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Contents

Case Report

Journal of Emergency Medicine Case Reports

Diffuse Axonal Damage and Status Epilepticus

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Abstract

Diffuse axonal injury (DAI), microscopic damage to axons in the brain neuralpath ways, corpus callosum, and brain stem, is associated with significant mortality and morbidity. Thetreatment of patients with DAI is geared to ward spreventing secondary injuries and facilitating rehabilitation. A 57-year-old male patient was brought to the emergency room by the EMS team with the complaint of seizures in the form of an incision on the scalp, change in consciousness, confusion and convulsions after he lost his head to a cutting tool at work. In his neurological examination, it was found that the pupils were isochoric, the patient had seizures repetitively every two minutes, his consciousness was confused, and he could not obey orders. The patient was intubated with the diagnosis of status epilepticus due to diffuse axonal damage after a sharp object injury and was followed up in the intensive care unit. The patient improved clinically after the intensive care unit and was discharged with a follow-up recommendation. The possibility of diffuse axonal damage due to the mechanism of the trauma should be considered in cases that are not of high severity and no etiology has been detected and presenting with post-traumati cunconsciousness.

Keywords: Diffuse axonal injury, trauma, seizure

Introduction

Epilepsy is one the diseases that frequently leads to disability and can affect individuals of all ages, races, social classes, and geographical regions (1). Diffuse axonal injury (DAI), microscopic damage to axons in the brain neuralpath ways, corpus callosum, and brain stem, is associated with significant mortality and morbidity. The occurrence of DAI depends on the mechanism of injury; it is more common in high-energy traumas, especially in traffic accidents (2-4). Diffuse axonal injury is defined clinically by comalasting 6 hours or longer after traumatic brain injury (TBI), excluding swelling or ischemic brain lesions (2). DAI is considered the most important factor in determining morbidity and mortality in TBI survivors and is the most common cause of post-traumatic coma, disability, and persistent near-vegetative state (2,3).

Outcome of patients after DAI correlated with the number of lesion sidentified by imaging. A longitudinal study analyzing the evolution of traumatic axonal injuryusing magnetic resonance imaging (MRI) in 58 patients with moderateor severe TBI showed that as the number of lesions observed early after trauma increases, so does impairment in functioning after 12 months (5). A study of 26 DAI patients showed that the volume and number of MRI-identified lesions performed within 48 hours of hospitalization were strongly associated with the level of disability observed at hospital discharge (6). Treatment of patients with DAI is geared towards preventing secondary injuries and facilitating rehabilitation. Secondary injuries and hypotension, edema, intracranial hypertension, and hypoxia are the leading causes of increased mortality. Therefore, emergency care is recommended to prevent hypotension, hypoxia, cerebral edema, and high intracranial pressure (ICP) (7).

Case

A 57-year-old male patient was brought to the emergency room by the EMS team with the complaint of seizures in the form of an incision on the scalp, change in consciousness, confusion and convulsions after he lost his headto a cuttingtool at work. The vital signs of the patient at the time of admission were fever 36.4 °C, pulse 110/min, respiratory rate 18/min, systolic blood pressure 180 mmHg, diastolic blood pressure 126 mmHg, blood sugar 136 mg/dL. The Glasgow Coma Score (GCS) of the patient at the time of admission was 12 (M:4 M:4 V:4). In the anamnesis, it was learned that the EMS team was informed about the patient,

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who caught his hair in a cuttingtool at work and sat on the ground after an incision on the scalp, and that he was conscious and normal at that time. The ambulance team reported that diazepam was administered to the patient, who started to have altered consciousness and seizures in the ambulance during his transfer.

In the physical examination of the patient, incisions containing the skin and subcutaneous tissue of medium depth, approximately 5 cm in length, with irregular paralel edges, were observed in the scalp area of the left parietal bone close to the occipital bone. No acute traumatic lesion was detected in any other part of the body in his external examination. In his neurological examination, it was found that the light reflex was positive bilaterally, the pupils were isochoric, the patient had convulsive seizures recurring every two minutes, his consciousness was confused, and he could not obey orders. When the GCS was re-evaluated, this time was calculated as 10 (E:4 M:4 V:2). No pathological findings were found in other system examinations (respiratory, cardiac, abdominal, extremity).

The incisions of the patient were closed with stapler and dressing was applied. Tetanus and antibiotic prophylaxis were performed. Brain computed tomography (CT) imaging was performed in the patient who was re-treated with diazepam and given a loading dose of levatiracetam despite his recurrent seizures. The patient, who seseizures continued after imaging, was intubated on a planned basis due to the risk of status epilepticus and aspiration. Diffusion magnetic resonance (MR) imaging and SWI imaging were applied to the patient who was followed up under phenytoin and midazolam infusion after intubation. No acute traumatic pathology was observed in the brain CT examination, except subcutaneous hematoma in the area of the incision (Figure-1). On the other hand, in diffusion MR imaging, an increase in intensity thickness compatible with posttraumatic hematoma in the subcutaneouss of planes in the left parietal and millimetric-sized no specific signal increases in both frontal subcortical white matter were detected (Figure-1). In the cranial SWI MR examination, linear hemorrhagic signal losses in the right frontobasal white matter were evaluated in favor of grade 1 diffuse axonal injury (Figure -2).

