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Case Report

Journal of Emergency Medicine Case Reports

Brain Abscess Developing After Auto-Mastoidectomy: A Case Report

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Abstract

Auto mastoidectomy is a rare complication that can develop after a chronic middle ear infection and can be seen in cases where no response to classical infection treatment is obtained. The destruction of bones and tissues may cause the current infection in the brain.A 55-year-old male patient applied to the emergency department for a left ear pain complaint. In his examination, he was conscious, but orientation and cooperation were not complete. The brain CT showed a mastoidectomy cavity on the left, but he had never been operated on. The destruction of bones and tissues may cause the current condition in the brain. This case report examines a case of auto mastoidectomy due to cholesteatoma, which can be diagnosed when the brain abscess develops.

Keywords: Brain Abscess, Automastoidectomy, Chronic Otitis Media

Introduction

Auto mastoidectomy is a rare complication of mastoid destruction due to infection or mass, observed in people without a history of surgery. Damaged mastoid tissue can predispose to another infection or progress to the eardrum and ear bones, leading to hearing loss. Cases of auto mastoidectomy caused by cholesteatoma have been reported most frequently in the literature (1). Cholesteatoma is a cystic lesion of keratinised stratified squamous epithelium (2). In this case report, while studying the aetiology of brain abscess, one case with otitis media due to cholesteatoma and determined auto mastoidectomy will be examined.

Case Report

A 55-year-old male patient applied to the emergency department complaining of left ear pain that had been present for three weeks and spreading to the left side of his head for a few days. Antibiotics and analgesics had prescribed two weeks ago with the diagnosis of otitis media, but he hasn't used his medications regularly. He has a history of hypertension (HT), diabetes (DM) and chronic obstructive pulmonary disease (COPD). There was no history of trauma or surgery. His fever was 39ºC, and his blood pressure was 120/80 mm/Hg. In his examination, he was conscious, orientation and cooperation were incomplete, nuchal rigidity was positive, kernig and brudgenzy were negative, bilateral muscle strength was good function, left eardrum was edematous, and there was a slight discharge in the external auditory canal. There were no acute pathologies on the other system exams. Leukocytes 22,000 109/L, neutrophils 84%, and C-RP (C-reactive protein) 9.34 mg/L were detected for blood tests. The PCR (SARS-CoV-2) requested was negative, and no viral pneumonia was found in chest computer tomography (CT).

For the preliminary diagnosis of intracranial disease, brain CT and magnetic resonance (MRI) examinations were performed. A mastoidectomy cavity was observed on the left. Soft tissue densities were observed in the left mastoidectomy cavity and middle ear, and a signal change showing diffusion restriction in the medial part of the left temporal lobe was observed (acute infarct? Abscess?). The nodular diffusion restriction area was marked in the left temporal lobe inferior part (Figure 1). He was referred to the academic hospital with preliminary diagnoses of abscess, meningitis, encephalitis, and mass.

It was learned that the patient was thought to have a brain abscess in the examinations performed at the hospital. He was hospitalised and operated on by otolaryngologists and neurosurgeons (Figure 2).

During the operation, it was reported that the incus and stapes were destroyed, and the malleus was partially preserved. Tympanoplasty was not performed due to suppurative discharge. Proteus Mirabilis was grown in

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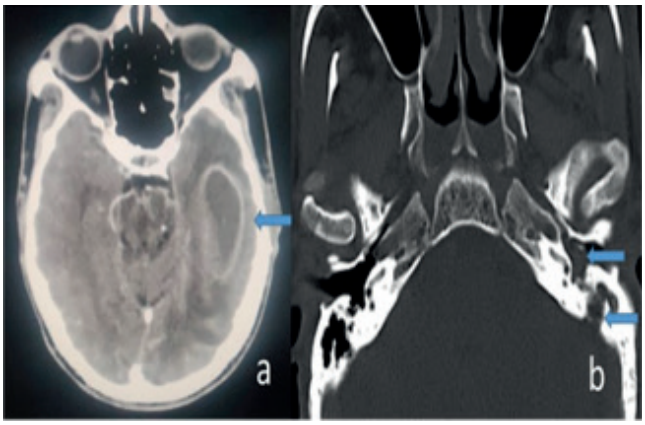

Figure 1a-b. Due to chronic auto mastoiditis complicated by the formation of an intracranial abscess (a) and bone destruction of the posteromedial wall of the mastoid of the temporal bone (b) are represented by arrows.

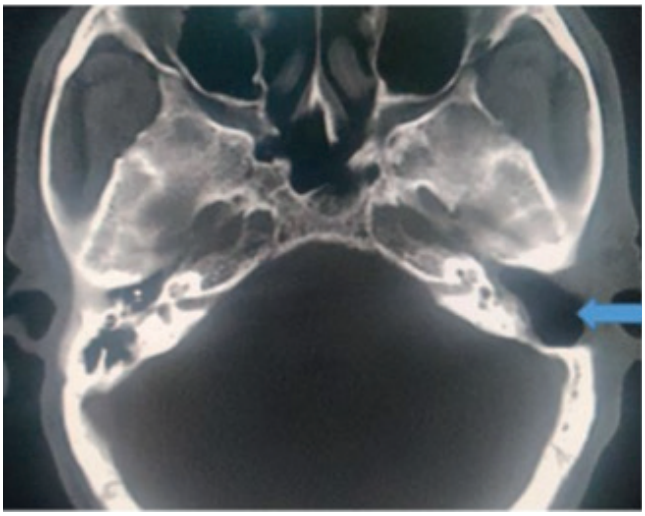

Figure 2. The patient's post-operative mastoidectomy cavity (arrow).

aspiration from the inner ear and brain tissue. Antibiotic therapy was administered. Complete hearing loss was observed before and after surgery. The patient, who was hospitalised for 30 days, had no neurological sequelae except slowing down in speech. He was discharged with the recommendation of Neurosurgery and Otorhinolaryngology outpatient control.

Discussion

In the literature, there is not enough information about auto mastoidectomy cases. Otorrhea, hearing problems, ear pain, and facial paralysis are the most common reasons for admission. There are patients with these complaints for an average of 20 years. When facial paralysis develops, the diagnosis is made in a shorter time (1). The factor that causes this situation may be that patients consider it normal when their complaints do not go away. Still, when facial paralysis develops, it is noticed by the patient's relatives, and the treatment is directed. In the case presented in this article, some complaints were probably longer but increased to 15 days. It is thought that the diagnosis was made more quickly when external signs such as impaired consciousness appeared (possibly after the development of an abscess).

The progression of the epithelial layer in the outer ear canal to the inner ear is responsible for the development of cholesteatoma. The epithelial layer that covers the ear canal and the membrane's outer surface will continue to produce keratin if it enters the middle ear cavity. This substance, utterly foreign to the middle ear, provokes a severe reaction. A cystic lesion is formed by creating a predisposing ground for infection, traumatising the surrounding tissues and may cause the development of chronic otitis media (3). Cholesteatoma is a common cause of chronic otitis media. In a study by Sun et al., intracranial complications due to chronic otitis media were examined, and it was found that complications developed in patients with cholesteatoma at the highest rate. P. Mirabilis was the most common factor among these patients (4). The same factor was detected in the case of this study.

In a study in which CT scans of auto mastoidectomy and post-mastoidectomy cases were examined, patients of auto mastoidectomy, defects of soft tissue and bones in the external auditory canal were observed together in cases of post-mastoidectomy. It was determined that there was a posterior and lateral wall defect in the external auditory canal, and there was no Henle's spine (5). In the case of this study, the presence of soft tissue and destruction of the posteromedial wall of the mastoid bone was detected. The patient's lack of an operating history also supports the diagnosis of auto mastoidectomy. The difference became evident with the tissue destruction that developed post-op. In patients with impaired consciousness or insufficient information with this information, it can be decided whether further examination will be performed, the type of treatment and a precise diagnosis.

Compatible with the literature, in this case, chronic infection progressed to the intracranial tissue and intracranial complication due to developed otitis media. This suggests that cases thought to develop otitis due to cholesteatoma should be promptly treated and followed up. In addition, the clinician should warn the patient about possible complications.

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Case Report

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Diagnosis of Multisystem Inflammatory Syndrome in Child (MIS-C) Case Presenting with Acute Dystonia Secondary to Use of Metoclopramide

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Abstract

Metoclopramide is a dopamine antagonist in the central nervous system and an antiemetic agent. It can cause extrapyramidal symptoms side effects such as dystonic reactions characterized by involuntary, sustained or spasmodic contractions of muscle groups, resulting in twisting, repetitive or abnormal postures. In this study, we aimed to report a pediatric patient who presented with acute dystonia due to metoclopramide use and was finally diagnosed with MIS-C.

Keywords: Acute Dystonia, Metoclopramide, Multisystem Inflammatory Syndrome in Child.

Introduction

Metoclopramide is a dopamine antagonist in the central nervous system and an antiemetic agent. Although it is recommended for adults, it is used in gastroesophageal reflux disease and acute gastroenteritis in pediatric population. It can cause extrapyramidal symptoms side effects such as dystonic reactions characterized by involuntary, sustained or spasmodic contractions of muscle groups, resulting in twisting, repetitive or abnormal postures. The incidence of acute dystonias is approximately 0.2%. It has been reported that the incidence of acute dystonias increases up to 25% with increasing dose and decreasing age. (1-3)

A growing number of reports from different countries have now described a severe inflammatory syndrome named Multisystem Inflammatory Syndrome in Children (MIS-C) with acute COVID-19 infection. To date, case series of MIS-C have described multisystem organ involvement including the gastrointestinal, mucocutaneous, respiratory and cardiac systems.(4-6) In children, COVID-19 has usually mild prognosis. Furthermore, the disease course in MIS-C can be quite severe, with many children requiring intensive care interventions. Rapid diagnosis and appropriate treatment of MIS-C are important for prognosis. The vast majority of children survive, but deaths have been reported. (5) In this study, we aimed to report a pediatric patient who presented with acute dystonia due to metoclopramide use and was finally diagnosed with MIS-C.

