemoglobin: 12.1 g/dL, and a platelet count: 136,000/mm^3. The inflammatory markers showed a erythrocyte sedimentation rate: 68 mm/h, C-reactive protein: 2.55 mg/dL. Liver function tests, renal function tests and other biochemical test results showed no abnormality. The serum coagulation were in normal

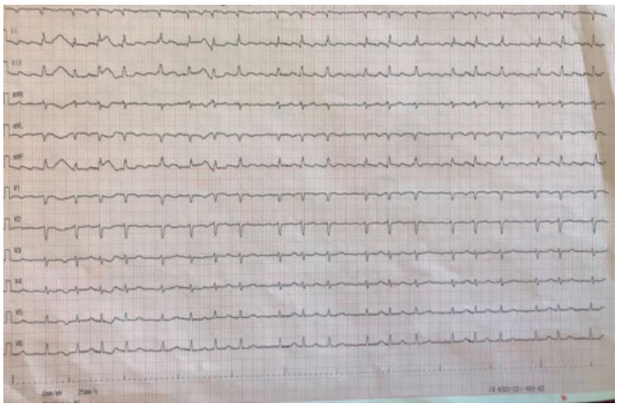

Figure 1.

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ranges, respectively. Posterior-anterior (PA) chest X-ray and urinalysis were normal. Atrial flutter was observed in the patient on admission electrocardiogram. She was hospitalized for further examination and treatment. In the detailed history of the patient, she had a history of mechanical prosthetic valve implantation 25 years ago due to rheumatic mitral valve stenosis. She had no other known comorbidities and chronic drug use. She applied to a medical center and antiarrhythmic therapy was prescribed, and an appointment was made for a control examination. About 10 days later, she was referred to our hospital when there was no improvement in her symptoms.

Firstly, transthoracic echocardiography (TTE) was performed to rule out IE; this examination revealed no suspicious mass-like lesion at the prosthetic mitral valve. But the image of dehiscence was observed in the prosthetic mitral valve. Then advanced imaging methods were planned. Transesophageal echocardiography (TEE) shows severe mitral regurgitation (Figure-2). Three-dimensional echocardiography shows dehiscence of the prosthetic valve (Figure-3). Consultation with an infectious disease's specialist was requested to investigate other etiological causes of the patient's high fever and manage antibiotic treatment, and these recommendations were followed. Three sets blood cultures and serological tests were requested. The treatment was started according to recommendations of 'The Diagnosis, Treatment and Prevention of Infective Endocarditis: Turkish Consensus Report'[5]. Empirically intravenous (i.v) vancomycin IV (30-60 mg/kg, 3 doses/day), gentamicin (3 mg/kg, single dose/day) , cefepime (6 gr, 3 doses/day) and orally rifampicin (900 mg, 3 doses/day) treatments were started. No pathological findings were detected in abdominal ultrasonography.

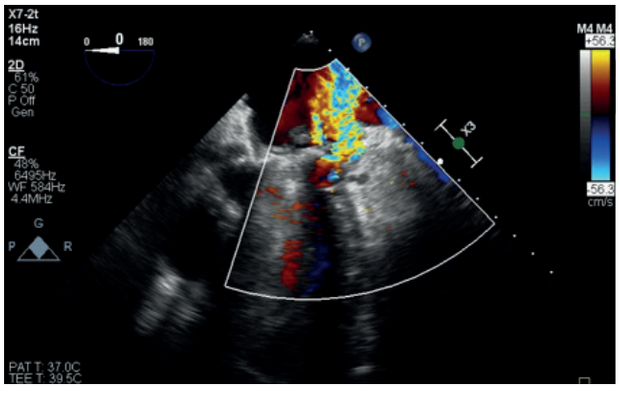

Figure 2.

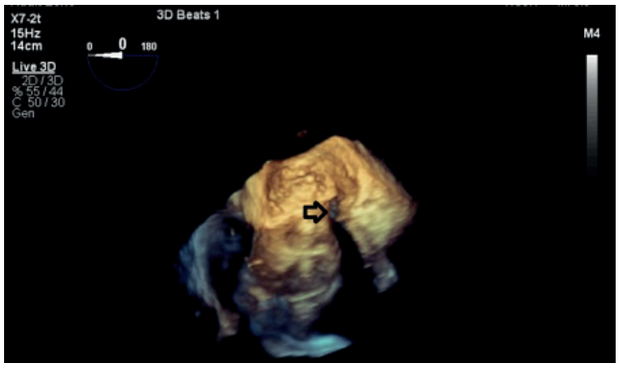

Figure 3.

The results of all blood culture results, and the antibody titers to *Coxiella burneti*, *Chlamydia spp., Mycoplasma spp*. or *Legionella, Brucella* tube agglutination test were negative. *Tropheryma whipplei* and *Bartonella henselae* were found to be negative in blood polymerase chain reactions. Galactomannan antigen test in serum was negative for *Aspergillus spp.*

Emergency cardiac surgery was planned due to the mobile vegetation and severe mitral valve regurgitation.

Cardiovascular surgery consultation was made for emergency surgery. However, in the 5th day of the follow-up cardiogenic shock developed in the patient. The patient was intubated due to the decrease in O2 saturation. The patient was started on inotropic therapy, but despite all interventions, the patient died.

Discussion

Prosthetic valve endocarditis (PVE) is defined as endovascular infection that affects sections of a prosthetic valve or the heart's repaired native valve. It's the most serious kind of IEs, as higher fatality/complication rates and difficulties in the management[6]. The presented case died of sudden onset of heart failure due to mechanical mitral prosthesis valve dehiscence secondary to culture-negative endocarditis.

This illness is divided into two categories based on the time it takes to develop the condition: early PVE and late PVE. The early type of PVE develops within a year following surgery, while the late type of PVE develops beyond a year. The distinction in microbiological profiles between the former and the latter is of clinical consequence[6]. The presented case had a history of mechanical prosthetic valve implantation 25 years ago.

Late PVE is caused by infection acquired in the community, and the pathogens are identical to those seen in native valve endocarditis. Gram positive microorganisms (Streptococci and *Staphylococcus aureus,* followed by coagulase-negative *staphylococci* and *enterococci*) are the most common pathogens. The patients with various comorbidities who are hospitalized to hospitals for other reasons and develop endocarditis have a significant mortality rate in late PVE[6,7]. In our report, the microbiological and serological test results were negative.

Vegetation may not always be seen on prosthetic valve imaging, as in our case. An important part of the etiology of dehiscence valve are endocarditis, degeneration and calcification in the valve suture regions. Both small vegetation and prosthetics with artifacts are the most important reasons for this. Duke criteria used for the diagnosis of native valve endocarditis have low sensitivity for the diagnosis of prosthetic

valve endocarditis. The infection is usually localized at the junction of the prosthetic valve and tissue in the suture ring. It causes severe paravalvular leakage secondary to destruction of tissue[3,8]. If TEE fails to diagnose PVE, cardiac computed tomography angiography (CTA) can be used as a further diagnostic method. As cardiac CT is better than TEE at detecting perivalvular abscess, CTA can be utilized as advanced method in diagnosis for patients who have a negative TTE or could not perform TEE due to contraindications[9,10]. Although TTE and TEE are useful in imaging, three-dimensional echocardiographic imaging should be used in cases with suspected prosthetic valve endocarditis, too[11]. We performed three-dimensional echocardiographic imaging.

Late prosthetic valve dehiscence is rare and mortality rates are high. Good long-term results can be achieved with early diagnosis and timely surgical treatment [2,3,6,8]. The presented did not operated as case sudden cardiogenic shock developed and she died.

Conclusion

As a result, the complications of PVE must be diagnosed early and treated surgically as soon as possible. In this case, three-dimensional TEE is helpful in determining the cause of the severity of PVE. Even if no other symptoms of IE (fever, positive blood cultures, or vegetations) are present, acute valve insufficiency (particularly an eccentric jet) and heart failure should raise suspicion of culture negative endocarditis.

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Case Report

Journal of Emergency Medicine Case Reports

Non-traumatic Spontaneous Spinal Subdural Hematoma in a Patient with Acute Paraplegia

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Abstract

Acute non-traumatic spinal subdural hematoma (SSDH) is a rare clinical condition in the emergency medicine practice and difficult to diagnose during the primary physical examination. It mostly occurs at thoracal vertebra levels due to trauma, use of anticoagulants, medical procedures such as acupuncture, arteriovenous malformations, hematological disorders or space-occupying lesions. Here, we discussed an elderly female patient who was not on anticoagulant and described sudden loss of muscle strength and sensation in both lower extremities. Initial laboratory and imaging including brain computerized tomography (CT) and magnetic resonance imaging (MRI) were in normal range. Patient's secondary examination revealed anesthesia under the T4 dermatome level. Cervical spine-MRI in Spin Eco (SE) T1, Fast Spin Eco (FSE) T2 in sagittal plane; SE T1 weighted in axial and sagittal planes revealed a subdural hematoma significantly compressing the spinal cord on the C7-T1 dermatome segments. Any emergency neurosurgical intervention was not considered and the patient was interned in the neurosurgical clinic for conservative treatment and further examination. Patient, with no progress seen in consecutive MRI scans, was discharged after offering an outpatient check-up. We recommend secondary physical examination in emergency department (ED). In the case of appearance of sensory deficits of certain dermatomes, spine-MRI may lead to put the diagnose early in ED.