The patient was intubated and followed up in the intensive care unit with the diagnosis of status epilepticus due to diffuse axonal damage after a sharp object injury. The patient improved clinically after four days in the intensive care unit, and was discharged with a follow-up recommendation after being followed up in the service for four days.

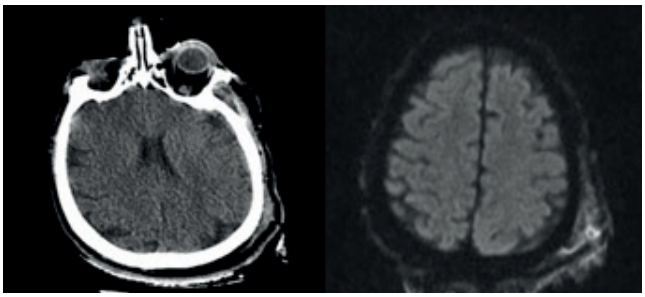

Figure 1. Subcutaneous hematoma in left parietooccipital on brain CT image, intensity thickness increase consistent with post-traumatic hematoma in the subcutaneouss oftplanes in the left parietal in diffusion MR examination

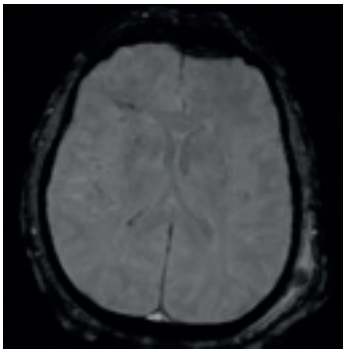

Figure 2. Cranial SWI MRI inthe right frontobasal linear hemorrhagic in white matter signal losses grade 1 diffuse compatible with axonal injury hypointense area

Discussion

Diffuse axonal injury (DAI) is a “hidden” pathology of traumatic brain injury (TBI). Although found throughout the white matter, it mainly contains microscopic damage, making it nearly invisible to current imaging techniques. As a classification, two general categories of brain trauma have emerged, defined as “focal” and “diffuse” brain injury (3,8,9). In our case, according to this classification, a focal brain injury can be mentioned when the results in the imaging findings and theclinic are examined.

In approximately 50% of cases, DAI occurs as a result of high-speed vehicle crashes, falls, and assaults (10,11,12). According to Moe et al, the same mechanisms of injury are observed in low-speed traumatic accidents such as sports injuries, falling from stairs or standing up, which can also lead to DAI (13). In our case, the etiology developed due to a sharp instrument injury, apart from other common causes. This showed that we should consider diffuse axonal damage in unexpected clinical situations.

According to the histopathological findings, Adams et al. classified DAI into three grades: Grade I–DAI with axonal lesions in the cerebral hemispheres; Grade II–DAI with focal axonal lesions in the corpus callosum; Grade III–DAI with focalor multiple axonal lesions in the brain stem (14). In the MRI examination of our case, it was evaluated as Grade I because axonal injury was detected in the right frontobasal white matter.

CT examination is stil the gold Standard for imaging DAI from an emergency stand point. CT scan may be negative or show typical DAI findings including multiple hemorrhagic lesions 5 to 15 mm in diameter at the gray-white matter

interface (15). MRI is the recommended tool for imaging DAI, but its usability is limited comparedto CT, especially in emergencies. According to Gentry et al, MRI may show diffuse, small, focal abnormalities confined to the white matter tracts. They tend to be multiple and non-hemorrhagic when present (16). In our case, there was no DAI finding on CT imaging, but axonal damage was detected on MRI imaging.

Conclusion

High-speed motor vehicle accidents are often involved in the etiology of diffuse axonal injury. The most common mechanism involves an accelerating and decelerating movement in the white matter path ways of the brain. It was note worthy that diffuse axonal damage, which we usually follow after or in association with hemorrhagic or ischemic cerebrovascular accident, did not accompany these diagnoses in our case. It comestomind that the patient in our case caused diffuse axonal damage during his rescue effort with a throwing motion after his hair was caught in the cuttingtool. As a result, the possibility of diffuse axonal damage due to the mechanism of the trauma should be considered in cases that are not of high severity and no etiology has been detected and presenting with post-traumatic unconsciousness.