Case Report

A seven-year-old previously healthy girl was admitted to the pediatric emergency department with complaints of involuntary spasm of the neck, eyes, head, upper extremities and sleepiness for five hours. It was learned that two days ago, fever and nausea started with intermittent vomiting. Due to nausea and vomiting, her family gave oral metoclopramide to her 5 mg four times, one day ago. A few hours after administration of last dose of metoclopramide, she complained of involuntary spasm.

In her past, she was close contact with her father who had COVID-19 a month ago. On physical examination, she had body temperature of 36.8oC, hearth rate 75 beats/minutes, blood pressure 80/55 mmHg, respiratory rate 18/min and 97% oxygen saturation on room air. She was awake but agitated, avoided eye contact and not answered questions. The patient's head and eyes were deviated to the left, and muscle spasm was present in the left sternocleidomastoid and upper extremities. She was able to answer all the questions but had dysarthria. Both pupils were isochoric. Meningeal irritation signs were absent and deep tendon reflexes were normal. She had bilateral conjunctival

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hyperemia. The remainder of her physical examination was unremarkable. Initial laboratory tests showed blood glucose 107 mg/dL, hemoglobin of 12.3 g/dL, thrombocytopenia (82000 platelets/µl), 11050 leukocytes/µl but with only 800 lymphocytes/µl, hyponatremia (127 mmol/L), d-dimer 1.32 µg/mL (Positive; 0.50 µg/mL or greater). In addition, the C-reactive protein (CRP) level 39.5 mg/L (Positive; 5 mg/L or greater) and procalcitonin 17.7 µg/L (Positive; 0.5 µg/L or greater) were significantly high. Renal and liver function test results were within normal values and urinalysis was normal.

We considered that acute dystonic reaction originated from metoclopramide according to her history and physical examination findings. Therefore, biperiden was performed intramuscularly and then dystonic reaction disappeared dramatically within 30 minutes. Dystonia was not observed again, but 8 hours after biperiden injection, she developed headache and altered mental status with agitation, hallucination and confusion. Blood pressure, body temperature and saturation values were within normal ranges. Her deep tendon reflexes were normal with negative Babinski reflex bilaterally. No papilledema was noted. The remainder of physical examination was unremarkable.

Blood glucose level was within normal range. Chest radiograph, computed tomography and diffusion-weighted magnetic resonance imaging of the brain reported as normal on report. Lumbar puncture was performed for the diagnosis of encephalopathy. Cerebrospinal fluid (CSF) analysis showed leukocytes 2 cells/mm3, protein 110 mg/L and glucose 59 mg/dL with simultaneous blood glucose 105 mg/dL. The Gram stain of CSF revealed no cells or organisms. Combination therapy consisting of intravenous ceftriaxone, vancomycin and acyclovir was started for encephalopathy. The result of real-time reverse transcription quantitative polymerase chain reaction (RT-qPCR) for 2019-nCoV from nasopharyngeal swab was negative for two times. Whereas serum 2019-nCoV IgM and IgG were negative and positive, respectively.

Due to the presence of fever, mucocutaneous, neurological, gastrointestinal and hematologic systems involvement, the possibility of MIS-C associated with COVID-19 was entertained. She was treated with intravenous immunoglobulin (IVIG) 2 g/kg in a single infusion based on treatment guidelines recommended for pediatric patients with MIS-C.(4) The patient was admitted from emergency department to a tertiary center hospital for further care and management. After the negative test results of CSF for herpes virus and cytomegalovirus, empiric acyclovir was discontinued 5 days later. Blood and CSF cultures were negative after 7 days. Her neurological symptoms started to improve on the fourth day follow-up. She was discharged home 10 days later with improved symptoms and signs to follow up with outpatient clinic. Follow-up visit 2 weeks later was unremarkable.

Discussion

The inhibitory dopaminergic receptors are susceptible to blockage by metoclopramide. The potential adverse extrapyramidal effects are parkinsonism, tardive dyskinesia, akathisia, and dystonia that may be confused with seizures, tetanus, strychnine poisoning and electrolyte imbalances. Female patients, children, adults younger than 30, and patients taking high doses of metoclopramide have higher chances of developing dystonic reactions.(2, 7) About 70% of dystonic patients are female(2), which was similar to our patient, a 7-year-old female, and female gender could be a risk factor. Normal or toxic metaclopramide doses can produce side effects, which are usually seen within the first 24–72 hours of drug intake.(3, 7) In this case, dystonic reactions had occurred after 24 hours of drug intake.

Standard treatment involves discontinuation of metoclopramide and administration of injectable antihistaminic or anticholinergic drugs. Benztropine and diphenhydramine are both effective in relieving dystonic reactions.(3, 7) Symptoms usually resolve dramatically within 10 - 30 minutes of administration of parenteral anticholinergics.(8) In this case, biperiden was used to treat our patient and dystonic reaction had disappeared within 30 minutes.

Biperiden that shows atropine-like properties with its central anticholinergic effect is regularly used predominantly in the symptomatic therapy of parkinson disease and movement disorders. Biperiden causes the anticholinergic syndrome by competitive inhibition of acetylcholine at the receptor level. Central effects; It is dose dependent and is observed as altered mental status, hallucinations, recent memory loss, agitation, respiratory failure and collapse of the cardiovascular system, while its peripheral effects are; mydriasis, blurred vision, appears as dryness of mucous membranes, high fever, tachycardia, dry skin, ileus and urinary retention.(9) There is evidence that COVID-19 infection is associated with neurological symptoms and central nervous system complications. Clinical manifestations of neurological symptoms of COVID-19 include meningitis, encephalitis, encephalopathy, headaches, seizures, and peripheral nervous system manifestations, such as myelitis, neuralgia, and Guillain-Barré syndrome. In addition, reports of neurological complications in children and adolescents have been associated with MIS-C.(10) According to this literature information, the side effects of biperiden can be confused with neurological findings of MIS-C. Although the neurological findings in our case occurred after biperiden injection, our case was diagnosed as MISC with laboratory tests. This suggested that the neurological findings developed due to MIS-C.

Since April of 2020, there have been case reports with clinical findings are similar to incomplete Kawasaki disease, toxic shock syndrome or secondary macrophage activation

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syndrome.(11, 12) On May 15th 2020, World Health Organisation (WHO) termed this condition as MIS-C. Case definition for MIS-C was reported by WHO and Centers for Disease Control and Prevention (CDC). Both definitions require fever, elevated inflammatory markers, multisystemic involvement (cardiac, renal, respiratory, hematologic, gastrointestinal, mucocutaneous, or neurological), hematologic findings, evidence of COVID-19, or likely contact with patients with COVID-19, and no other obvious microbial cause of inflammation or infection. In CDC case definition the patient must be under 21 years old and has to be inpatient, while according to WHO case definition patient could be between 0 and 19 ages and no need to be hospitalized. (13, 14) Gastrointestinal symptoms are dominant in patients with MIS-C. Furthermore, respiratory symptoms may not be present in pediatric patients with MIS-C. And also MIS-C associated with COVID-19 infection occurs two or more weeks after acute infection. Because of these reasons, the diagnosis of MIS-C is difficult.(4) Clinical, biological features and inflammatory markers of our patient were consistent with the diagnosis of MIS-C. We diagnosed an unusual case of MIS-C in a previously healthy female child with acute dystonia secondary to use of metoclopramide.

Patients with MIS-C are treated with different regimens, mostly the use of immunomudulatory medications, including IVIG and glucocorticoids as first-tier therapy. If there is no response to these drugs, biologic agents such as anakinra, tocilizumab and infliximab are among second-line treatment options.(15) In this case, our patient was successfully treated with IVIG and improved without sequelae.

Conclusion

In conclusion, physicians working in the pediatric emergency department should prompt alertness for symptoms and signs of MIS-C, and also keep in mind that metoclopramide has the potential to cause extrapyramidal side effects in children. This case report demonstrates that drug history should be questioned in pediatric patients with acute dystonic reaction, and MIS-C should definitely be kept in mind in the etiology of vomiting and fever during the COVID-19 pandemic.

Informed consent: Informed consent was obtained from the patient's family for the publication of the case report.