Keywords: Non-traumatic, spinal subdural hematoma, spontaneous paraplegia

Introduction

Acute spontaneous spinal subdural hematoma (SSDH) is a rare clinical condition in the emergency medicine practice. In most cases, it is very difficult to diagnose during the primary physical examination. Most of the time no pathology is identified to explain the current clinical condition, repeated physical examinations and suspicion may lead to further investigation and diagnosis. Spinal subdural hematomas (SSDHs) may develop post-traumatically as well as due to interventional procedures such as spinal anesthesia or puncture on the spinal region, acupuncture, use of anticoagulants, arteriovenous malformations, hematological disorders or space-occupying lesions[1]. While SSDHs account for about 4.1% of all spinal hematomas, their spontaneous development is more rarely seen and can lead to severe neurological loss[2]. Early clinical signs vary depending on the level and size of the hematoma. Complaints such as weakness and sensation loss in the extremities and pain can often develop, but also sudden death may occur as a result of cervical upper level compression[3,4,5]. Diagnosis is made by magnetic resonance imaging (MRI).

Here, we discussed the SSDH diagnosis process in an elderly female patient who was not on anticoagulant and described sudden loss of muscle strength and sensation in both lower extremities.

Case Report

A 75-year-old female patient was brought to our emergency department due to neck pain and headache started about four hours ago followed by nausea-vomiting and change in consciousness. Patient had history of hypertension and diabetes, but patient's treatment compliance had been irregular. Patient's vital signs were arterial blood pressure (BP): 165/85 mmHg, temperature: 36.0°C, pulse rate: 85/min, electrocardiography in normal sinus rhythm, and fingertip SpO2: 96%. The neurological examination revealed confusion, partial cooperation, partial orientation, Glasgow Coma Scale (GCS):14 (E:3 V:5 M:6), isochoric pupils with normal light reflexes. She had sensory and motor deficit in both lower extremities. Deep tendon reflexes and plantar reflexes were absent. Complete blood count, prothrombin

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time, activated partial thromboplastin time and International normalized ratio low (INR) as well as biochemical parameters were in normal range. Patient's non-contrast-enhanced and contrast-enhanced computerized tomography (CT) imaging and diffusion MRI results were considered normal. The secondary examination revealed anesthesia under the level from T4 vertebral segment. Further spinal canal examinations to explain the possible causes paraplegia revealed a subdural hematoma significantly compressing the spinal cord between the C7-T1 vertebral segments (Figure 1). The patient was than hospitalized for further investigation and treatment after neurosurgery consultation. A detailed consent statement report that has written by patient is present. 'In the neurological examination performed at the discharge, it was learned that the patient had paraplegia and hypoestehesia starting from the T4 level.'

Discussion

Acute SSDH is a rare condition with unpredictable clinical presentation. Rapidly progressive symptoms due to spinal cord pressure such as sudden onset headache and back pain, sensory-motor change, and autonomous and cardiac dysfunction may develop[6]. Our patient had motor and sensory loss in both lower extremities accompanied by sudden confusion. Known causes usually include trauma, coagulopathy, vascular malformations, spinal inflammatory diseases, spinal space-occupying lesions, systemic comorbidities, psychosomatic disorders and iatrogenic injuries. However, we could not identify any additional pathology except for the history of hypertension and diabetes. The age of cases range between 45 and 60 years, and it was frequently observed in the thoracic part of the spinal canal[2,7,8]. Our 65-years-old patient had hemorrhage at the level of cervical spines. While its pathogenesis is uncertain, SSDH is the mostly recognized subarachnoid hemorrhage (SAH). The subarachnoid space has a dense plexus of capillaries, and rupture of these radiculomedullary vessels as a result of minor trauma caused by increased intra-abdominal and intra-thoracic pressure associated with events such as cough or yawning has been previously accused. Because of the continuation of cerebrospinal fluid (CSF) flow during the early phase of bleeding and dilution of clotting formation by CSF, thrombosis may not be sufficient leading to SDH[6,8,9]. Therefor in SSDH, the duration of initial clinical symptoms may range from several hours to as long as several months[10]. In our patient, the complains had started about four hours before the admission.

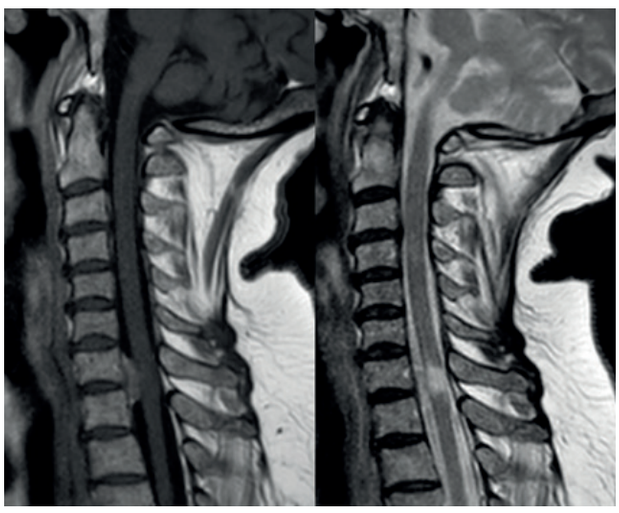

Figure 1. 60th day control cervical MRI sagittal plan T1 and T2 images; on the T1-weighted examination, it was noted that the subdural hematoma caused pressure on the medulla, and that T2 hyperintentisty (myelomalacia) was present in the medulla.

The treatment options include conservative treatments such as intravenous methylprednisolone therapy and percutaneous drainage as well as surgical decompression. There is consensus on conservative treatment in patients with comorbidities and minimal neurological deficits. Our patient was referred to the Neurosurgery for further investigation to determine the etiology of the hematoma and treatment. Patient , with no progress seen in consecutive MRI scans, was discharged after offering an outpatient check-up.

SSDH is a condition which is very difficult to diagnose during the primary examination in cases accompanied by change in consciousness in the emergency medicine practice, and delayed diagnosis may lead to severe neurological loss. We recommend secondary physical examination in ED. In the case of appearance of sensory deficits of certain dermatomes, spine-MRI may lead to put the diagnose early in ED. Repeated MRI guides the way to determine the optimal treatment method for the patient by showing dynamic changes.

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Case Report

Journal of Emergency Medicine Case Reports

A Case of New-Onset Refractory Status Epilepticus (Norse) Due to Herpes Simplex Virus-1 Encephalitis

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Abstract

In the medical literature, the term of "New-Onset Refractory Status Epilepticus" (NORSE) is a novel term. Herpes Simplex Virus (HSV)-1 and other viral infections can be cause to NORSE. Seizures are a rare sign of HSV encephalitis, but they can occur as the first symptom. Herein, we present a case of NORSE triggered by HSV-1 encephalitis, which had been diagnosed via cerebrospinal fluid polymerase chain reaction (PCR) method and magnetic resonance imaging findings.

Keywords: Herpes Simplex Virus, encephalitis, new-onset refractory status epilepticus, seizures

Introduction

New-Onset Refractory Status Epilepticus (NORSE) is defined as the onset of refractory status epilepticus (RSE) abroad a clearly identifiable acute or active toxic, structural, or metabolic causes. NORSE should be considered a rather than a specific diagnosis, the clinical manifestation, according to a recent consensus definition document, and can be applied to a patient with new-onset status epilepticus who has no pre-existing ailment[1]. The patients with new-onset viral pathogenes (e.g., Herpes Simplex Virus-1[HSV-1]) and autoimmune disorders are included in NORSE, even if they are detected within the first 72 hours. In about half of all patients, the probable cause of NORSE can be identified[2]. Confusion, fatigue, headache, recent moderate febrile illness, behavioural changes, and memory problems are all prodromal symptoms that occur in 60 % of individuals prior to the beginning of NORSE[2,3].

Variety of infectious / non-infectious causes may lead inflammation of the brain parenchyma (encephalitis) and this can cause neurological dysfunction. Although viruses constitute most of the infectious causes, the HSV virus is the leading reason of acute encephalitis[4,5].

Herein, we present a case of NORSE triggered by HSV-1 encephalitis, which had been diagnosed via cerebrospinal fluid polymerase chain reaction (PCR) method and magnetic resonance imaging findings.

Case Report

A 46-year-old woman who had hitherto been healthy, was transferred from public hospital neurology clinic to our emergency department (ED). She had complaints of sudden onset of headache, speech difficulty and new-onset refractory seizures in last two days and she had developed epilepsy. Despite one day of antiepileptic treatment, her seizures could not be controlled. She had no notable medical history of seizures such as drug use, toxin exposure, trauma, immunosuppression, chronic illness, and fever. She had no animal contact and no history for zoonoses.

In ED, her vital signs on admission were normal. She was not oriented to time, place, and person. She was lethargic and a mild stiffness in the neck was discovered during a neurological test. Babinski sign was positive. No other suspicious abnormalities were found. There was no sign of an injury anywhere on the body. The rest of her physical examination was showed no abnormality.

Hemogram parameters, routine inflammatory markers (e.g., erythrocyte sedimentation rate and C-reactive protein), blood sugar, renal and hepatic functions, electrolytes were all within normal limits in the admission day. The hepatitis serology and human immunodeficiency virus (HIV) test were negative. The computed tomography (CT) brain scan without contrast revealed no hemorrhage or evidence of an acute ischemia. The magnetic resonance imaging (MRI)

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Table 1: Cerebrospinal fluid findings of the patient.