Ethics

The case report has written in an anonymous characteristic, thus secretand 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

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Radiological Findings of Recurrent Pyogenic Cholangiopathy

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Abstract

Recurrent pyogenic cholangiopathy (RPC), formerly known as oriental cholangiopathy, is an entity characterized by stenosis of the intrahepatic and extrahepatic bile duct, formation of pigmented intraductal Stones and dilation. Our objective was to discuss the presence of recurrent pyogenic cholangiopathy in our present presentation with radiological images. RPC is a destructive disease caused by the intrahepatic and extrahepatic bile ducts, recurrent attacks of cholangitis, stasis in the bile ducts, formation of abscesses, strictures, and dilatations, as well as parasitic infections in etiology. Patients with RPC produce most commonly E. coli, pseudomonas, klebsiella, proteus species, and anaerobes in biliary cultures. Sonographic and CT findings include intrahepatic or extrahepatic duct stones, extrahepatic duct dilatation, relatively intermediate or intrahepatic duct dilatation, focal or large area bile duct dilatation, increased periportal echogenicity, segmental hepaticatrophy, and existinggall stones. Stones in the intrahepatic biliary tract can be removed by interventional radiology or surgery.

Keywords: Charcot trio, cholangiopathy, magnetic resonance imaging, pigmented stone, ultrasound

Introduction

Recurrent pyogenic cholangiopathy (RPC), formerly known as oriental cholangiopathy, is an entity characterized by intra and extrahepatic bile duct stenosis, intraductal pigmented stone formation, and dilatation (1). Disease can rarely be confused with malignancies in the bile ducts. Our objective was to discuss the presence of recurrent pyogenic cholangiopathy in our present presentation with radiological images.

Case

47-year-old patient; patient with known myelodysplastic syndrome and chronic renal failure has had a history of hospitalization several times in the last few months due to pain in the right upper quadrant, fever, jaundice (Charcot triad). She had been referred to our clinic with cholangitis and preliminary mass diagnosis at the external center, which she had previously referred to with similar complaints.

Ultrasonography revealed that the intrahepatic bile ducts were located in the central duct, proximal to the common bile duct, with an echogenic nodular appearance (palestones) causing moderate dilation in the intrahepatic bile ducts (Figure-1a, 1b).

Discussion

RPC is a destructive disease caused by the intrahepatic and extrahepatic bile ducts, recurrent attacks of cholangitis, stasis in the bile ducts, formation of abscesses, strictures, and dilatations, as well as parasitic infections in etiology. In Singapore, Japan, and Hong Kong, 2-5% of biliary stone diseases are associated with RPC (2). RPC is

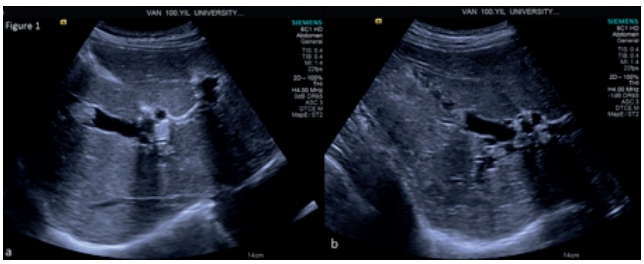

Figure 1a, 1b. Ultrasonography revealed that the intrahepatic bile ducts were located in the central duct, proximal to the common bile duct, with an echogenic nodular appearance (palestones) causing moderate dilation in the intrahepatic bile ducts.

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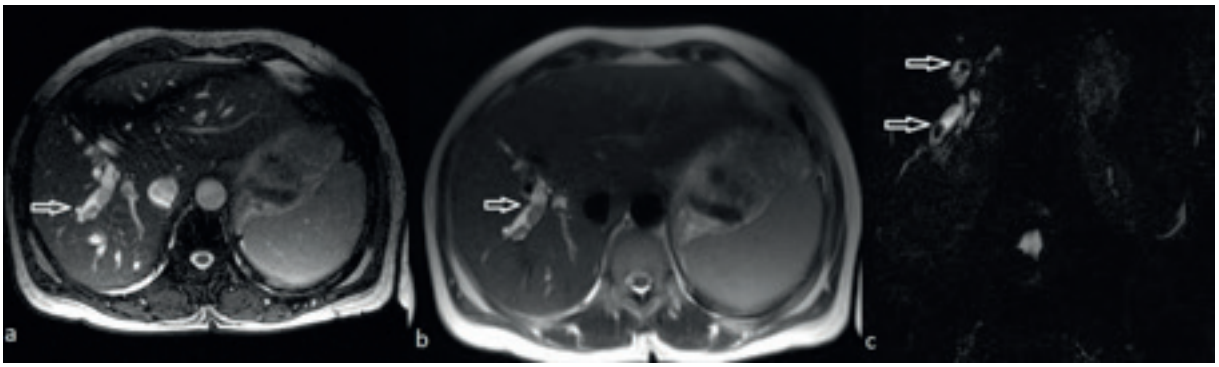

Figure 2a, 2b, 2c. Magnetic resonance images; intrahepatic bile ducts are dilated, hyperintense, and hypointense nodular images are observed in the lumen.