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Case Report

Journal of Emergency Medicine Case Reports

Significance of Differential Diagnosis for Febrile and Fatigued Patients in an Endemic Area During The COVID-19 Pandemic: Consideration of COVID-19, Brucellosis, and Crimean-Congo Hemorrhagic Fever

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Abstract

Brucellosis and Crimean-Congo Hemorrhagic Fever are diseases that can present with similar clinical and laboratory findings to those of COVID-19. This can delay diagnosis and increase the risk of nosocomial transmission in the case of Crimean-Congo Hemorrhagic Fever. Although misdiagnosis of Brucellosis and Crimean-Congo Hemorrhagic Fever, and even a case of coinfection have been reported in the literature, no case report mentioning Crimean-Congo Hemorrhagic Fever and Brucellosis coinfection hospitalized with the pre-diagnosis of COVID-19 was found. A 35-year-old female patient presented to the emergency service with complaints of fever and fatigue. The patient was evaluated in the emergency triage and was taken to the area where COVID-19 pre-diagnosed patients were being examined. A thorax computed tomography without intravenous contrast usage was reported as normal, and the patient was discharged after being informed about COVID-19 transmission routes. The patient re-applied to the emergency service with complaints of fever, fatigue, headache, and myalgia four days later. The laboratory findings showed a white-cell count of 1600/mm^3, haemoglobin of 12,8 g/liter, platelet of 146000/mm^3, urea of 21,5 mg/dl, creatinine of 0,81 mg/dl, alanine aminotransferase of 134 U/liter, aspartate aminotransferase of 303 U/liter, lactate dehydrogenase of 714 U/liter, creatine kinase of 1796 U/liter, C-reactive protein of 3 mg/liter, D-dimer of 2000 µg/liter, and a thorax computed tomography showed minimal ground-glass opacity. The patient was hospitalized with a preliminary diagnosis of COVID-19 by the chest diseases clinic. This case highlights the importance of considering other diseases with similar clinical and laboratory findings in endemic regions of Brucellosis and Crimean-Congo Hemorrhagic

Keywords: Brucellosis, COVID-19, Crimean-Congo Hemorrhagic Fever

Introduction

We are still dealing with the COVID-19 disease, which is affecting the entire world and is caused by SARS-CoV-2 (1). Brucellosis is one of the common zoonotic diseases and is generally transmitted by contact with infected animal tissues or by consuming the products of these animals. The disease, which is endemic in our country, is especially common in people living in rural areas and dealing with animal husbandry (2). Crimean-Congo Hemorrhagic Fever (CCHF), another common disease in this population, is one of the viral hemorrhagic fevers with endothelial damage (3). Patients with brucellosis and CCHF in endemic regions can also present with similar clinical and laboratory findings to those of COVID-19 (4), leading to misdiagnosis or confusion by visiting multiple departments. In addition, the diagnosis of the patient can be delayed, and the risk of nosocomial transmission may increase in the case of CCHF. Although misdiagnosis of Brucellosis and CCHF, and even a case of coinfection have been reported in the literature (5), no case report mentioning CCHF and brucellosis coinfection hospitalized with the pre-diagnosis of COVID-19 was found. In this report, we present a case of a patient who was admitted to the clinic with a preliminary diagnosis of COVID-19 and was detected to have brucellosis and CCHF coinfection.

Case Report

A 35-year-old female patient presented to the emergency service with complaints of fever and fatigue that had been ongoing for two days. The patient was evaluated in the emergency triage and was taken to the area where COVID-19 pre-diagnosed patients were being examined. There was no pathological finding in the physical examination of the patient, and no pathological finding was found in the serum

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parameters, except for an absolute lymphocyte count of 310/mm³. A thorax computed tomography (CT) without intravenous contrast usage was reported as normal. The patient was informed about COVID-19 transmission routes and discharged.

The patient re-applied to the emergency service with complaints of fever, fatigue, headache, and myalgia four days later. The laboratory findings of the patient, who was again evaluated in the area reserved for COVID-19 patients at the emergency department, showed a white-cell count of 1600/mm³ (reference range, 4490 to 12680), haemoglobin of 12,8 g/liter (reference range, 12 to 16), platelet of 146000/mm³ (reference range, 150000 to 450000), urea of 21,5 mg/dl (reference range, 17 to 43), creatinine of 0,81 mg/dl (reference range, 0,66 to 1,09), alanine aminotransferase (ALT) of 134 U/liter (reference range, 0 to 35), aspartate aminotransferase (AST) of 303 U/liter (reference range, 0 to 35), lactate dehydrogenase (LDH) of 714 U/liter (reference range, 0 to 248), creatine kinase (CK) of 1796 U/liter (reference range, 0 to 145), C-reactive protein (CRP) of 3 mg/liter (reference range, 0 to 5), D-dimer of 2000 µg/liter (reference range, 80 to 583). A low-dose thorax CT was performed and showed a minimal ground-glass opacity with a peripheral location limited to the fissure in the left lobe basal segment (Figure 1).

The patient was hospitalized with a preliminary diagnosis of COVID-19 by the chest diseases clinic. We evaluated the patient in the chest diseases service. The general state was good, with the patient being conscious, cooperative, and oriented. The temperature was 39,2 °C, blood pressure was 110/70 mmHg, and the heart rate was 102 beats per minute. Systemic examination was normal. We learned that the patient resided in a rural area and engaged in animal husbandry. Although the patient had no history of tick bites, she reported catching ticks on her body several times and disposing of them. The patient was transferred to our infectious diseases service with a pre-diagnosis of CCHF. Serum samples were sent to the Public Health Agency Microbiology Reference Laboratory Department for CCHF diagnostic tests, and supportive treatment was initiated. Detailed viral hepatitis diagnostic tests were performed due to high serum aminotransferase levels. Brucella Rose Bengal and standard tube agglutination tests were also requested, given the patient's engagement in animal husbandry, history of consuming fresh milk and dairy products, and residence in an endemic region for brucellosis. The patient's SARS-CoV-2 polymerase chain reaction (PCR) test was negative. On the second day of hospitalization, laboratory tests revealed leukopenia (white-cell count: 1200/mm³), a hemoglobin level of 12,2 g/liter, a platelet count of 114000/mm³, a urea level of 14 mg/dl, a creatinine level of 0,74 mg/dl, elevated ALT levels of 130 U/liter, AST levels of 196 U/liter, LDH levels of 523 U/liter, CK levels of 887 U/liter, a CRP level of 3 mg/liter, an international normalized ratio (INR) of 1,07 %, and blood type A Rh positive. Acute viral hepatitis was not detected, but the Brucella Rose Bengal test was positive, and the standard tube agglutination and Coombs' immunocapture agglutination tests were positive at 1/320 titers. Two sets of blood cultures were collected from the patient whose fever persisted. On the third day of hospitalization, laboratory tests showed leukopenia (white-cell count: 1600/mm³), a hemoglobin level of 13 g/liter, a platelet count of 86000/mm³, ALT levels of 111 U/liter, AST levels of 138 U/liter, LDH levels of 451 U/liter, CK levels of 510 U/liter, and an INR of 1,2 %. At the reference laboratory, the immunofluorescence method detected the presence of CCHF specific IgM antibodies (CCHFV Mosaic 2, Euroimmun Labordiagnostika AG, Germany). The diagnosis was also confirmed by real-time reverse transcriptase PCR (RT-PCR). Viral RNA isolation was performed using the High Pure Viral Nucleic Acid Kit (Roche Diagnostics GmbH, Germany), and the presence of viral RNA was tested using TaqMan-based single-stage RT-PCR. The Perkin-Elmer 7700 Sequence Detection System (Applied BioSystems, USA) was used for detection, with a combination of reverse transcriptase (MBI Fermentas, Germany) and Hot Start Taq DNA polymerase (Bioron GmBH, Germany). On the third day of hospitalization, specific treatment was initiated for brucellosis (rifampicin 600 mg per day and doxycycline 100 mg twice a day per oral). The patient's clinical course was closely monitored, and serum parameters were followed closely. Along with treatment for brucellosis, supportive treatment was given for CCHF. The patient's fever showed regression after the fourth day of follow-up; serum parameters such as leukopenia and aminotransferase levels began to improve (Table 1), and the patient's complaints were alleviated.

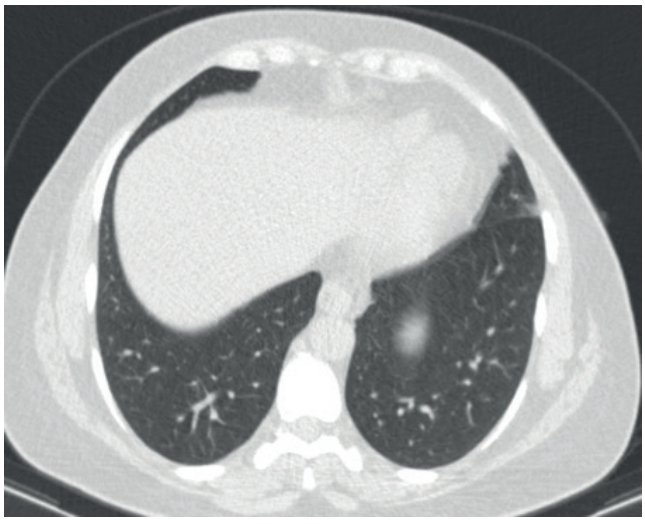

Figure 1. Axial CT Image of the Chest Showing A Minimal Ground-glass Opacity.

Table 1: Laboratory data

	Hospitalization Period				
Variable	**Day 1**	**Day 2**	**Day 3**	**Day 4**	**Day 8**
WBC (/mm³)	1600	1200	1600	2600	4500
Hb (g/liter)	12,8	12,2	13	13,1	13
Platelet (/mm³)	146000	114000	86000	74000	149000
Urea (mg/dl)	21,5	14	-	-	20
Creatinine (mg/dl)	0,81	0,74	-	-	0,7
ALT (U/liter)	134	130	111	100	32
AST (U/liter)	303	196	138	113	34
LDH (U/liter)	714	523	451	353	220
CK (U/liter)	1796	887	510	348	120
INR (%)	-	1,07	1,2	1,04	1,04

On the eighth day of hospitalization, all serum parameters were within normal limits, and the patient's symptoms had significantly improved. Blood cultures obtained on the second day of hospitalization were negative. The patient was hospitalized for ten days and then discharged with instructions for oral brucellosis treatment. The patient's follow-up is currently ongoing on an outpatient basis. Informed consent was obtained from the patient during their hospitalization in our facility.

Discussion

During the current COVID-19 pandemic, patients may present with many non-specific symptoms such as fever, fatigue, myalgia, headache, and back pain (1). The symptoms of brucellosis and CCHF patients also have a wide range, many of which are similar to those of COVID-19.