Variable of CSF	
Color	clear
Intracranial pressure (mmH_2O)	300
WBC ($\times 10^6$/L)	550
LY (%)	70
Protein (g/L)	697
Glucose (mg/dl)	62
Synchronous blood glucose (mg/dl)	115
Brucella Wright test	negative
EZN strain	negative
HSV-1 PCR	Positive (93.000.000 copies/ml)

brain scan was performed for further investigation. There were bilaterally inflammatory T2 signals in the hippocampus, temporal lobe, and insula, as shown on an MRI brain scan (Fig. 1). She was consulted a neurologist in the ED and was diagnosed clinically as NORSE and antiepileptic therapy was recommended.

Further, cerebrospinal fluid (CSF) examination revealed 550 leukocytes/mm^3 and elevated protein levels that indicating a serious risk of encephalitis caused by viruses. No microorganisms were seen in Gram and Ziehl Neelsen stainings from CSF (Table 1).

Abbrevations: WBC: White Blood Cell, EZN: Erlich Ziehl Neelsen EZN, HSV-1 PCR: Herpes Simplex Virus -1 Polymerase chain reaction.

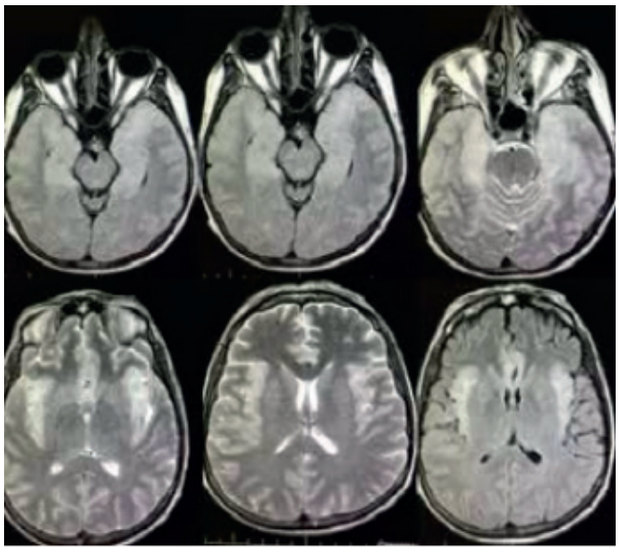

Figure 1. MRI scan of the patient shows bilaterally inflammatory T2 signals in the hippocampus, temporal lobe, and insula.

The patient was hospitalized with a preliminary diagnosis of HSV encephalitis and NORSE. The presence of the HSV-1 pathogen in the CSF with was tested with PCR method, based on the MRI findings and her clinical presentation. HSV-DNA was found to be positive (Table 1). HSV encephalitis (HSE) was confirmed as a result. The patient was subsequently diagnosed with NORSE due to HSV infection based on these findings. The patient received antiviral treatment with 300 mg acyclovir every 8 hours through intravenous (I.V) route, antiepileptic treatment with 300 mg oxcarbazepine, and 500 mg sodium valproate twice a day orally, during his three weeks in the hospital. In addition, the patient was given 10 mg dexamethasone for three days to treat inflammation.

After three weeks of treatment, the patient was in total remission. The patient was placed on continuing orally sodium valproate antiepileptic medication 500 mg twice was given after discharge, and she returned for a follow-up visit four weeks later. No recurrent seizures were occur and she had no neurological sequels.

Discussion

The HSV is a DNA virus, that almost everybody is infected with by the time they reach adulthood. When the virus replicates in the brain, it can induce encephalitis; it's unclear whether this is due to more retrograde axonal transport after reactivation in the trigeminal ganglion, or whether it's due to reactivation of virus that's been latent in the brain. HSE is a disorder that causes both general and localized symptoms of brain dysfunction. It can also be acute or subacute. Brain infection is hypothesized to be caused through direct neuronal transmission of the virus from a peripheral site to the brain via the trigeminal or olfactory nerve, as well as indirect immune-mediated pathways causing neuroinflammation. The true mechanism of HSE is yet unknown. HSE has a bimodal age distribution, with maxima in teenagers and the elderly[4-6]. The presented case was previously healthy 46-year-old woman and with no immunosuppression.

Fever, headache, decreased mental condition, focal neurological abnormalities, and epilepsy are common acute symptoms of HSE6. The patients with new-onset viral infections (including HSV-1) were defined as NORSE, even if they are detected within the first 72 hours[2]. A case of HSE with NORSE is presented in this paper, who was diagnosed within 48 hours. The first signs were a headache, followed by convulsions, but there were no obvious signs of HSV infection, such as a skin lesion. The diagnosis of HSE was confirmed by a HSV PCR test positivity in the CSF examination and typical MRI findings.

In the available literature, the HSE cases with seizures are rarely exist[7,8]. Within 7 days of an acute central nervous system (CNS) infection, 2–67 % of individuals with encephalitis experienced acute symptomatic seizures, according to the International League Against Epilepsy (ILAE). There is a scarcity of trustworthy data from epidemiology studies on the occurrence of seizures in viral encephalitis, the likely underlying processes, and prognosis: this is especially true in resource-constrained settings, where data is restricted to passive monitoring at best[9]. Although there are above 50,000 incidences of CSF infection, encephalitis cases as causes of epilepsy per year in the United States10. Epileptic seizures in approximately 40% of patients with HSV-1 encephalitis, and they may be the first symptom and fever may be mild, and around 11% of HSE patients are afebrile at the time of admission[9]. Associated clinical manifestations include a severe headache, nausea, and vomiting, as well as signs of meningitis (neck stiffness) papilledema[4,6,9]. The presented case had generally seizures for one day as first symptom, that not respond to antiepileptic treatment. Also the case had no fever. She had mild headache and neck stiffness.

HSE is not associated with any pathognomonic clinical symptoms. Initially, focal neurologic abnormalities, CSF pleocytosis, and diagnostic imaging abnormalities may be absent. The preferred imaging study is a brain MRI as diagnostic method and PCR assay of CSF for HSV has largely superseded brain biopsy as the gold standard for diagnosis[10]. The presented case had CSF abnormalities, PCR test positivity and had pathological MRI findings.

Antiviral medication in the IV form of acyclovir is the most common treatment for HSE. Because acyclovir is largely non-toxic and the prognosis of untreated HSE is dire, patients with suspected HSE should be initiated on empirical IV acyclovir therapy as soon as possible until the diagnosis is confirmed[4,10]. Seizures in the HSE cases are treated similarly to patients with other focal epilepsies and frequently necessitating combination treatments[9,11]. The patient was treated with antiviral treatment (acyclovir/IV), antiepileptic treatment (oxcarbazepine and sodium valproate) and anti-inflammatory (dexamethasone) drugs.

All HSE cases should be treated for 14 days of IV acyclovir, followed by a repeat lumbar puncture, according to the United Kingdom guidelines, for immunocompetent individuals. If the CSF PCR tests are still positive despite the treatment, acyclovir should be completed to 21 days, with the LP performed at the conclusion of each cycle to demonstrate viral clearance before ending the medication12. As the presented case did not accept control LP, the antiviral treatment was completed to 21 days.

Conclusion

Finally, in the differential diagnosis of sudden-onset epilepsies, HSE should be examined, and cranial imaging and herpes PCR in CSF should be tested for differential diagnosis.

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

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Case Report

Journal of Emergency Medicine Case Reports

Arterial and Venous Embolism Due to COVID-19 Vaccine (Biontech): Case Series

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Abstract

Although the most prominent effect of Corona disease, respiratory disease it disrupts the vascular structure and causes vascular occlusion. Intravascular coagulation, has also begun to be observed in people who have been vaccinated. Therefore, clinicians had to add anti-coagulants or antiaggregants to the treatment. In this article; We will try to present the patient who developed arterial and venous embolism after BioNTech vaccine. These patients without known vascular disease responded well to treatment. The rate of sickness dropped after the vaccine was discovered to be the most effective strategy to protect against Covid-19. It was discovered that vaccinated patients exhibited symptoms similar to corona disease but had a moderate course.

Keywords: Covid-19, arterial embolism, venous embolism, BioNTech, vaccine

Introduction

A Corona disease is caused by the SARS-COV2 (Severe Acute Respiratory Syndrome causing Coronavirus-2); Initially, it was thought to develop respiratory tract diseases, but over time it was observed that it also led to vascular pathology. Among these, large artery occlusion (1) such as pulmonary artery embolism can be seen, as well as small artery occlusion (2) such as radial artery. Consequently, anticoagulant therapy was added to the Department of Health's guideline on treatment (3).

Although the Covid-19 (Coronavirus Disease Infection-2019) pandemic slowed somewhat with the discovery of the vaccine, the adverse effects of the vaccine were observed to be similar.

A BNT162b2 mRNA vaccine against Covid-19 (Pfizer-BioNTech), one of the most used vaccines in the world in the Covid-19 pandemic, has been in use in Turkey since April 2021 (4). Unlike traditional vaccines, in this type of mRNA vaccine, when the synthetic part of the RNA of the virus is injected into the person, the body forms antibodies faster than other vaccines. Therefore, its effectiveness is very high, but its side effects are less.

In this article, We will try to present two cases with arteria digitalis propria embolism at the middle and distal phalanx level on the 2nd finger of the left hand and femoral vein embolism at left leg that developed after BioNTech vaccine.

The case series has written in an anonymous characteristic, thus secret and detailed data about the patients have removed. Editor and reviewers can know and see these detailed data. These data may back up by editor and by reviewers.

Case Reports

Case-1

A 76-year-old male patient presented to the emergency department with complaints of pain in his left hand and bruising on his second finger. He did not have a history of trauma or COVID-19. He had covid vaccine (Pfizer-BioNTech) 1 day ago, that he developed pain in his left arm at the place where the vaccine was given, and that he noticed numbness and bruising in the 2nd finger of his left hand for 1-2 hours. He had no known chronic disease or history of drug use. He was smoking (25 packs/year). Hypertension (TA: 150/80) was detected in his vital signs. No pathology was detected in the blood tests (including D-Dimer and coagulation tests). EKG was in sinus rhythm. Arterial Doppler ultrasound was requested for cyanosis in his finger (Figure-1).