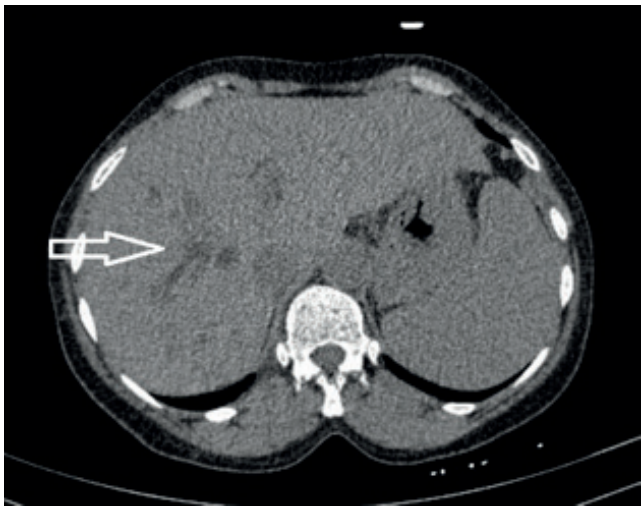

Figure 3. The unenhanced thorax tomography images of the cross section showed a slight dilatation of the intrahepatic bile ducts in the right lobe of the liver.

characterized by recurrent episodes of abdominal pain, fever, and jaundice (Charcot triad) involving dilatation of the intra and extrahepatic channels and the feather with soft, pigmented stones. The cause of the disease is unknown, but the socioeconomic status, which is malnutrition, is more common in societies with low levels (3). The geographical coexistence of parasitic infection with recurrent pyogenic cholangitic biliary tract infection in regions where parasites are endemic should suggest that biliary tract parasitic infection plays a role in its pathogenesis. The three liver trematodes that most commonly infect humans are Clonorchis sinensis, Opisthorchis species and Fasciolahepatica. These organisms can cause disease by causing epithelial damage or bile duct obstruction in the biliary tract (4). Patients with RPC most commonly produce E. coli, pseudomonas, klebsiella, proteus species, and anaerobes in biliary cultures 4. Sonographicand CT findings include intrahepatic or extrahepatic duct stones, extrahepatic duct dilatation, relatively moderate or intrahepatic duct dilatation, localized dilatation of lobar or segmental bile ducts, increased periportal echogenicity, segmental hepaticatrophy, and bile stones as currently present (5).

Conclusion

Interventional radiologyplays an important role in percutaneous biliary drainage of the affected liver segment, removal of pigment stones, balloon dilation of bile duct strictures, and repeated percutaneous procedures to clear pigment Stones and sludge-like bile deposits. Lobectomy or segmentectomy operations can be performed when surgery is appropriate. Radiologists should keep RPC in mind because it can be mixed with tumors of the biliary tract.

Data Availability

The data used to support this study are available from the corresponding author upon reques

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Consent

Written informed consent was obtained from all participants involved in this study.

Conflict of Interest

The author declares that the investigation was conducted in the absence of any commercial or financial relationship that could be construed as a potential conflict of interest.

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Case Report

Journal of Emergency Medicine Case Reports

5 mm Before Death: Important of Transport in Thoracic Stab Wounds

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Abstract

Penetrating thoracic injury is a major medical problem due to the high mortality incidence in thoracic surgery practice. Proper and effective resuscitation and prompt transfer decreases mortality in this patients. In this study, we presented a patient who was injured with a bread knife. After transportation was provided in a seated position for 4 hours, the bread knife, which was located between the esophagus and the aorta, was removed via right thoracotomy, followed by intubation in the same position.

Keywords: Thoracic, trauma, penetrating, death, transport

Introduction

Penetrating thoracic injury is a major medical problem due to the high mortality incidence in thoracic surgery practice. A lot of patients dieshortly after the incident due to major cardiac and large vessel injuries (1,2). One of the most important factor reducing mortality in patients is proper and effective resuscitation and prompt transfer (3). Additionally, if the object causing the penetrating injury, such as an ironrod, knife, glass, etc., is stil present in the thorax, it should be removed in a controlled manner through exploration via thoracatomy and/or sternotomy. Furthermore, utmost care should be taken during the transport of these patients, and appropriate positioning should be ensured based on the position of the sharp penetrating object. Communication between the team members who first encounter the patient and those who will provide treatment is crucial in this situation (4).

In this study, the aim is topresent a patient who was injured with a bread knife. After transportation was provided in a seated position for 4 hours, the bread knife, which was located between the esophagus and the aorta, was removed via right thoracotomy, followed by intubation in the same position.

Case

It was learned that a 49-year-old female patient was injured in the back with a bread knife, resulting in stable vital signs and a Glasgow Coma Scalescore of 15. It was suggested that the patient be transported without changing position. Four hours later, the patient, seen in the emergency department, was found to have the bread knife lodged in the medial corner of the right scapula and the 6th intercostal space on the back (Figure-1). On thoracic computed tomography (in the lateral decubitus position), bread knife was located approximately 5 mm away from the aorta and esophagus was observed (Figure-2). The patient was intubated in the same position and then placed in the left lateral decubitus position (Figure-3). Upon exploration via right thoracotomy, after opening the mediastinal pleura, the tip of the bread knife was palpated between the esophagus and the aorta at the level of the azygos vein. The aorta and esophagus were lateral lydeviated, and the cutting tool was carefully removed with controlled traction (Figure-4). Subsequently, the patient, without any additional issues, was hospitalized to our clinic forfollow-up. The chest tube was removed on postoperative (PO) day 1. On PO day 2, the patient was discharged.