Brucellosis is a zoonosis caused by an intracellular bacterium and can therefore affect many organs and systems. The most common findings are fatigue, fever, night sweats, myalgia, and arthralgia (6, 7). None of these findings are specific to brucellosis. Hematological abnormalities such as leukopenia, anemia, and thrombocytopenia can occur due to bone marrow involvement and hypersplenism. Diseases causing bone marrow involvement and hypersplenism should be considered in the differential diagnosis (8, 9). Another disease that often causes similar findings is CCHF. The clinical presentation and laboratory findings of CCHF resemble those of brucellosis, and they can be differentiated by visible skin, mucous membranes, and other organ bleeding at the advanced stage of the disease (10). Both diseases are more common in people engaged in animal husbandry and living in rural areas (11). Similar clinical and laboratory findings have been reported in different studies (12, 13).

COVID-19 can also manifest as similar clinical (fever, fatigue, headache, etc.) and laboratory (cytopenia, aminotransferase elevation, D-dimer, INR elevation) findings. Rural residents have also been affected by the COVID-19 pandemic. In addition, SARS-CoV-2 and Nairovirus are RNA viruses. Brucella is also an intracellular pathogen, like viruses. Symptoms and clinical findings of infections caused by viruses and intracellular pathogens can be very similar. In such cases, a careful differential diagnosis is essential.

Our patient, who lives in a rural area and works with animal husbandry, presented with leukopenia, thrombocytopenia, and elevated levels of AST, ALT, LDH, and CK. Based on these findings, we diagnosed CCHF. To confirm the diagnosis, we sent a serum sample taken on the second day of hospitalization to the reference laboratory and closely monitored routine laboratory values. Brucellosis was also suspected, based on a previously reported case by Karakeçili et al. (5), and Brucella Rose Bengal, standard tube agglutination (1/320), and Coombs' immunocapture (1/320) tests were positive. The results from the reference laboratory showed that IgM and RT-PCR tests were positive for CCHF. Consequently, we diagnosed our patient with co-infection of brucellosis and CCHF, who was initially hospitalized with a pre-diagnosis of COVID-19. We considered the brucellosis as acute, given the sudden onset of the patient's symptoms and the absence of a previous history of the disease. The lack of long incubation period was suspected as the reason for the negativity of the two blood cultures gathered during hospitalization. The patient received rifampicin and doxycycline therapy for brucellosis, and we closely monitored the serum parameters after the positive PCR result for CCHF. With specific brucellosis treatment and supportive treatment for CCHF, the patient's symptoms improved, and serum parameters returned to normal.

To our knowledge, no case of co-infection of brucellosis and CCHF with a pre-diagnosis of COVID-19 has been reported previously. Our case underscores the importance of considering CCHF and brucellosis in the differential diagnosis of patients presenting with similar symptoms, particularly in endemic regions during the COVID-19 pandemic. Our case also highlights that the two diseases can coexist. Fortunately, the droplet and contact isolation precautions applied for the preliminary diagnosis of COVID-19 were also sufficient to prevent the transmission of CCHF. Early diagnosis of CCHF in these patients can prevent nosocomial transmission and ensure appropriate diagnosis and treatment.

In conclusion, a careful differential diagnosis is crucial in patients pre-diagnosed with COVID-19 who present with vague symptoms, particularly in regions where both brucellosis and CCHF are endemic

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Case Report

Journal of Emergency Medicine Case Reports

Upper Extremity Injury Related to Wheeled Recreational Device-Hoverboard: A Case Report

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Abstract

Hoverboards were designed for making our daily life easier. However, lots of patients admitted to emergency services due to these devices require a high level of balance, coordination and strength. Traumatic injuries are more common especially in children and young adults. Forearm fractures are one of the important trauma outcomes in the pediatric age group. Although supracondylar or forearm fractures are common, we rarely encounter a combination of both, defined as floating arm fractures. A 9-year-old boy falling from a hoverboard had supracondylar humerus fractures accompanied by olecranon and distal Radius fractures in the same arm. A temporary long arm splint was applied to the patient who was transferred to the orthopedic clinic and surgical operation was planned for stabilization. In our case, we aimed to discuss the coexistence of olecranon and distal radius fracture accompanying ipsilateral supracondylar humerus fracture in a child hoverboard user..

Keywords: Supracondylar Humerus Fracture, Hoverboard, Olecranon Fracture, Radius Fracture

Introduction

Hoverboards were designed and started to be used in 2005 with the idea of making our daily life easier. More than 2.5 million sales were reported in the US in 2015. However, it was determined that the number of patients admitted to emergency care centers, emergency services and clinical practices due to trauma increased in the same years (1-2). In 2015, there was an avarage 208% increase a number of injuries compared to any of the previous 4 years (3). Hoverboards (Figure-1) are capable of moving at a speed of 20 km/h. These devices require a high level of balance, coordination and strength (4). Traumatic injuries are more common especially in children and young adults who do not have sufficient experience or have attention deficits. Forearm fractures are one of the important trauma outcomes in the pediatric age group. Although supracondylar or forearm fractures are common, we rarely encounter a combination of both, defined as floating arm fractures (5). In our case, we aimed to discuss the coexistence of olecranon and distal radius fracture accompanying ipsilateral supracondylar humerus fracture in a child hoverboard user.

Case Report

A 9-year-old boy who presented with an injury as a result of falling from a hoverboard had diffuse pain sensitivity in the right forearm and forearm was deformed. No additional

Figure 1.

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Journal of emergency medicine case reports. 2023;14(2): 37-39

trauma-related injury was detected in the systemic physical examination of the patient who had no known past history of chronic disease. Forearm peripheral pulses are palpable and no sensory/motor deficit was detected in neurological examination. X-ray images of the forearm and elbow revealed supracondylar humerus fractures (AO classification 13-m/3 types II) accompanied by olecranon and distal radius fractures in the same arm (Figure- 2). A temporary long arm splint was applied to the patient who was transferred to the orthopedic clinic and surgical operation was planned for stabilization.

Discussion

In this case, olecranon and distal closed arm fracture accompanying supracondylar humerus fracture in the same arm is discussed. Although arm fractures are frequently encountered in clinical pratice, ipsilateral fractures involving both the upper and forearms are rarely seen. The reported incidence of forearm fractures associated with supracondylar fractures ranges 2% to 13 % (6). Even if elbow injuries and forearm fractures are common in children, the incidence of these injuries is approximately 3-13% (6-7). According to Taylor et al., normal anteversion of the distal humerus by falling on the open hand while the elbow is partially flexed, converts the compressive forces to shear forces, resulting in the formation of a supracondylar fracture with distal or middle forearm fractures. Supracondylar fractures in children usually occur by falling on the open hand while the elbow is in hyperextension, and it is also seen with forearm fractures when the force reflected on the wrist is excessive (8). In our case, we consider that the humeral midbody fractue was caused by a second force, which was caused by falling on the open hand and after the radius distal and supracondylar fractures occured, and the arm hitting the edge of the stairs. Although bicycle injuries are an important cause of trauma in childhood, nowadays, in addition to scooter and skateboard injuries, hoverboard injuries, which attract the attention of the child age group, are also encountered.

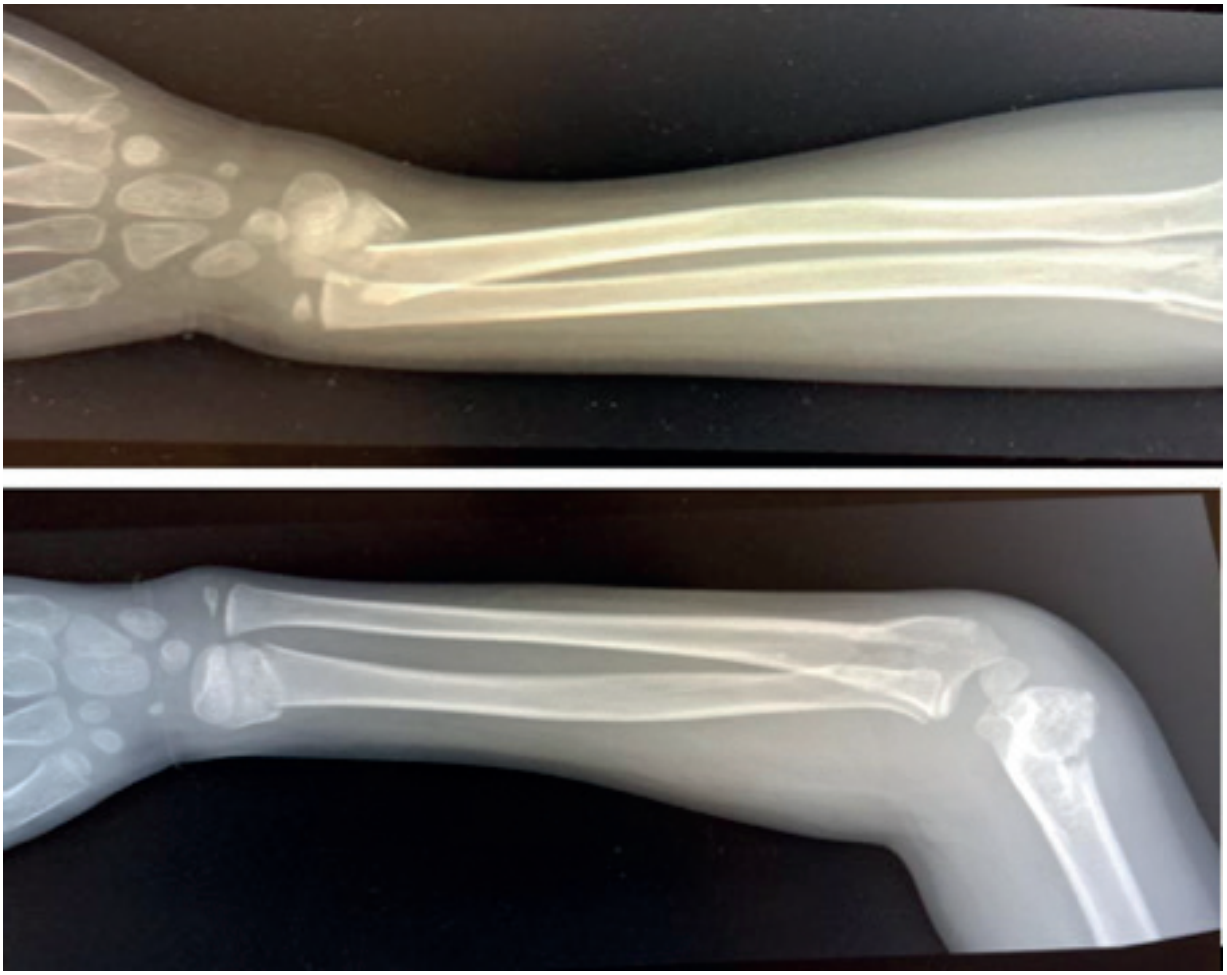

Figure 2.