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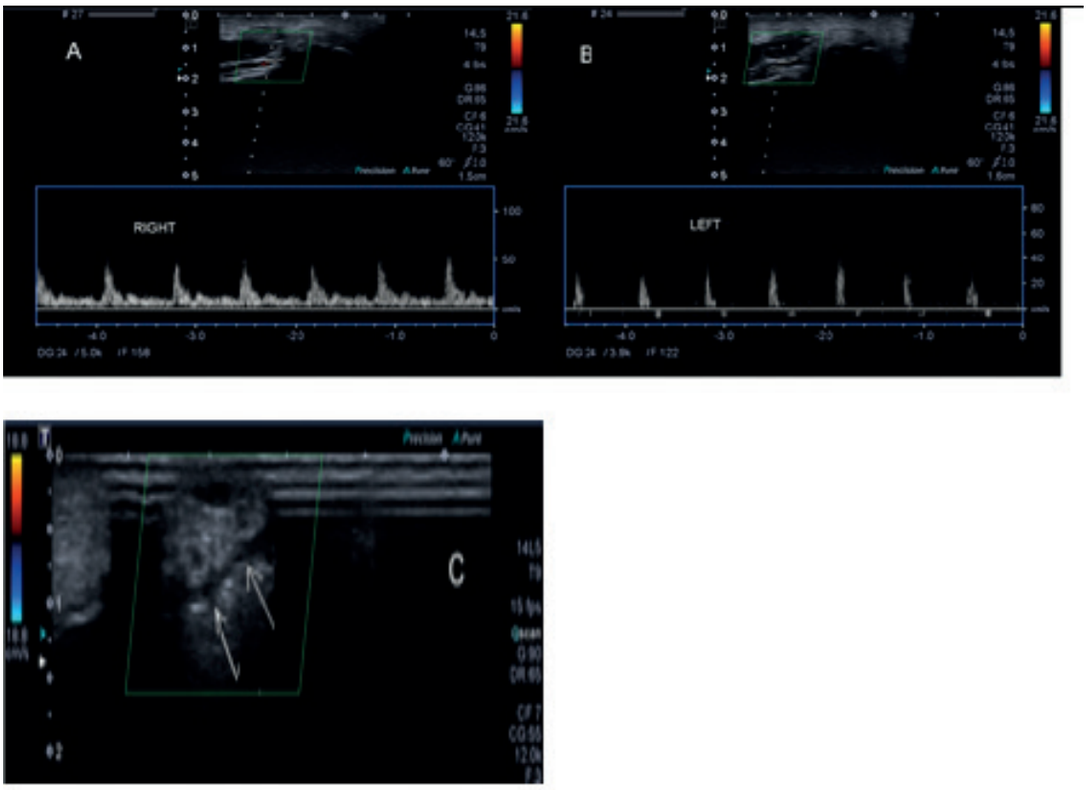

Figure 1. Arterial doppler ultrasound

A. Spectral Doppler waveform in the arteria radialis indicis of the right hand: normal flow pattern
B. Spectral Doppler waveform in the arteria radialis indicis of the left hand: biphasic flow pattern due to resistance
C. No flow was detected in the digitalis propria artery of the left second finger (white arrows).

Contrast-enhanced computed tomography (CT) was requested as an advanced examination (Figure-2).

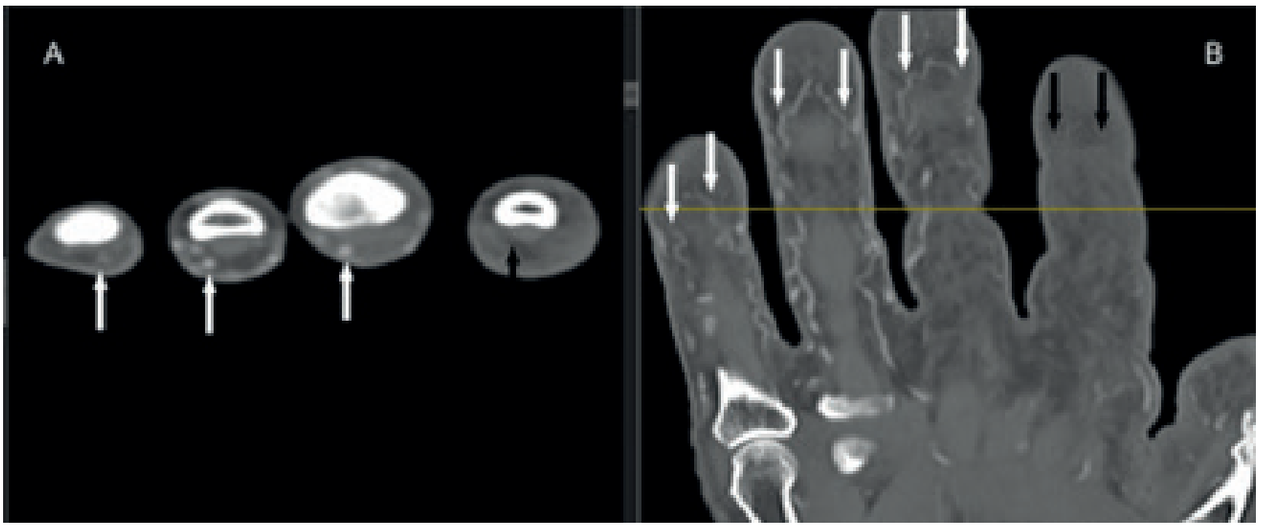

Figure 2. Computed tomography angiography (CTA) of the left hand

A. Contrast uptake was not observed in the second finger in the image obtained in the axial plane (black arrow). No vascular pathology was observed in other fingers (white arrows).
B. In the same case, contrast enhancement was not observed in the second finger in the coronal plane image (black arrow). No vascular pathology was observed in other fingers (white arrows).

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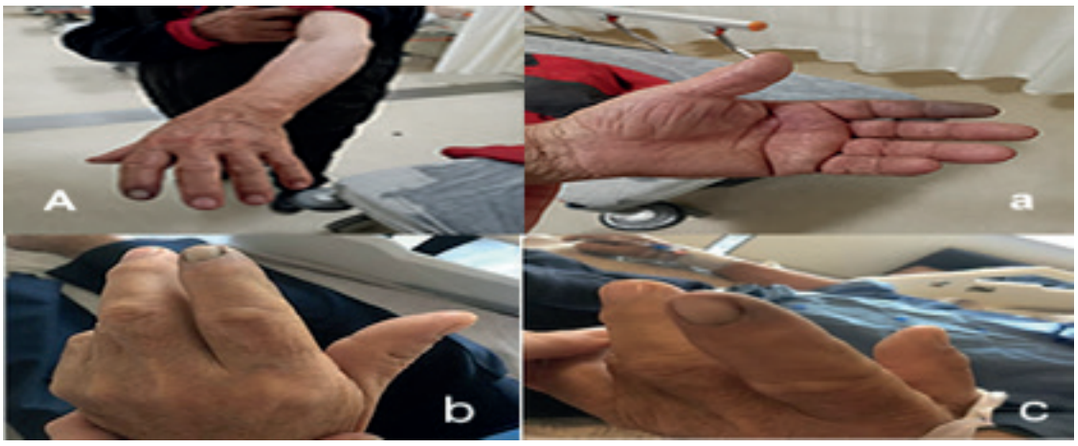

Figure 3. On the 1st day, it was observed that the bruise on the finger was on the mid-distal phalanx (A-a). It was observed that it regressed to the distal phalanx on the 2nd day of the treatment (b) and remained only at the base of the nail on the 5th day (c).

The patient was hospitalized by cardiovascular surgery. Low molecular weight heparin (LMWH) (enoxaparin sodium 12000 IU/dayx2), Ilioprost 1.58 ng/kg/min, Pentoxifylline 200 mg/day, N-acetylcysteine 1200 mg/day, Acetylsalicylic acid 100 mg and analgesic treatment were given. It was observed that the cyanosis, which spread to the middle phalanx at the first application, regressed to the base of the nail on the 5th day (Figure-3). After 5 days of treatment, LMWH and Pentoxifylline, Acetylsalicylic acid, Clopidogrel were prescribed, and he was discharged. He was called follow-up examination 10 days later.

Case-2

A 65-year-old male patient presented with complaints of pain and edema in his left leg. He had hypertension and not smoked for 5 years. He did not have a history of trauma or Covid-19. He had covid vaccine (Pfizer-BioNTech) 5-day ago, that he developed pain in his left leg. In his examination, the left leg was painful on palpation, there was no temperature difference or color change (Figure-4a-b). When compared to the right leg, the edema increased somewhat, but there was no phlegmasia. Hypertension (TA: 170/90) was detected in his vital signs. No pathology was detected in the blood tests (including D-Dimer and coagulation tests). First of all, x-ray was requested, no osseous pathology was detected. The pulses are clean on arterial examination, but a venous doppler test showed subacute DVT (deep vein thrombosis) in the left superficial femoral vein and its distal segment. LMWH 6000 IU/dayx2 and Acetylsalicylic acid 100 mg 1x1 prescribed for 10 days. The pain had subsided and the difference in diameter had reduced 15 days later, according to the follow-up assessment.