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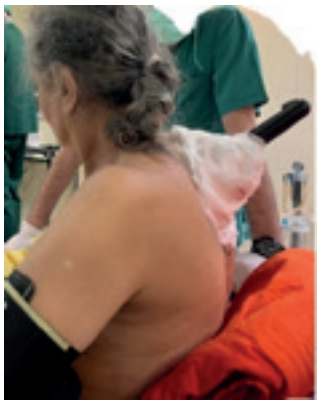

Figure 1. Patient's transfer position

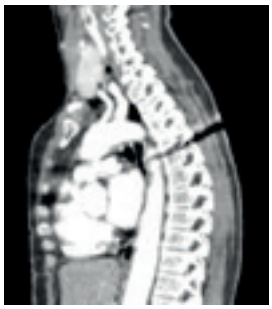

Figure 2. Bread knife between the aorta and esophagus on thoracic tomography.

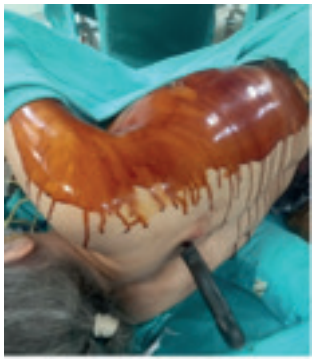

Figure 3. Patient was intubated in a seated position and transitioned to lateral decubitus position.

Figure 4. Bread knife removed by thoracotomy

Discussion

The World Health Organisation reports that more than 1.3 million people die on road severy year and as many as 50 million others are injured (5). Only a small fraction, approximately 4%, of thoracic traumas involve injuries to thoracic vessels, such as the aorta, innominate vein sandartery, left carotid artery, internal mammaries, and pulmonary hilar vessels. Proper and effective prehospital treatment management have contributed to an increase in the number of patients surviving in the field and reaching the hospital despite life-threatening vascular injuries(3).

In the sepatients group, pre-hospital care (patient transport, emergency management (ABC etc) significantly enhances the prognosis of critical emergency situations. Simply having Access to pre-hospital care leads to a 25% decrease in mortality rates, and this effect is even more pronounced when coupled with timely transportation to emergency facilities (6). Swift evaluation during the initial assessment and interventions to ensure airway, breathing, and circulation are maintained can be crucial for saving lives. The patient's hemodynamic condition often guides the extent of the initial evaluation and determines whether immediate surgical intervention is necessary (7). In patients who need a long-distance transfer, the most important points are the most proper transport choice for patients, the patient's level of discomfort, joruney's time and the likelihood that the patient will survive the journey. And perhaps the most important point is the accurate and effective communication between the transferring team and the team providing definitive care (6). During the transfer of our patient, continuous communication has been maintained between the team providing initial emergency treatment to the patient and us until the patient is brought to our hospital. Our patient remained in a seated position from the initial intervention until arrival at the operating room.

Chest traumas caused by penetrating sharp instrument spose a high risk to life. In many patients, the foreign object causing the injury is not in the thorax. Rarely, as in the case of our patient, the instrument causing the injury is in the thorax when patients are brought to the emergency department. In both scenarios, the clinical condition should be rapidly assessed, and necessary interventions should be promptly performed. If the patient is not stable hemodynamically and/or respiratorily, surgery should be initiated based on clinical evaluational one without imaging modalities (1,2). Despite our patient being hemodynamically stable, emergency thoracotomy was performed due to the proximity of the blade to vital organs such as the aorta and esophagus, without additional imaging. The blade was carefully withdrawn between the aorta and esophagus under direct exploration.

Conclusion

Patients with penetrating thoracic and/or mediastinal trauma should be taken to surgery within minutes without

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waiting for any imaging method. Thoracotomy is the most appropriate and safe treatment option for these patients. Additionally, in cases of stabwounds penetrating the thorax and/or mediastinum, extreme caution should be exercised during the transfer of these patients due to the possibility of vital organ injury, and the cutting tool should be removed in a controlled mannervia thoracotomy or sternotomy.