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Case Report

Journal of Emergency Medicine Case Reports

Rare Case of Coronary Anomaly, Overview of Hypertrophic Cardiomyopathy with A Different Presentation

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Abstract

Coronary artery anomalies are uncommon cardiac diseases. It is mostly detected incidentally in the adult population as well as being usually asymptomatic. Although its prevalence is low, its association with atherosclerosis, arrhythmias, and hypertrophic cardiomyopathy is important for sudden cardiac death. According to the literature, the association of hypertrophic cardiomyopathy with the coronary anomaly is rare. No case of hypertrophic cardiomyopathy accompanied by the absence of the left anterior descending artery has been reported. Therefore our case is special and valuable..

Keywords: Atrial Fibrillation, Coronary Anomaly, Hipertrophic Cardiomyopathy , Single Coronary Artery

Introduction

Coronary artery anomalies are uncommon cardiac diseases. Although its prevalence is low, its association with atherosclerosis, arrhythmias, and hypertrophic cardiomyopathy is important for sudden cardiac death. (1,2,3) In order to talk about the coronary anomaly, we need to start by knowing the normal path of the coronary arteries. The left anterior descending artery (LAD) is the subepicardial artery, located in the anterior interventricular sulcus, giving septal penetrating branches. The circumflex artery (Cx) is the subepicardial artery, located in the left atrioventricular sulcus, giving at least one obtuse marginal branch. The right coronary artery (RCA) is the subepicardial artery, located in the right atrioventricular sulcus, giving at least an acute marginal branch. (4) Coronary artery anomalies can be classified in different ways. Shriki et al. grouped them under two headings as anomalies with hemodynamic severity and anomalies without hemodynamic severity. (5) The classification developed by Angelini and later Khatami et al. can be accepted as the most widely used classification. Based on these classifications, coronary artery anomalies can be divided into three or four large groups. Group A: coronary artery outflow abnormalities and distribution abnormalities, Group B: Intrinsic coronary artery anatomy abnormalities and Group C: Coronary artery termination abnormalities, Group D: Abnormal collateral vessels. (6,7) The most common coronary anomaly is the atypical origin of CX from the right coronary circulation. However, data on left anterior descending artery anomalies are still insufficient in the literature. (4,8)

Hypertrophic cardiomyopathy is a genetic disease resulting from the thickening of the myocardium. It can cause complications such as heart failure, mitral valve disorders, atrial fibrillation, and sudden cardiac death. According to the literature, its association with coronary anomalies is rare. (2,3,9,10,11) Hypertrophic cardiomyopathy can cause sudden cardiac death, this risk increases when it is related to coronary anomaly.(11) For this reason, patients with hypertrophic cardiomyopathy with coronary anomaly should be observed closely.

Case Report

A 37-year-old female patient presented to the emergency department with complaints of constricting chest pain and palpitations for several days. We were consulted by the emergency department with the initial diagnoses of atrial fibrillation with rapid ventricular response and acute coronary syndrome**.** It has been learned from her anamnesis that she had no known systemic disease or rhythm disorder and that she had not experienced syncope before. When her

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family history was examined, it was revealed that there was no sudden cardiac death in any of her relatives. On physical examination, rales were heard in the bilateral lower zones of the lungs and two positive pretibial edemas were observed. In laboratory results, troponin value was 0.4 ug/L (normal range 0- 0.16 ug/L), C-reactive protein (CRP) value was 16 mg/dL (normal range 0- 5 mg/dL), D-Dimer value was 1.5 mg/L (normal range 0- 0,55 mg/L), creatinine value was 0.7 mg/dl (normal range 0.5-0.9 mg/dl). Electrocardiography (ECG) showed atrial fibrillation rhythm with rapid ventricular response and ST depressions in leads V2-5. (Figure 1) Echocardiography (ECHO) showed 40% ejection fraction, global hypokinesia, and septal hypertrophy. The septum thickness was 17 mm and the posterior wall was 18 mm. (Figure 2) In the patient's medical history, when he applied to an external center due to shortness of breath 2 years ago, on the cardiology consultation at that time, the ejection fraction was normal in ECHO and she was not taking any medication. Pulmonary computed tomography (CT) angiography was performed with the prediagnosis of pulmonary embolism in the patient who had D-dimer elevation and newly diagnosed atrial fibrillation. No image

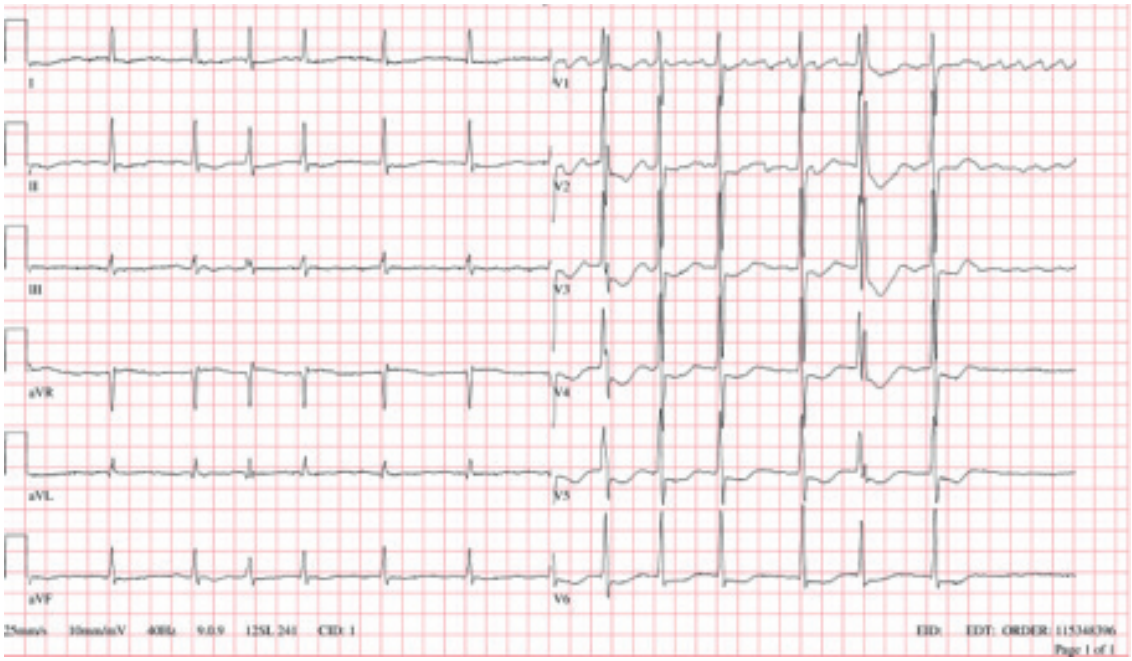

Figure 1. ECG atrial fibrilation.

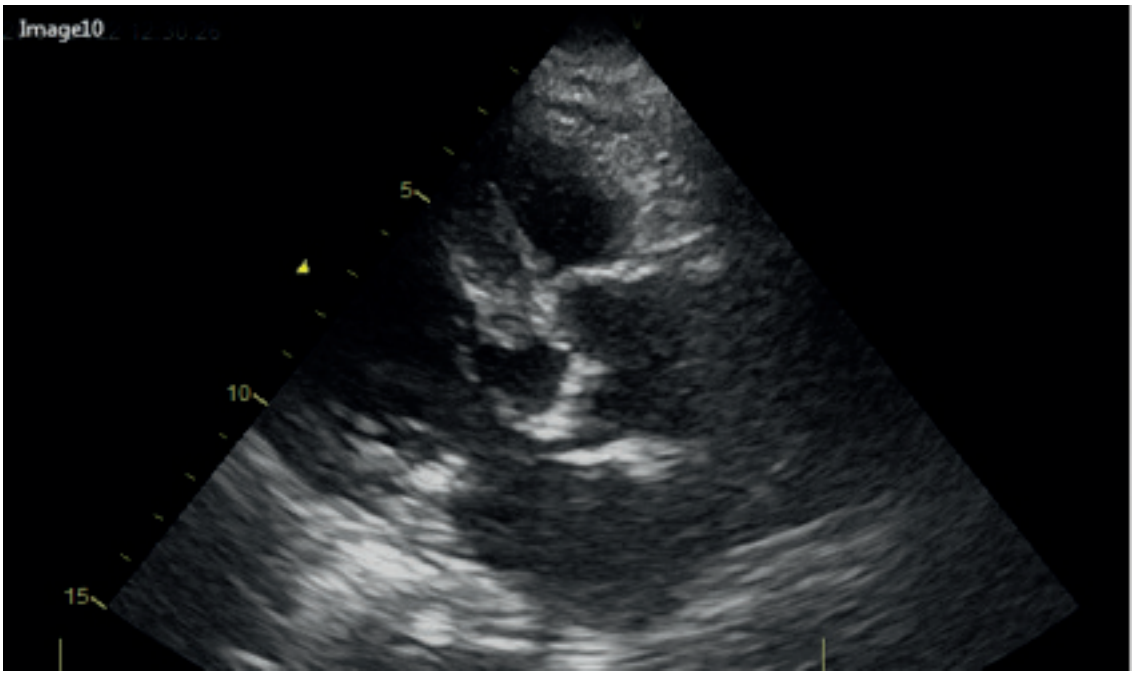

Figure 2. Transthoracic echocardiography image,septal hypertrophy.