Discussion

Corona disease can be asymptomatic or complaints of fever, cough, shortness of breath, headache, joint pain can be seen. Similar symptoms are also observed after vaccination. Cause of hypercoagulability that develops during the disease is presumed to be hyperviscosity secondary to hypoxia. Infection of cells, localized inflammation, endothelial activation, tissue injury, and dysregulated cytokine release are all symptoms of this condition. Coagulation problem and diffuse intravascular coagulation are caused by the septic state, which develops with an increase in leukocytes and

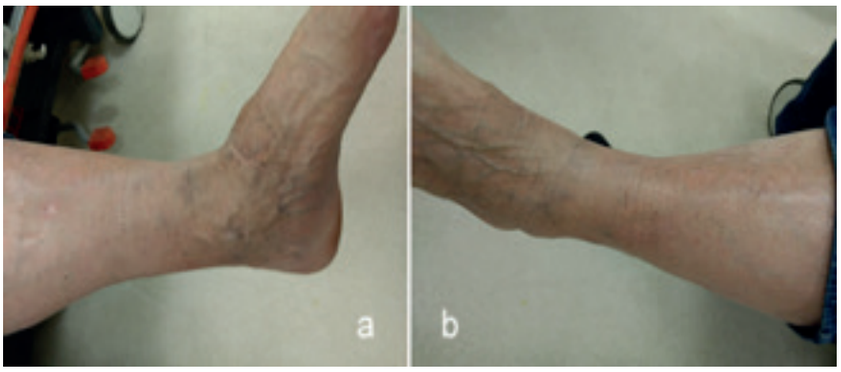

Figure 4. DVT was detected in the patient without edema or color change.

platelets. As a result, multiple organ failures are possible (5). There have been descriptions of the most common cases of pulmonary embolism and cardiac thrombosis.

In the 5-case study of Schultz et al.; After the ChAdOx1 nCoV-19 (AstraZeneca) vaccine, in the histories of the patients who were found to have embolism in the major arteries, it was seen that an average of 1 week passed after the vaccination, and all patients had significant thrombocytopenia. This embolism: they named it as immune thrombotic-thrombocytopenia caused by the antibody response developing due to the platelet factor 4 (PF4)-polyanion complex after vaccination (6). The side effect in our patients; that is a rare finding because the vaccine type is different. It developed within 24 hours after vaccination, all blood tests were normal, and minor arterial embolism was present in first case. In the other patient, Embolism was detected 5 days after vaccination. In comparison to other vaccines, the adverse effects that may arise after the BioNTech vaccine are at a manageable level, according to this study.

In a study by Smadja et al. with data from WHO; The data of 3 vaccines (BioNtech, Moderna, Astra Zeneca) were examined, and it was found that arterial embolism cases were more common (67.9%) in patients who received BioNTech. However, it was emphasized that this result may have occurred because this vaccine was administered to more people and in an older population compared to the other group (7). Since our cases presented with the same post-vaccine arterial and venous embolism, it is compatible with the literature.

In a study conducted; It has been found that people who have had Corona before, experience more side effects after a single dose of BioNTech vaccine (8). In this study, emphasized that it provides a rapid immune response in who with high antibody titers after vaccination. The first patient reported an adverse effect 7 months after receiving two doses of Sinovac vaccination and one dose of BioNTech vaccine. In the second patient, 4 months after receiving the BioNTech vaccination and two doses of Sinovac, findings improved. The reason for this is the high antibody level after previous vaccinations may have been the cause.

During Corona disease and at discharge, if the patient has other conditions, anticoagulant therapy should be added to the treatment (9). If, as in our cases, thrombosis findings are detected in a short time, as in the during the disease, in people who have been vaccinated, or if there is a tendency to thrombosis in the history; maybe after vaccination, prophylactic anticoagulant treatment can be started and followed. We think that this may be possible with a clear identification of the cases. In our cases, we found smoking (or his history) and unmanaged hypertension as factors that may predispose to vascular pathology. With LMWH, we saw a significant improvement in a short period of time. In our cases, even if there is a predisposition to hypercoagulation, we may have received a rapid response to treatment. Because treatment began quickly after the symptoms appeared and peripheral minor embolism was detected, we may have had a speedy response.

The development of the vaccine provided optimism for the end of the pandemic; the epidemic slowed, but identical adverse effects, albeit slightly, were reported following the vaccine (10).

Since BioNTech vaccine is in the new generation m-RNA vaccine group, its side effects are not predictable. However, its side effects should be ignored due to its high effectiveness in the fight against Corona. These side effects should not discourage of getting vaccinated. Our theory is reinforced by the fact that we have found that developing side effects improve with effective treatment.

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Case Report

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Acute Motor and Sensory Axonal Neuropathy (AMSAN) Associated with COVID-19 Infection; A Case Report

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Abstract

Myalgia and headache are relatively common in COVID-19 disease, but a serious neurological disease is uncommon. In this case, we describe the symptoms and clinic of AMSAN, a rare variant of Guillain Barre syndrome (GBS) due to COVID 19. We presented a case of AMSAN, a rare variant of GBS, in a 46-year-old male patient with poor overall condition that did not recover after COVID-19 disease, loss of strength and decreased sensation in distal limbs. electromyography-nerve conduction study findings were suggestive acute motor and sensory axonal neuropathy. Cerebrospinal fluid analysis was elevated protein with a normal white blood cell count. The clinical diagnosis of AMSAN supported by results of diagnostic testing such as cerebrospinal fluid and electromyography-nerve conduction study. We added another GBS case due to Covid-19 infection to the literature. It should always be kept in mind that GBS may develop after the COVID-19 disease.

Keywords: AMSAN, COVID-19, Guillain Barre Syndrome, neuropathy

Introduction

COVID 19 diseases surprised the scientific world with various clinical findings. Its plain findings range from mild viral syndrome with asymptomatic infection, fever, myalgia or cough to severe pneumonia that requires a ventilator, worsens rapidly and leads to early death (1). The spike proteins of the virus determine tissue tropism by using the angiotensin converting enzyme type 2 (ACE-2) receptor to bind to cells. The ACE-2 receptor can be found in nervous system tissue and endothelial cells, between tissues of many other organs (2).

Myalgia and headache are relatively common in COVID-19 disease, but a serious neurological disease is rare. Similar to other coronaviruses, severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS), SARS-CoV-2 have been found to have central and peripheral nervous system manifestations (3, 4). Studies have reported that the most common neurological symptoms among individuals infected with COVID-19 are ischemic stroke, Guillain-Barré Syndrome (GBS), and ICU syndrome-related encephalopathy (5).

The most common variant forms of Guillain-Barré syndrome are acute inflammatory demyelinating polyradiculoneuropathy (AIDP), Acute motor axonal neuropathy (AMAN), Acute motor and sensory axonal neuropathy (AMSAN), Miller Fisher syndrome (MFS), Bickerstaff brainstem encephalitis (BBE). In a meta-analysis, acute AIDP variant, one of the subtypes of Gullian Barre syndrome, was demonstrated to be associated with COVID 19 more frequently in the published cases. However, other subtypes were observed very rarely (6).

In this case, we describe the symptoms and clinic of AMSAN, a rare variant of Guillian Barre due to COVID 19.

Case Report

A 46-year-old male patient presented to the emergency department with complaints of shortness of breath and cough. The patient was hospitalized due to hypoxia and positive SARS-CoV2 PCR test. He had a history of Type 2 diabetes mellitus, hypertension, hypothyroidism and asthma. Dexamethasone 8 mg/day, IL-1 antagonist (Anakinra subcutaneous (100 mg/day)), ceftriaxone 2 gr twice/day, Clarithromycin 500 mg twice/day was given when he was hospitalized due to COVID-19 disease. He was discharged on oxygen therapy. When the patient was discharged, there was loss of strength in his lower extremities, but thought to be caused by dexamethasone

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treatment and malnutrition. He was admitted to the emergency service again because his general condition did not improve and progressive, symmetrical weakness and loss of strength in both lower extremities increased.

On physical examination, the patient did not have fever, and his blood pressure was 110/60 mm/hg, heart rate was 70 beats/minute, respiratory rate was 12/minute, and oxygen saturation in room air was 70%. During the hospitalization of the patient, his consciousness was slow and his orientation and cooperation were poor. In the muscle strength examination, 4/5 in the bilateral upper extremity distal and 3/5 in the bilateral lower extremity distal weakness were observed according to the Medical Research Council (MRC) scale. Deep tendon reflexes were hypoactive. Drop foot was present in both feet bilaterally. There was a decrease in vibration and fine touch sensation in the distal ankle joints.

In laboratory examination;
Laboratory examination results were as follows: serum glucose 188 mg/dL; blood urea nitrogen: 10 mg/dL; creatinine 0.9 mg/dL; alanine aminotransferase 8 IU/L; aspartate aminotransferase 5 IU/L; sodium 138 mmol /L; potassium 3.5 mmol/L; hemoglobin 8.9 g/dL white blood cell count 7420 cells per microliter, erythrocyte sedimentation rate 11 mm/hr, C-reactive protein 52.1 mg/L, procalsitonin 0,2 ug/L, hemoglobin A1c 8.9 g/dL. Negative glucose and ketone were observed in complete urinalysis **(Table 1).** Potassium chloride (40 mEq KCL in 1000 cc isotonic saline) replacement was given to the patient with hypokalemia. Blood and urine culture was performed from the patient with elevated CRP on admission. On the third and fifth days of hospitalization(respectively), the CRP level was 84.5 -111.6, procalcitonin 0.5-0.2. Multidrug resistance klebsiella pneumonia was grown in the blood culture taken at his hospitalization. The patient was started on 150 mg Polymyxin E (Colistin) twice a day and 1 gr Meropenem three times a day. CRP and procalcitonin levels decreased in the follow-ups.