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Case Report

Journal of Emergency Medicine Case Reports

A Fatal Complication of Tracheostomy Cannula Exchange: Bilateral Pneumothorax, Pneumomediastinum, and Subcutaneous Emphysema

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Abstract

Although tracheostomy opening or replacement is generally a safe procedure, it carries varying risks of complications. Serious complications include false lumen formation between tissues surrounding the trachea, tracheal posterior wall damage, bleeding, pneumomediastinum, pneumothorax, and death. The incidence of these complications increases especially in patients who are obese, have a shortneck or anatomical deformity. A 24-year-old male patient was brought to the emergency department with respiratory distress. His family stated that his complaints started four days ago after the change of the tracheostomy cannula in a hospital. Computed tomography of the neck and chest revealed subcutaneous emphysema in the neck and anterior chest, free air between deep tissues in the neck, pneumomediastinum and bilateral pneumothorax. Supportive oxygen therapy was started and the patient was consulted for thoracic surgery. After tube thoracostomy of the right hemithorax, intensive care unit hospitalization was performed. The patient died on the 25 th day of intensive care unit hospitalization due to type 2 respiratory failure. In patients with tracheostomy presenting to the emergency department with dyspnea, cannula-related causes should also be considered in the differential diagnosis.

Keywords: Bilateral pneumothorax, tracheostomy, complication, subcutaneous emphysema, pneumomediastinum

Introduction

Tracheostomy is the permanent or temporary opening of the anterior wall of the trachea to the skin by surgical or percutaneous methods. It is performed to relieve upper airway obstruction, facilitate weaning from the mechanical ventilator, relieve the need for prolonged mechanical ventilation and aspirate respiratory tract secretions (1). Tracheostomy tubes are recommended to be replace devery 1-6 months due to bacterial biofilm formation, stromal granulation tissue formation and deterioration in their physical structure (2).

Although tracheostomy opening or replacement is generally a safe procedure, it carries varying risks of complications. Serious complications include false lumen formation between tissues surrounding the trachea, tracheal posterior wall damage, bleeding, pneumomediastinum, pneumothorax and death. The incidence of these complications increases especially in patients who are obese, have a short neck or anatomical deformity (3,4).

In this article, we present a case who presented to the emergency department with respiratory distress four days after tracheostomy cannula exchange and was found to have subcutaneous emphysema, pneumomediastinum and bilateral pneumothorax.

Case

A 24-year-old male patient was brought to the emergency department with respiratory distress. His family stated that his complaints started four days ago after the change of the tracheostomy cannula in a hospital and he was brought to the emergency department after pulse rate was 150 beats/min and oxygen saturation was 65% in room air when measured by pulse-oximetry at home. In his medical history, it was reported that he had cerebralpalsy (CP), regularly used haloperidol drops, and his tracheostomy cannula was changed at regular intervals. It was stated that he had an oxygen concentrator at home but no mechanical ventilator. His family history was unremarkable.

At the time of presentation to the emergency department, blood pressure was 120/80 mmHg, pulse rate was 155 beats/min, body temperature was 36.7 °C, respiratory rate was 30 breaths/min. His oxygen saturation was 65% in room air and 91% under oxygen with 4 lt/min nasal cannula. Cooperation was limited due to CP. Inspection revealed cervical and thoracic deformity due to CP, subcutaneous crepitation on the neck and anterior chest surface on palpation, and decreased breath sounds,rales, and wheezing in both lungs on auscultation. No other pathologic findings were found during the physical examination.

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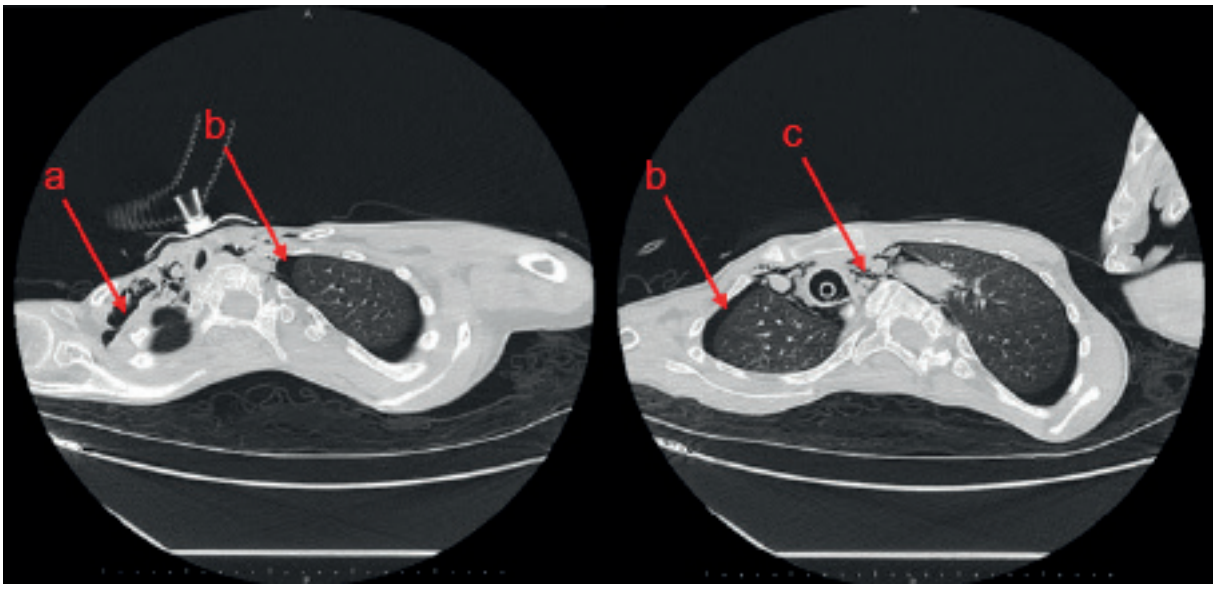