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compatible with embolism was detected in CT angiography. First of all, rate control was achieved and attempted to treat symptoms of decompensated heart failure. Upon detection of ST depressions in ECG and wall motion defect in ECHO, the patient was taken to coronary angiography after the patient's loading regressed. Coronary angiography of the patient revealed left main coronary artery agenesis, RCA, and CX originating from the same ostium and feeding the LAD area together, and a muscular bridge causing 80% stenosis in the distal RCA. (Figure 3) Afterward, cardioversion was planned to provide rhythm control to the patient. In the transesophageal echocardiography (TEE) performed before, apical hypertrophy and 0.8x2.0 cm thrombus in the left atrium were seen.(Figure 4) Cardioversion was delayed due to the detection of a thrombus in the left atrium. We started rivaroxaban 20 mg and arranged the heart failure treatment. We discharged the patient to be re-evaluated with TEE and cardioversion, after using anticoagulant therapy for six months.

Discussion

Although coronary anomalies are usually innocent, they have been shown to cause sudden cardiac deaths, although rarely. Many anomalies are found incidentally in coronary angiography and autopsies (12). The majority of these anomalies are incidentally detected benign (81%) anomalies that do not induce a major threat to myocardial perfusion (13,14). The spectrum of symptoms thought to be caused by coronary anomalies includes angina, syncope, congestive heart failure, and sudden death. Coronary artery anomalies are the second cardiovascular reason of sudden death in

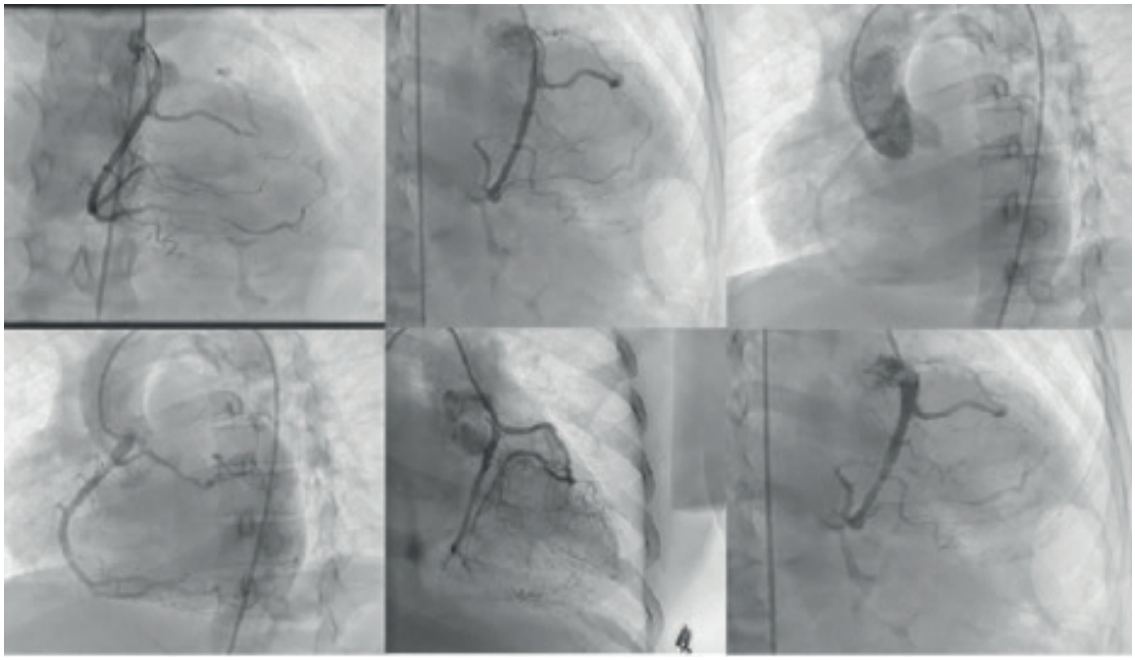

Figure 3. Coronary angiography image (LMCA agenesis Absence of LAD , RCA and CX originate from the same ostium)

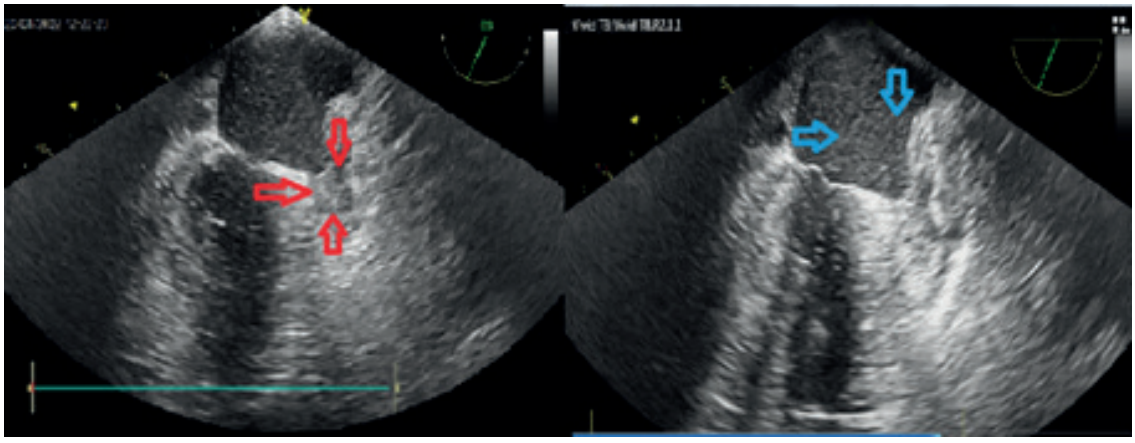

Figure 4. TEE image of left atrial appendix thrombus (red arrows) and left atrium SEC (blue arrows)

young people after hypertrophic cardiomyopathy.(13) Therefore, the combination of hypertrophic cardiomyopathy and coronary anomaly has great importance in terms of sudden cardiac death. These patients require close clinical follow-up and they may need implantable cardioverter-defibrillators (ICD). When we look at the literature, the scarcity of anomalies and the absence of LAD in our patient makes our case special. At the same time, considering that the detection of these cases are mostly coincidental, the fact that our case was diagnosed with atrial fibrillation with rapid ventricular response has made our case more interesting. We hope that this will put our case at the one of the top of the list of coronary anomalies in the literature and we hope to discover different coronary anomalies with new presentations.

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

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Case Report

Journal of Emergency Medicine Case Reports

A Case Report of Metformin Related Lactic Acidosis

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Abstract

Metformin is an oral antidiabetic drug of the biguanide classused in type 2 diabetic patients with normal renal function. The mortality rate is high in cases of lactic acidos is developing in metformin intoxication. In the emergency department, metformin intoxication should be considered in the differential diagnosis of patients whouse metformin and have high anion gap metabolic acidosis (lactic acidosis). The most important and effective treatment with early diagnosis is correction of metabolic acidosis with hemodialysis or hemofiltration methods, bicarbonate treatment, adjustment of blood glucose level. Cardiovascular system support therapy significantly reduces morbidity/mortality. In this case report, we present the early diagnosis and successful treatment of a patient with lactic acidosis due to metformin intoxication.

Keywords: Metformin, Poisoning, Lactic Acidosis

Introduction

Poisoning; It is the situation that stops vital functions where a substance enters the living organism through mouth, skin, respiration, circulation, and damages its function (1). According to the first report of Turkish National Poison Control Center on the application by poisoning in Turkey is located medical drugs (69.74%) (1). Metformin is a biguanide group insulin-sensitizing drug used in the treatment of type 2 Diabetes Mellitus (DM) (2). Metformin exertsits effect by decreasing hepatic glucose production and gastrointestinal glucose absorption and increasing peripheral glucose utilization (2, 3, 4). The blood glucose lowering effect of metformin is largely due to the 25-30% decrease in endogenous glucose production. To a less erextent, it also decreases plasma glucose levels by increasing glucose uptake by skeletal muscles and adipose tissue. Unlike sulfonylurea group oral antidiabetics, it does not stimulate insulin release. It is excreted from the body through the kidney and may accumulate in the body due to decreased clearance in cases of renal failure (2, 3, 4, 5, 6). Side effects of metformin usually include gastrointestinal complaints such as nausea, anorexia, diarrhea, abdominal cramps and hypothermia (2, 4, 6). Side effects occur in more than 50% of patients. The most important known side effect of metformin is lactic acidosis, which has a high mortality rate and should not be used in patients with creatinine levels>1.4 mg/dL (2, 3). Metformin-induced lactic acidosis is characterized by high blood lactate concentration, decreased blood pH and electrolyte disorders with increased anion gap. Although lactic acidosis due to metformin use has a beter prognosis than other types of lactic acidosis, reported mortality rates can be as high as 25-50%. According to current guidelines, if the estimated glomerular filtration rate (eGFR) falls to<45 mL/min/1.73 m2, the dose of metformin should be reviewed. In patients with an estimated GFR <30 mL/min/1.73 m2, drug use should be stopped. The oretically, impaired renal function decreases GFR and this increases metformin accumulation in the body (2, 3, 4). In this case, a patient hospitalized in the internal medicine clinic because of lactic acidosis due to metformin intoxication was successfully treated with early diagnosis.