In the follow-ups, the potassium level returned to normal. Since the patient's hemoglobin level was low, vitamin B12 1278 ng/L, folic acid 12 ug/L, percent tranferrin saturation 71, the patient's anemia was evaluated as anemia of chronic disease. The patient's TSH level was 150 mU/ml. The dose of Levothyroxine was increased, and the TSH level decreased in the follow-ups. The respiratory rate was 12/minute and the oxygen saturation was 70% in room air, which may have been due to the patient's severe hypothyroidism.

Cervical and brain magnetic resonance imaging (MRI) was performed. Brain MRI was normal, bulging was present between C2-7. Electromyography (EMG) was performed with the preliminary diagnosis of critical illness myopathy and Gullian Barre. There was right upper ulnar motor and sensory loss. The motor action potential of the right median nerve was extremely low, and the motor action potentials of the tibialis anterior were extremely low. In needle EMG, there were widespread denervation potentials in the right and upper and lower extremities. Findings are consistent with severe motor and sensory axonal neuropathy (AMSAN) in the acute phase (table 2.). In Cerebrospinal fluid white blood cell count was 4, albumin was 52.8mg/dL (normal:0-30), microprotein was 99.68mg/dL (normal:15-40).

Plasmapheresis treatment was started for the patient with the diagnosis of AMSAN. Plasma exchange was done with albumin. Before plasma exchange, the estimated plasma volume (EPV) was calculated from the patient's weight

Table 1: Laboratory values of the AMSAN patient associated with COVID-19

	VALUE	REFERANCE RANGE
COMPLETE BLOOD COUNT		
Total leucocyte count 10^3 uL	7,42	4,5-10
Neutrophils. %	62,3	42-75
Lymphocytes %	19,1	12-48
Haemoglobin. %	8,9	13-17
Haematocrit. %	26,2	40-49
Platelets. 10^3 uL	279	150-450
ELECTROLYTES		
Sodium mmol/L	138	136-145
Potassium mmol/L	3,5	3,5-5,1
Chloride mmol/L	96	98-107
OTHERS		
Glycemia mg/dL	188	70-110
Urea Nitrogen mg/dL	10	6-20
Serum Creatinine mg/dL	0,90	0,70-1,20
AST U/L	5	0-40
Albumin g/L	27,0	35-52
Percent saturation of transferrin	71	15-50
Ferritin ug/L	722	30-400
B12 vitamin ng/L	1278	197-771
Folic Acid ug/L	12	3,8-26,8
D-Dimer ug/L	532	0-500
CRP ug/L	52,1	0-5
Erythrocyte Sedimentation Rate (ESR) mm/h	62	0, -15
CEREBROSPİNAL FLUİD		
CSF Albumin mg/dL	52,8	0-30
CSF Sodium	144	
CSF Chloride	118	
CSF Glucose mg/dL	134	40-70
CSF Microprotein mg/dL	99,6	15-40
CSF WBC 10^3 uL	0,004	
CSF PMN % CSF PMN # 10^3 uL	25 0,001	
CSF RBC 10^6 uL	0	
CSF CULTURE	NEGATİVE	

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Table 2: EMG findings of an AMSAN patient associated COVİD- 19

MOTOR NERVE CONDUCTION STUDIES				
NERVE	LAT	AMP	CV	F-M LAT
MEDIANUS MOTOR RIGHT				
WRIST – APB	2,63 ms	1,65 mV		15,1 ms
ELBOW - WRIST	7,94 ms	0,87 mV	50,8 m/s	
PERONEUS MOTOR RIGHT				
AB. KNEE- FIB. HEAD	5,46 ms	0,026 mV		
TIBIALIS MOTOR RIGHT				
ANKLEE – ABD. HAL	4,58 ms	2,8 mV		51,3 ms
KNEE - ANKLE	15,6 ms	0,23 mV	41,5 m/s	
ULNARIS MOTOR RIGHT				
WRIST - ADM	35,1 ms	-		

SENSORY NERVE CONDUCTION STUDIES			
NERVE	PEAK LAT	AMP	CV
MEDIANUS SENSORY RIGHT			
DIG III-WRIST	3,06 ms	16,6 uV	62,0 m/s
SURALIS SENSORY RIGHT			
MID. LOWER LEG – LAT. MALLEOLUS	2,74 ms	2,7 uV	62,5
ULNARIS SENSORY RIGHT			
DIG V-WRIST	-	-	-

and hematocrit using the formula EPV = $(0.065 \times \text{wt [kg]}) \times (1 - \text{Hct})19$ (average 1-1.3 plasma volume change for each session). After the first day of plasmapheresis, status epilepticus developed in the patient, anticonvulsant treatment (keppra 1000mg twice/day) was started and the patient was intubated. No pathology was detected in the EEG of the patient. Since it was the weekend, EEG could be taken two days later.

The EEG may therefore be normal. The patient was extubated after the 5th session of plasmapheresis. After the 7th session of plasmapheresis. The patient was not hypoxemic before the seizure. The patient was intubated because he could not protect his breathing during the seizure. The patient who was extubated in the 9th session of plasmapheresis was re-intubated from the 10th session. While the first reason for intubation was status epilepticus, the patient was later intubated due to respiratory failure. The patient was extubated 3 days after the third intubation. The patient, whose muscle weakness relatively improved during the follow-ups, was discharged on the fourth day of his extubation (31st day of hospitalization) at the request of himself and his relatives.

Conclusion

In this study, we reported a case diagnosed as AMSAN, a rare variant form of Guillian Barre after COVID-19. We considered that GBS could be a possible diagnosis because of bilateral weakness and relatively symmetrical limb weakness and monophasic course after COVID 19 in our patient. We confirmed our diagnosis as a result of EMG and CSF sampling. COVID-19 often presents with respiratory symptoms, but acute cerebrovascular diseases, seizures, anosmia, meningitis, encephalitis, and skeletal muscle involvement are neurological manifestations (7). Recently, the increase in the number of Guillain Barre cases in COVID 19 disease is noteworthy (8). In the same meta-analysis, 80.5% of the patients had AIDP variant of GBS, and 3 patients had only AMSAN syndrome. The studies on coronaviruses have demonstrated that these viruses have neurotrophic and neuroinvasive properties. It is also unclear whether COVID-19 causes the production of antibodies to specific gangliosides, which usually occurs in certain forms of GBS (9).

GBS is defined as a rare, but potentially fatal, immune-mediated disease of nerve roots that is usually triggered by infections. Diagnosis of GBS in SARS-CoV-2 is particularly difficult because symptoms such as shortness of breath and fatigue can be misinterpreted as secondary to SARS-CoV-2, delaying GBS assessment. Since our patient was hospitalized in the intensive care unit, the symptoms were considered as critical illness myopathy in the first place and it was a late case.

As a result, GBS and its variants should be kept in mind in the presence of symptoms related to peripheral nervous system disorders such as decreased or absent reflexes, elevated and loose paralysis in COVID 19 patients.

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Case Report

Journal of Emergency Medicine Case Reports

Coexistence of Carbon Monoxide Intoxication and COVID-19

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Abstract

This case report aimed to report two patients admitted to the emergency department with a preliminary diagnosis of carbon monoxide intoxication and was diagnosed with COVID-19 during their follow-up. A 73-year-old female patient presented with weakness and shortness of breath complaints to the emergency department. Carboxyhemoglobin (COhgb) reached 36.2 %. Atypical pneumonic infiltration with peripheral and central patchy consolidations in the zones in hemothorax images from computerized tomography. The patient was tested positive for COVID-19 after a PCR test. The COhgb values of the patient reached 16 % after 3 h. Then, it dropped to 3.0 % after 8 h. A 77-year-old male patient presented with shortness of breath and nausea complaints to the emergency department. COhgb (carboxyhemoglobin) reached 30%. Emphysematous changes in the lung parenchyma and increased peribronchial densities in the lobes were shown in computed tomography. The patient was tested positive for COVID-19 after a PCR test. The COhgb values of the patient reached 13 after 3 h. It dropped to 2.4 after 8 h. Carbon monoxide intoxication is a significant public health problem with a high probability of death. Detailed studies and meta-analyses are needed to affect the prognosis of COVID-19 disease.

Keywords: Covid-19 carboxihemoglobin, carbon monoxide diffusion capacity

Introduction

It has been reported that the diffusion capacity of carbon monoxide, a colorless and odorless gas, may increase in patients diagnosed with COVID-19, which has a grave prognosis as in chronic lung diseases [1].

Carbon monoxide is a colorless, odorless gas that has the capacity to separate oxygen from hemoglobin and accordingly reduces the oxygen carrying capacity of the blood [2]. Carbon monoxide is produced by hemooxygenases, an enzyme that can be stimulated by biochemical stress. Hemooxygenase enzyme is most commonly found in the spleen and bone marrow, but it is also found in the liver, kidney and lung. An increase in exhaled carbon monoxide has been observed in inflammatory lung diseases [3]. In addition, there is not enough data that carbon monoxide intoxications cause a predisposition to chronic or acute lung diseases. In studies, the carboxyhemoglobin level is not even above 5%, including in patients who need mechanical ventilation for a long time [4-6].

Studies investigating carbon monoxide levels with Covid-19 are generally retrospective and it was not possible to obtain information from patients in terms of carbon monoxide exposure. [5]. Carbon monoxide intoxications are not generally questioned and many diagnoses are made instead of carbon monoxide intoxications [7]. Most of the time, in the diagnosis of obstructive diseases, although the use of domestic fuel is known in the etiology, carbon monoxide exposure is ignored.