Figure 1. Computed tomography images showing pathologic findings in the neck and chest (a: subcutaneous emphysema; b: pneumothorax; c: pneumomediastinum)

Biochemical parameters and complete blood count were normal except for increased white blood cell count. Venous blood gasshowed compensated respiratory acidosis. Computed tomography of the neck and chest revealed subcutaneous emphysema in the neck and anterior chest, free air between deep tissues in the neck, pneumomediastinum and bilateral pneumothorax (Figure-1).

Discussion

In patients with tracheostomy presenting to the emergency department with respiratory distress, all causes related or unrelated to tracheostomy should be considered in the differential diagnosis. Obstructive causes such as asthma attack, exacerbation of chronic obstructive pulmonary disease and anaphylaxis, cardiopulmonary causes such as pneumonia, abscess, empyema, pneumothorax, pleural effusion, acute decompensated heart failure, cardiac tamponade, pulmonary embolism, acute coronary syndrome, bradyarrhythmia, tachyarrhythmia should be excluded in all patients presenting with respiratory distress. In patients with tracheostomy, in addition to these, causes such as dislocation or displacement of the cannula, obstruction due to mucus or blood plug, deflation of the cuff, and ventilator-related causes if connected to a mechanical ventilator should be excluded (5,6).

Complications related to this procedure should be evaluated in patients who have recently had a tracheostomy or tracheostomy cannula replaced and who develop respiratory distress. Especially in obese patients, patients with short neck or patients with tracheal deviation, respiratory distress may develop due to pneumothorax and pneumomediastinum due to tracheal wall damage or false lumen formation during tracheostomy (7). In cannula exchange performed in recently opened tracheostomies, the risk of developing these complications is higher because the tracheocutaneous tract is not fully organized and the surrounding soft tissue is edematous (8).

Taking various precautions while opening the tracheostomy may decrease the complication rates. Effective sedoanalgesia and neuromuscular blockade should be applied to prevent coughing and neck movements during the procedure. If the incision in the anterior neck wall is too small, it may cause damage to the posterior wall of the trachea, leading to pneumothorax and pneumomediastinum. Using a flexible fiberoptic bronchoscope (or laryngoscope) during the procedure reduces the risk of posterior tracheal wall damage and complications. The use of bedside ultrasound during the procedure helps protect vascular structures and prevent tube misplacement. Ventilation, capnography and peak airway pressures should be checked after the procedure. Cannula placement should be checked with a fiberoptic bronchoscope (or laryngoscope), ultrasound, or chest radiography. It should be seen that the tube is open and not obstructed by blood clots or secretions (9).

Pneumothorax is a life-threatening condition that causes respiratory distress. Spontaneous, traumatic or iatrogenic pneumothoraxes are seen bilaterally in only 1% of cases. Bilateral iatrogenic pneumothorax is much rarer. Although computed tomography is the gold standard for detecting pneumothorax after the procedure, chest radiography and bedside ultrasound may also be helpful (9, 10).

The causes of bilateral iatrogenic pneumothorax include intubation, central venous cannulation, tracheobronchial treebiopsy and tracheostomy opening (10). Treatment options for iatrogenic pneumothorax include observation, oxygen, needle or catheter aspiration and tube thoracostomy. Pneumothoraxes that are stable and less than 20% of the lung

volume are suitable for oxygen therapy and observation. Aspiration and tube thoracostomy should be performed in tension pneumothoraxes, pneumothoraxes larger than 2 cm, and cases with a high probability of recurrence and air leakage (11).

Although the treatment of pneumomediastinum is controversial, supportive care, including bed rest, pain control, and stopping oral intake to prevent esophageal rupture, is recommended (12).

Conclusion

Although tracheostomy opening or cannula exchange is generally a safe procedure, it may cause life-threatening complications such as pneumothorax. It should be kept in mind that the complications of tracheostomy opening or cannula exchange are higher in the presence of cervical and thoracic deformity and tracheal deviation as in the patient we presented. In patients with tracheostomy presenting to the emergency department with dyspnea, cannula-related causes should also be considered in the differential diagnosis.

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Case Report

Journal of Emergency Medicine Case Reports

A Case of Severe Tracheal Stenosis After Short-Duration Endotracheal Intubation

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Abstract

Post-intubation tracheal stenosis is a rare but severe condition that occurs after long-term intubation. In this study, we highlight the case of a male patient who experienced severe tracheal stenosis after a brief period of endotracheal intubation. A 37-year-old man presented to the emergency department complaining of difficulty breathing with stridor; upon examination, the patient was noted to have stridor during both inspiration and expiration. It was discovered that ten weeks ago, he was admitted to the hospital following a motor vehicle accident and required mechanical ventilation for nine days. Given this medical history, a contrast-enhanced CT imaging of the neck was performed, which revealed grade III tracheal stenosis. The patient was transferred to a specialized center, where he underwent successful tracheal stenting. This case report underscores the importance of inquiring about tracheal intubation history in patients who present to the emergency department with respiratory symptoms.