Case Report

A 57 years old woman was admitted to the emergency department with vomiting, confusion and low blood glucose. According to the anamnesis obtained from the patient, she had consulted to the family physician with the same complaints about 1 hour ago and the patient's blood glucose level was 55 mg/dl at the family health center, dextrose infusion was started and the patient was referred to the emergency department when her complaints persisted although her blood glucose level increased to around 200 mg/dl. It was learned that the patient had diabetes mellitus and was taking metformin for this reason. When the drugs used by the patient were questioned, he stated that he used metformin irregularly. For this reason, he

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stated that he had taken extra metformin tablets recently. Initial evaluation of the patient was performed in the emergency department. Blood tests of the patient were analyzed. Blood glucose, sodium, calcium and carbonmonoxide values were found to be with in the normal range. Creatinine value was 0.94 mg/dL (minimally elevated). Computerized brain tomography and diffusion MRI performed due to clouding of consciousness revealed no pathology. In the venous blood gas analysis of the patient; pH: 7.2, pCO_2: 35.4 mmHg, pO_2: 40.4 mmHg, lactate: 13.5 mmol/L, metabolic acidosis with anion gap and increased base deficit was present. A differential diagnosis was made to explain vomiting, confusion and lactic acidosis and metformin-associated lactic acidosis was considered. The patient was hospitalized in the internal medicine clinic. In the continuation of the treatment, the general condition of the patient improved and the blood gas obtained during the treatment process showed pH: 7.29, pCO_2: 41 mmHg, lactate: 9.5mmol/L. Base deficit and bicarbonate were with in normal range. In the arterial blood gas analysis of the patient in the 1st week of treatment; pH: 7.38, pCO_2: 43 mmHg, pO_2: 97 mmHg, HCO_3: 24.3mmol/L and lactate: 1.2 mmol/L, his general condition improved completely and he was discharged with recovery.

Discussion

Metformin is a drug from the biguanide groupu sed in the treatment of type 2 diabetes mellitus (2). In high dose metformin intake, nonspecific symptoms including anorexia, lethargy, nausea, vomiting and epigastric pain as well as life-threatening symptoms including moderate renal failure, hypotension, hypothermia, respiratory failure and cardiac rhythm disturbances may develop (2, 3, 4, 7). Our patient had nausea/vomiting, minimally elevated creatinine levels and confusion. Eosinophilia in peripheral blood, hematuria and proteinuria in urine are possible findings in cases of interstitial nephritis (2, 3, 4). In our case, eosinophilia and hematuria/proteinuria in urine analysis were absent.

Lactate is one of the end products of anaerobic glycolysis. Lactate is utilized by hepatocytes and converted to glucose through gluconeogenesis. Normal blood lactate levels range between 0.5 and 1 mmol/L. Blood lactate levels greater than 2 mmol/L are defined as hyperlactatemia. Lactic acidemia occurs when serum lactate concentration exceeds 4 mmol/L with pH less than 7.35. In general, elevated lactate levels result from increased production or decreased urinary excretion. Etiology of the anaerobic metabolism required to cause a lactic acidosis includes various disease processes such as sepsis, hemorrhagic shock, cardiac arrest, trauma, intoxications (such as: metformin poisoning, metabolic poisons such as cyanide), burns, diabetic ketoacidosis, cancers and intense muscle activity (8). In a study by Wills et al. the incidence of metformin-induced severe lactic acidosis was reported to be 9.1% in patients who received a single over dose of metformin (9). In another study conducted by Li Cavoli et al. among 1014 renal patients, it was reported that acute renal failure accompanied by lactic acidosis was detected in 47 patients who used metformin for hyperglycemia control (10). In another case of metformin intoxication reported by Mustafa et al., acute renal failure with lactic acidosis and hypothermia was reported (11). In our case, there was an elevated lactate level in the blood gas obtained at the first presentation. In articles in the literature reporting the development of metformin-induced lactic acidosis and acute renal failure, it has been reported that hemodialysis is usually performed in treatment (12). In our patient, blood creatinine levels were found to be minimally elevated and normalized with hydration/fluid support. Hemodialysis was not performed. The effects of metformin on the kidney are thought to be related to renal hypoperfusion or direct renal toxic effect of metformin (12).

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Case Report

Journal of Emergency Medicine Case Reports

Can Facial Paralysis be a Rare Complication of Hypertension?

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Abstract

Facial paralysis is a disorder that can result from a wide spectrum of etiologies including traumatic, infectious, congenital, neurologic, systemic, neoplastic, and iatrogenic causes. It has significant functional, psychological, and social consequences. The most common cause of fascial nerve paralysis is idiopathic facial nerve palsy (Bell's palsy). There is a relationship between facial nerve paralysis and severe systemic hypertension. In this report we present a 43-year-old female patient with fascial paralysis who had a history of hypertension.

Keywords: Facial paralysis, Hypertension, Bell's palsy, Aetiology

Introduction

Facial paralysis is a disorder that can result from a wide spectrum of etiologies including traumatic, infectious, congenital, neurologic, systemic, neoplastic, and iatrogenic causes.[1] It has significant functional, psychological, and social consequences. Functionally diminished lacrimation, ectropion, epiphora, brow ptosis and lagophthalmos can lead to exposure keratopathy with resulting blindness and potential for globe rupture.[2] Nasal valve dysfunction is observed in facial paralysis. Oral competence with resulting difficulties with speech, swallowing, and drooling is also seen.[3]

The most common cause of fascial nerve paralysis is idiopathic facial nerve palsy (Bell's palsy).[4] Bell's palsy (BP) is defined as an acute peripheral facial paralysis, generally affecting one half of the face. The incidence of BP is 15-30/100,000 and it accounts for 60-75% of all unilateral facial palsies.[5] The clinical presentation of BP is usually a rapid onset, unilateral, lower motor neuron-type facial weakness accompanied by symptoms of subjective change in facial sensation, dysgeusia, postauricular pain and hyperacusis. This clinical presentation can be caused by the anatomical construct of the human facial nerve, and especially its mixed nerve profile containing sensory, motor and parasympathetic fibres.[6] It's medical treatment includes corticosteroids and antiviral agents, while physical therapy involves automassage and exercises. Corticosteroids may work best if they are initiated within several days of when the symptoms started.

There is a relationship between facial nerve paralysis and severe systemic hypertension, as was described for the first time by Moxon more than a century ago.[7] In a recently published case-control study it was stated that chronic, nonsevere hypertension may increase the risk of lower motor neuron facial nerve paralysis in patients older than 40 years of age.[8] In this report we present a 43-year-old female patient with fascial paralysis who had a history of hypertension.

Case Report

A 43-year-old female patient presented to our clinic with complaints of headache, facial asymmetry and numbness. Vitals of the patient were found as blood pressure: 155/112 mmHg, SpO_2:96%, and body temperature: 36.7°C. On physical examination of the patient, it was observed that her face was asymmetrical and there was no wrinkle on the right side of her forehead. The patient could not raise her right eyebrow, and could not close her right eye. When she wanted to close her eye, the eyeball was moving up. There was effacement in the right nasolabial groove and sagging

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in the ipsilateral mouth corner. The right half could not accompany laughing, and the patient could not show her teeth. Besides, she described numbness and loss of feeling on the right side of her face. The patient's ECG showed normal sinus rhythm, and there was no abnormality in respiratory and heart sounds. No abnormality was detected in the neurological examination. Although the patient had a history of hypertension, she had not been using any medication for the past year. Laboratory parameters of the patient was found as Hgb: 12.9 mg/dL, Urea: 2.8 mmol/L, creatinine: 52 umol/L, ALT: 68 U/L, AST: 55 U/L, total cholesterol: 4.91 mmol/L, triglyceride: 1.67 mmol/L, HDL: 1.55 mmol/L, HbA1C: 5.6, insulin: 53.7 mcu/mL, TSH: 0.89mIU/L, and FT4: 16.7 pmol/L. The patient was diagnosed with idiopathic Facial Paralysis and was evaluated as Grade 4 according to the House-Brackmann scale. The patient was administered 1 mg/day prednisolone PO tb, acyclovir 800 mg 5x1, synthetic tears and eye closure treatments. In addition, amlodipine 10 mg 1x1 treatment was started and blood pressure was followed up daily and it was controlled in the first week. In the fifth week after the diagnosis, full recovery was observed in all motor and neural functions of the patient. The patient was informed about the objective of this report and gave written informed consent.

Discussion

The facial nerve is a cranial nerve whose function is most impaired due to its long and curved journey in the facial canal between the infratemporal bones.[9] More than 50% of cases of facial paralysis are idiopathic (Bell's palsy).[5] Although the exact etiology of BP is not known, compression of the facial nerve in the canal as a result of edema due to infection/inflammation and vascular ischemia is the most accepted mechanism.[10] Agents such as Herpes simplex virus (HSV), Varicella-zoster virus (VZV), Borrelia burgdorferi have been detected in the cerebrospinal fluids of patients with BP.[11]

When the literature was reviewed to examine the relationship between facial paralysis and hypertension, articles on childhood hypertension and pregnancy-related facial paralysis came to the fore.[12-14] Especially MacArthur and Minson stated that timely diagnosis and treatment of hypertension in facial paralysis improves the prognosis, and blood pressure measurements in children can prevent the disease.[12]

It has been reported that facial paralysis in children is not limited to malignant hypertension and the incidence of facial paralysis increases in benign hypertension cases.[15] When the literature was searched for those over the age of 18, different results were found for the relationship between hypertension and BP. Some studies have reported that hypertension, especially over the age of 40, increases the incidence of BP.[8,16] Our case was 43-year-old and she had a history of uncontrolled hypertension.

Corticosteroids are recommended in the treatment of BP because they reduce edema, increase facial nerve regeneration and improve motor functions.[17] In our case we used prednisolone as corticosteroid.