This case report aimed to report two patients admitted to the emergency department with a preliminary diagnosis of carbon monoxide intoxication and was diagnosed with COVID-19 during their follow-up.

Case Reports

Case-1

A 73-year-old female patient presented with weakness and shortness of breath complaints to the emergency department. The general condition of the patient was moderate, cooperative, and oriented. The patient had a fever of 36.5°, a heart rate of 70/min, a BP of 140/90mmHg, a respiratory rate of 22/minute, and a saturation of 95%. The patient had known diseases of hypertension and diabetes mellitus. The patient stated that she had complained of weakness and

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nausea for a few days. Routine examinations were conducted. The following results were observed for the routine exams: pH reached 7.4, PCO_2 reached 40 mmHg, HCO_3 reached 24, carboxyhemoglobin (COhgb) reached 36.2 %, lactate reached 2.4 mmol/L, white blood cells (WBC) reached 7.59 K/µL, hemoglobin(hgb) reached 14.4 g/dl, platelet(plt) reached 222 K/µL, urea reached 24.4 mg/dl, creatinine reached 0.76 mg/dl, aspartate aminotransferase (AST) reached 23 U/L, alanine aminotransferase (ALT) reached 17 U/L, sodium reached 138 mmol/L, potassium reached 4.3 mmol/L, and troponin reached 18.03 ng/L (normal range is 2.5–100 ng/L). Atypical pneumonic infiltration with peripheral and central patchy consolidations in the upper, middle, and lower zones in hemothorax images from computerized tomography. (Figure 1).

Therefore, the patient was diagnosed with COVID-19 was tested positive for COVID-19 after a PCR test. The COhgb values of the patient increased to 16 % after 3 h. Then, it dropped to 3.0 % after 8 h.

Case-2

A 77-year-old male patient presented with shortness of breath and nausea complaints to the emergency department. His general condition was good. The patient had a GCS (Glasgow coma scale) of 15 and had a fever of 36°, the heart rate was 80/min, BP (blood plessure) was 125/78 mmHg, respiratory rate was 17/min, and saturation was 97%. The patient, who did not have any disease in his history, complained of weakness for a few days. In routine examinations, pH reached 7.42, PCO_2 reached 35 mmHg, HCO_3 reached 22.6 mEq/L, COhgb reached 30%, lactate reached 1.87 mmol/L, WBC was 9.08 K/µL, hgb reached 14.5 g/dl, plt reached 145 K/ µL, urea reached 49.5 mg/dl, creatinine reached 1.03 mg/dl, AST reached 33 U/L, ALT reached 19 U/L, sodium reached 139.4 mmol/L, potassium reached 4.12 mmol/ L, troponin reached 31.53 ng/L (normal range 2.5–100 ng/L). Emphysematous changes in the lung parenchyma and increased peribronchial densities in the bilateral lower lobes were shown in computed tomography (Figure 2).

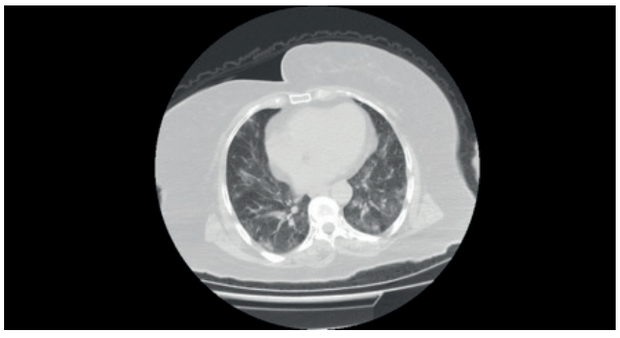

Figure 1. CT image of atypical pneumonic infiltration of our female patient.

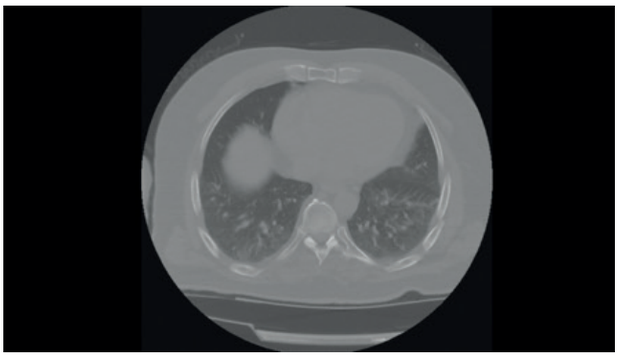

Figure 2. CT image of emphysematous changes in the lung parenchyma of our male patient.

The patient tested positive for COVID-19 after a PCR test. The COhgb values of the patient reached 13 after 3 h. It dropped to 2.4 after 8 h. Both patients also said they were affected by stove smoke at their house for 2 h before they reached the hospital. Both patients were given intravenous fluids and oxygen with a reservoir mask (10lt/min). Hyperbaric oxygen therapy was recommended due to high COhgb, but both patients did not accept it. Both patients, who did not develop chest pain, clouding of consciousness, or relieving shortness of breath during their follow-up, were discharged with recommendations after 48 h of follow-up.

Discussion

Carbon monoxide poisoning is more common in Turkey in winter and indoors. It has been shown in studies that carbon monoxide poisoning is more common in females. This is because women spend more time indoors [7]. Although there was exposure to carbon monoxide simultaneously and in the same place, in our case report, the carboxyhemoglobin level was higher in our female patient. Fatigue, shortness of breath, and nausea complaints can be observed in carbon monoxide intoxication and during COVID-19 disease. Carboxyhemoglobin levels increased by up to 30 after domestic stove use and 2 h of exposure. It was observed in a prospectively planned study that the carboxyhemoglobin level was lower in the mortal group of patients diagnosed with COVID-19 [5]. Ledoux et al. found no statistically significant difference in carboxyhemoglobin levels between mortal and non-mortal groups in a study conducted with intensive care patients [6]. An increase in carboxyhemoglobin levels was observed in 26.7% of 431 patients diagnosed with COVID-19 in a retrospectively planned study. There was a statistically significant correlation between high carboxyhemoglobin levels and intubation and mechanical ventilation [8]. In the 30-day follow-up of our patients, their diseases did not progress, and they did not have symptoms that required hospital admission. The elevation in the carboxyhemoglobin level did not affect the COVID-19

prognosis. Faisal et al. reported methemoglobinemia in a patient followed up in the intensive care unit. Although the patient stayed in the intensive care unit for a long time, the methemoglobin level returned to normal during the recovery period [9].

As far as we can detect, there are no reported cases or articles associating carbon monoxide intoxication and COVID-19 positivity in the literature. COVID-19 can affect the diffusion capacity of carbon monoxide, but in limited studies, no statistical relationship was found between mortality and carboxyhemoglobin levels. However, carboxyhemoglobin levels were relatively low in these studies [5,6].

Conclusion

Carbon monoxide intoxication is a significant public health problem with a high probability of death. There are not enough studies on the possibility of predisposing to infection or the fact that the transmission will be even more with carbon monoxide exposure. Our aim was to state that there is a need for prospective studies on carbon monoxide intoxications. Detailed studies and meta-analyses are needed to affect the prognosis of COVID-19 disease.

The case report has written in an anonymous characteristic, thus secret and detailed data about the patient has removed. Editor and reviewers can know and see these detailed data. These data are backed up by editor and by reviewers.

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Case Report

Journal of Emergency Medicine Case Reports

Isolated Dorsal Dislocation of the 5th Carpometacarpal Joint; A Missed Injury

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Abstract

5th carpometacarpal (CMC) joint dislocations of the wrist region are very rare and easily overlooked injuries. In this type of injury, which causes serious functional problems in the hand if not treated appropriately, closed methods are generally used in the treatment. In this study, we present a case with fracture-dislocation of the 5th CMC joint. Fracture-dislocation of the 5th CMC joint of a 25-year-old patient who was admitted to the emergency department due to wrist pain was not noticed. The diagnosis was made during the follow-up of the patient, and the joint was reduced with closed method and fixed with a K wire. No serious problems were encountered in the patient's controls. Undiagnosed 5th CMC joint dislocation disrupts the transverse and longitudinal arches of the hand, so the patient has a weak and painful gripping function. Radiological findings may be unclear on anterior-posterior and lateral views, and additional images may be obtained if in doubt.

Keywords: Carpometacarpal dislocation, missed injury, fracture, closed reduction

Introduction

CMC joint dislocations constitute less than 1% of all carpal region injuries. Isolated dislocations of the fifth carpometacarpal - hamatometacarpal (HMC) joint are even rarer[1]. The most common mechanism of this type of injury is blunt trauma to the ulnar side of the hand. Fractures and dislocations of the fifth CMC joint are more common than dislocations alone, and dorsal dislocations occur more frequently than volar dislocations[2]. Insufficient care of the physician in clinical examination and radiographic examinations may cause such injuries to be easily overlooked, especially in clinics with a large patient population, and this may lead to long-term pain and poor grip strength in the hand[2,3]. While stable HMC joint dislocations can be easily treated with reduction and casting, stabilizing the 5th CMC joint with various methods such as K wire in unstable injuries prevents long-term complications [4]. This case report aims to increase awareness regarding 5th CMC dislocations.