Although the idea of using antiviral agents in the treatment of BP due to the viral activity shown in the etiology of Bell's palsy, thanks to the increasingly widespread serological tests, has begun to be accepted more and more, in the systematic reviews and meta-analyses conducted to date, definitive and sufficient clinical evidence of the effectiveness of antiviral agents in BP has not yet been obtained.[18] We used acyclovir as the antiviral agent in our patient. Antiviral agents are used as standard treatment in Ramsay Hunt syndrome, which is a disease characterized by reactivation of Varicella zoster virus, which remains latent only in the geniculate ganglion, and subsequent acute peripheral facial paralysis, vesicular rash in the auricle and external auditory canal, and severe ear pain.[19] However, the use of antiviral therapy is still controversial.

Although it has been reported that successful results are obtained when surgical treatment is applied in the early period (within 2-3 weeks after the onset of symptoms) in patients who do not improve despite steroid treatment and who have degeneration over 90-95% in electrodiagnostic evaluations, further studies are needed to determine a common treatment method.[20]

In conclusion, corticosteroids, antiviral agents and antihypertansive medications can be used in the treatment of fascial paralysis caused by Bell's palsy in patients with systemic hypertension. However, since there is no consensus especially on the use of corticosteroids in these cases, further comprehensive studies are needed to enlighten this issue.

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Case Report

Journal of Emergency Medicine Case Reports

A Case Report of a Patient Who Had Intravaginal Leech Application

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Abstract

Intravaginal leech application is a traditional medical practice used in some cultures to treat various gynecological conditions. Leeches are believed to improve blood circulation and promote healing in the affected area. Studies have shown that the bioactive substances injected by leeches, when their mechanisms of action are examined, can be used in the treatment of many diseases. In this case report, we present a case of vaginal bleeding after intravaginal leech application.

Keywords: Leech therapy, Intravaginal bleeding, Hirudotherapy

Introduction

Intravaginal leech application is a traditional medical practice used in some cultures to treat various gynecological conditions (1). Leeches are believed to improve blood circulation and promote healing in the affected area. The mechanism of action of leech therapy occurs when leeches inject a secretion containing many bioactive substances into the circulation while sucking blood (Table 1). Studies have shown that the bioactive substances injected by leeches, when their mechanisms of action are examined, can be used in the treatment of many diseases (2, 3). Leech therapy is a treatment method that may lead to undesirable effects if not performed by competent practitioners. Improperly performed leech therapy may result in complications such as prolonged bleeding, wound infections, hypovolemic shock, anemia, and allergic reactions (4).

In this case report, we present a case of vaginal bleeding after intravaginal leech application.

Case Report

A 40-year-old female patient (G3-P2-A1) consulted a traditional healer who recommended intravaginal leech application to increase her sexual pleasure and boost her immunity. The patient agreed to undergo the procedure, and 11 intravaginal leeches were placed in a traditional complementary medicine center two days ago. The leeches were removed approximately six hours after the application, after which the patient experienced mild pain and discomfort in her lower abdomen. On the first day, the patient was aware that these symptoms could occur after leech therapy and did not seek medical attention. However, on the second day following the procedure, she began to experience heavy vaginal bleeding. The patient then went to the emergency service, complaining of persistent abdominal pain and bleeding in the genital area that would not stop.

On physical examination in the emergency department, the patient was pale, anxious, and tachycardic (pulse 110/min), and blood pressure was 90/60 mmHg. In the pelvic examination, active bleeding foci with clots were observed in the vaginal dome. Due to the proximity of the anatomical region, melena and fresh blood were not detected in the rectal examination performed to exclude bleeding from the lower gastrointestinal tract. There was no evidence of trauma or infection. Intravenous fluid (2000-3000 cc saline) was started immediately and blood transfusion (erythrocyte suspension) was considered. He was also given antibiotics to prevent infection and analgesics for pain relief. In our case, hemoglobin value, platelet count, prothrombin time and activated partial thromboplastin time were within normal limits. The b-hcg result of our patient was negative and it was determined that she was not pregnant.

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Table 1: Some bioactive substances in leech secretion and their effects (2)

Hirudin	Inhibits blood coagulation by binding to thrombin
Calin	Inhibits blood coagulation by blocking the binding of Von Willebrand Factor to collagen, inhibits collagen -mediated platelet aggregation
Destabilase	Dissolves fibrin with monomeric activity and exhibits thrombolytic effect
Hirustatin	Inhibits cathepsin G, kallikrein, triptin, chymotriptin, and neurofolic
Bdellins	Exhibits anti-inflammatory effects, inhibits plasmin, tristine, and acrosin
Hyaluronidase	Increases interstitial viscosity and shows antibiotic effect
Tryptase Inhibitor	Inhibits proteolytic enzymes of mast cells
Eglinler	Exhibits anti-inflammatory effects, inhibits the activity of α-chymotrypsin, chymase, subtilisin, elastase, and cathepsin G
Factor Xa inhibitor	Inhibits the activity of coagulation factor Xa by forming complexes with the same molecular densities
Carboxypeptidase-A	Increases blood flow at the bite site
Acetylcholine	It is a vasodilator
Histamine-like substances	Vasodilator, increases blood flow in the bite area

The patient, whose bleeding continued despite the first intervention, was consulted to the obstetrics and gynecology clinic for further examination and treatment. In the speculum examination performed on the patient, several active vaginal bleeding foci were observed.

Leeches were not visible upon examination; it was thought that the leeches had fallen off, been removed, or were completely thrown out due to bleeding. Additionally, clotted blood contamination was detected in the vagina. No free fluid was observed in Douglas. Vaginal bleeding foci were cauterized. After the procedure, the patient's vaginal bleeding stopped. The patient was discharged after being followed up in the hospital for 24 hours. The patient was advised to abstain from sexual intercourse for 4 weeks and to not use tampons to aid in the healing of the cervix. They were also advised to see their primary care physician for further evaluation and counseling regarding the use of traditional medicines.

Discussion

Genitourinary system and lower gastrointestinal system bleeding (GIS) are health problems frequently encountered as reasons for admission to the emergency department in hospitals. They (genitourinary system and lower gastrointestinal system bleeding) can be confused due to their anatomical proximity and misexpressed, and they have the potential to threaten life. Genital tract bleeding refers to bleeding from the uterus, cervix, vagina, and urethra. Lower gastrointestinal tract bleeding is considered bleeding distal to the ligament of Treitz (5, 6). Due to the proximity of the anatomical regions, patients may not be able to distinguish between genitourinary bleeding and GIS bleeding. In these patients, rectal examination (rectal touch) and vaginal examination make the correct diagnosis. Healthcare professionals should be able to differentiate between genitourinary system bleeding and GIS bleeding. Hypovolemic shock may develop as a result of genitourinary system bleeding and lower gastrointestinal system bleeding. If hypovolemic shock is not diagnosed and treated early, it increases the risk of mortality. Because of the risk of mortality, the evaluation, diagnosis, and treatment approaches for patients with bleeding in the emergency department are of particular importance.

Intravaginal leech application is a traditional medical practice that has been used for centuries in the treatment of various gynecological ailments. Leeches are believed to improve blood circulation and promote healing in the affected area (1, 7). However, this application may cause various complications, such as bleeding, infection, and allergic reactions. Vaginal bleeding following intravaginal leech application is a rare but serious complication that can lead to significant morbidity and mortality if not recognized and treated promptly (7). The exact mechanism of bleeding after intravaginal leech administration is not fully understood. It is believed that leeches can cause trauma to the vaginal mucosa, which can lead to bleeding. Due to the anticoagulant component of leech saliva, a leech bite may cause prolonged bleeding that cannot be stopped with compression (8, 9, 10). The risk of mortality increases in patients with a history of anticoagulant, antiplatelet, and non-steroidal anti-inflammatory drug (NSAID) use.

In the case report by Zengin et al. (11), it was noted that a patient with a leech bite had prolonged prothrombin time and activated partial thromboplastin time. In the case report by İkizceli et al. (11), a 19-year-old male patient developed bleeding that lasted up to 18 hours in the area where leech therapy was applied, but laboratory parameters such as

platelet count, prothrombin time, and activated partial thromboplastin time did not show any changes. Similarly, in our case, the platelet count, prothrombin time, and activated partial thromboplastin time were within normal limits. However, normal prothrombin and activated partial thromboplastin times may not exclude severe coagulation disorders. Hirudin and other bioactive substances secreted by leeches can impair coagulation without affecting the results of coagulation tests (1, 9).

The management of vaginal bleeding after intravaginal leech administration depends on the severity of the bleeding. Superficial skin bleeding can typically be stopped with short-term compression, unless the patient has a coagulation disorder or is taking anticoagulants. In mild cases, conservative treatment with observation, bed rest, and analgesia may suffice. However, in more severe cases, surgical interventions such as cautery, ligation, or hysterectomy may be necessary.

Güven et al. (13) stated that bleeding stopped after fresh frozen plasma and tranexamic acid treatment in a patient who presented with stage 3 shock due to bleeding after leech application. Cases benefiting from primary suture after local bleeding have been reported previously (12, 13). In our case, intravenous fluids (2000-3000 cc saline) were immediately started, and blood transfusion (erythrocyte suspension) was considered. Additionally, antibiotics and analgesics were given to prevent infection and alleviate pain. Vaginal bleeding foci were cauterized, and after the procedure, the patient's vaginal bleeding stopped. However, further studies are required to establish bleeding control after leech therapy.

Conclusion

Consequently, healthcare providers should be aware of the potential risks associated with traditional medical practices such as intravaginal leech administration. Patients should be educated about the potential risks and encouraged to seek medical attention if they experience any adverse effects after using bleeding-enhancing drugs or after applications such as intravaginal leeches. More research is necessary to better understand, prevent, and manage the mechanisms of complications associated with traditional medical practices and develop effective strategies.

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