Case Report

A 25- years- old girl patient applied to the emergency service because of right wrist pain as a result of a motorcycle accident. On physical examination, there was swelling and tenderness in the wrist, especially in the lateral region, and no neurovascular injury. The patient was told that there was no problem with the radiographs (Figure 1) and a follow-up visit was required one week later, making a short arm splint with the anti-inflammatory drug and recommendations. The patient came to the follow-up examination one month later and stated that she had removed her splint voluntarily one week after the event. In the radiographs of the patient (Figure 2) whose pain continued, there was a fracture-dislocation of the right-hand 5th CMC joint. The patient who did not want additional examination due to the fear of magnetic resonance imaging was recommended to intervene under anesthesia in terms of reduction because of a long time elapsed. Closed reduction was attempted under anesthesia but due to the instability observed, the 5th metacarp was reduced to the hamatum with one K wire (Figure 3 A, B). When the patient came to the follow-up after 4 weeks, the K wire was removed and the splint was terminated and the exercise was started. In the 3rd month, joint movements and muscle strength were observed to be complete. At the 10th month follow-up, such as postoperative radiographs it was observed that the 5th CMC joint was in the proper position in the hand radiography (Figure 3 C, D). A written informed consent was obtained from the patient for the publication of this study.

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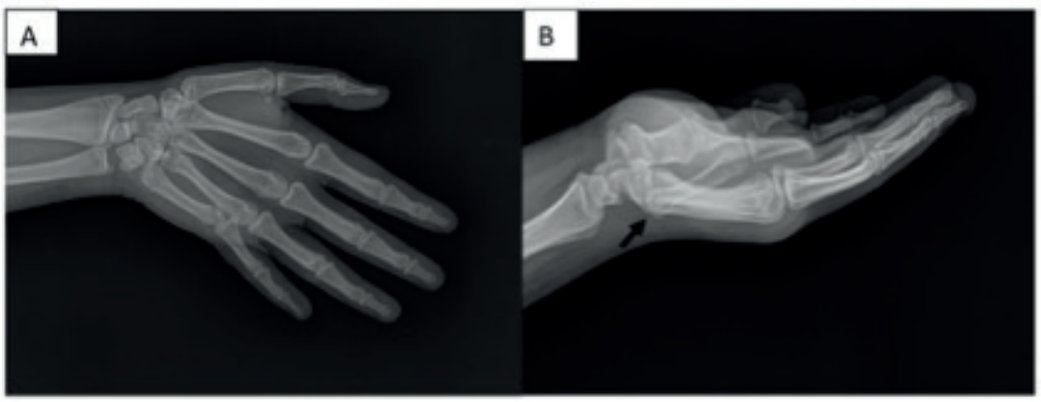

Figure 1. A) AP and B) lateral radiographs of the first arrival at the hospital, arrow; undiagnosed 5th CMC joint dislocation.

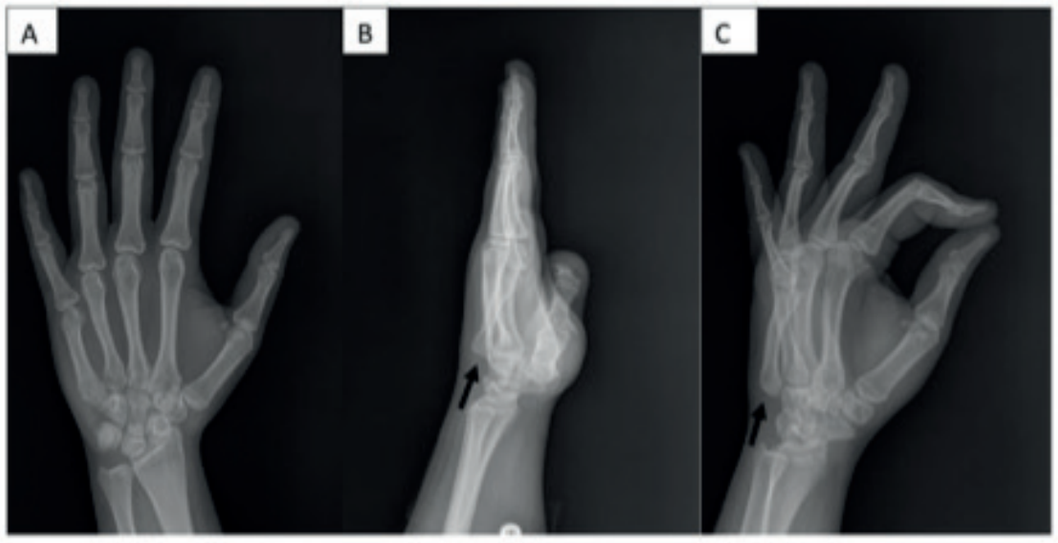

Figure 2. A) AP, B) lateral, and C) oblique radiographs of the second arrival at the hospital, arrow; fracture-dislocation of the 5th CMC joint.

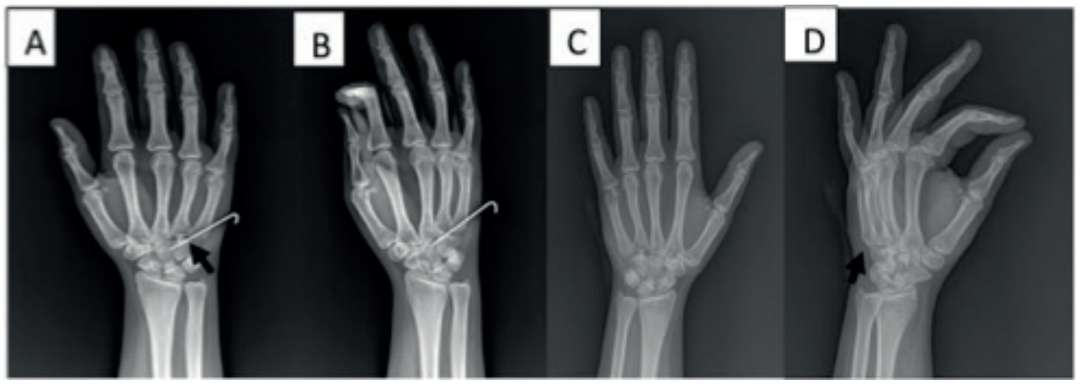

Figure 3. A) AP, B) lateral, and C) oblique radiographs of the second arrival at the hospital, arrow; fracture-dislocation of the 5th CMC joint.

Discussion

Dislocation of the 5th CMC joint, which can easily be overlooked in trauma patients, is seen rarely[5,6]. These types of injuries are usually the result of severe events such as traffic accidents and mostly concern the young age group. It was stated that 7% of dislocations in this region were missed in the first application of patients to the emergency department, and 25% of those that were overlooked were realized by senior registrars and orthopedic consultants.

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Only one-third of the patients were correctly diagnosed at the time of the first injury[7]. In another study, decreased joint movements and weakened grip strength were observed in all patients with delayed diagnosis, and CMC arthrodesis was applied to approximately half of these cases[8].

A remarkable feature of the[5]. CMC joint that distinguishes it from other CMC joints is that the radioulnar inclination in the frontal plane allows 10-20° supination, as seen when the little finger is opposing the thumb. This additional mobility causes the instability of the 5th CMC joint to increase and dislocations to be seen more in this region compared to other CMC joints[9]. The HMC joint has strong interosseous, dorsal, and volar ligaments. Although the dorsal ligaments are stronger, dorsal dislocations occur more frequently. However, the most prominent structure for stability is the intermetacarpal ligament between the 4th and 5th metacarpals. It was showed that even if other attachments of this region were damaged, keeping this ligament intact prevented subluxation or dislocation of the 5th CMC joint[10].

Physical examination and radiological evaluation are important in the diagnosis of CMC joint dislocations. Dynamic muscles (extensor-flexor carpi ulnaris, hypothenar muscles) adhered to the base of the 5th metacarp with the deterioration of the articular surface pull the bone structure proximally and overlap the hamate, causing the little finger to appear proximally on physical examination[5]. AP images reveal loss of parallelism between CMC joints and overlapping of joint surfaces[2]. An appropriate lateral radiograph is critical, especially to identify occult CMC joint subluxations, and should be carefully evaluated due to overlapping bony structures. Oblique radiographs are particularly stimulating to evaluate the ulnar region CMC joints[3,6]. However, it may not be sufficient to define the features of fractures and to see occult subluxations with radiographs alone. Ultrasonography is an auxiliary method in evaluations and it should be considered that it provides dynamic research without radiation, its reproducibility is low and it has factors such as dependence on the person applying[3]. Therefore, computed tomography is the best diagnostic tool that makes the diagnosis definitively and clearly shows the relationship of the articular surfaces with each other[2].

This type of injury can be easily reduced by physical manipulation for up to ten days. In unstable reductions, fixation can be achieved with a K wire[7]. It can be healed by closed reduction and well molding in plaster without the need for internal fixation[3,4,6]. Also the reduction loss can be seen in the first two weeks after physical manipulation[6]. Therefore, close follow-up with postoperative radiographs is important. This complication can be avoided with a K wire sent in one go after the reduction of the dislocated joint. In cases encountered after 3 weeks, open reduction may be required[2,3]. The deep motor branch of the ulnar nerve runs on the palmar surface of the 5th CMC joint and runs around the hook of the hamate. Therefore, the neurological evaluation of the hand should not be missed in the physical examination[5].

Conclusion

CMC joint injuries are rare and can be overlooked. Undiagnosed 5th CMC joint dislocation disrupts the transverse and longitudinal arches of the hand, so the patient has a weak and painful gripping function. Radiological findings may be unclear on anterior-posterior and lateral views, and additional images may be obtained if in doubt. An unstable HMC joint dislocation is generally treated by closed or open reduction and internal fixation using a K wire.

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