Keywords: Tracheal stenosis, endotracheal intubation, stridor, medical history

Introduction

Tracheal stenosis is a medical condition characterized by the abnormal narrowing of the trachea, often as a complication following intubation. Research indicates that tracheal stenosis post-intubation can vary from 10% to 22%, with severe symptoms occurring in only 1% to 2% of cases (1). The etiology of post-intubation tracheal stenosis is multifaceted. Notably, elevated cuff pressures surpassing the mucosal capillary perfusion pressure (20–30 mm Hg) are a significant contributor. This can precipitate ischemic insult within as little as fifteen minutes post-intubation, contributing to ulceration and subsequent fibrotic changes within a period of three to six weeks (2). The condition typically manifests as a gradual onset of dyspnea and dry cough, with additional symptoms including shortness of breath, difficulty speaking, and stridor (3). Here, we present a case study detailing the experience of a male patient who developed severe tracheal stenosis following a brief period of endotracheal intubation.

Case

A 37-year-old man presented to the emergency department with complaints of experiencing difficulty in breathing accompanied by stridor on inhalation and exhalation. Upon examination, it was noted that the patient who had stridor was facing challenges in both inspiration and expiration and was in moderate respiratory distress, with limited ability to speak. The patient reported a history of being hospitalized ten weeks prior due to a car accident, during which he required invasive mechanical ventilation for a period of 9 days. Considering this statement, the patient's hospitalization history was accessed from the personal health record system (e-Nabız, Republic of Türkiye Ministry of Health) profile by the patient himself. It was learned that the patient was intubated with a cuffed endotracheal tube (ETT) size of 8.0 mm. The patient denied experiencing any complications during intubation and stated that this was his only encounter with a ventilator. Upon admission, the patient's peripheral oxygen saturation (SpO2) was measured at 91% on room air. The initial course of treatment involved

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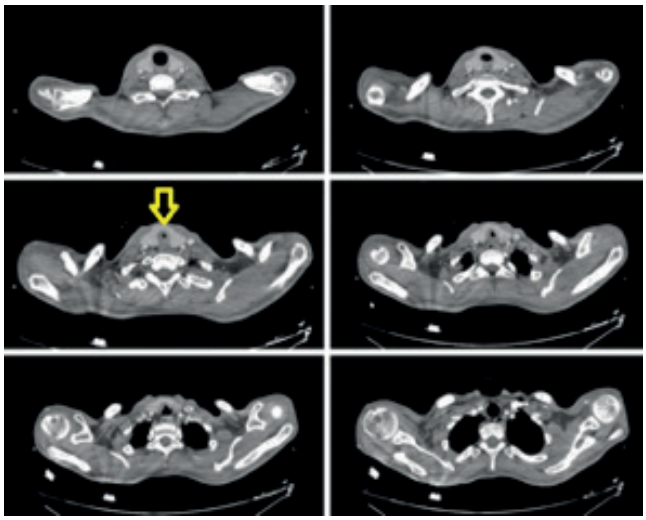

Figure 1. A contrast-enhanced CT imaging of the neck

the administration of nebulized salbutamol, adrenaline, intravenous dexamethasone, and antibiotics. As the patient's SpO2 levels improved to above 95%, a contrast-enhanced CT imaging of the neck revealed a 3 cm long tracheal stenosis located 5 cm below the vocal cords, with a minimum diameter of 3.3 mm (Figure-1). Due to the unavailability of a tracheoscopy, the patient was transferred to the intensive care unit and subsequently to an advanced medical center. A tracheoscopy performed at the advanced medical center revealed severe narrowing, categorized as Grade III, with a reduction of the tracheal lumen exceeding 71%. The stenosis was successfully managed with tracheal stenting.

Discussion

Post-intubation tracheal stenosis, a rare yet severe condition, arises subsequent to prolonged intubation and can lead to significant implications. This stenosis results from compression by the ETT cuff, causing ischemia in the tracheal mucosal tissue and subsequent fibrotic scarring. Ramalingam et al. have identified multiple factors contributing to tracheal stenosis, includingthe patient's age and sex, steroid use, ETT size relative to the tracheal lumen, ETT material, intubation duration, hemodynamic state, and tube movement (3). Although information on cuff pressure is not available, the size of the ETT may be relatively disproportionate, explaining the tracheal stenosis in our case. Frioui et al. reported an estimated incidence of 4.9 cases of tracheal stenosis per million post-intubation (4). The onset of tracheal stenosis typically occurs 2 to 24 weeks after extubation. The likelihood of stenosis increases with more prolonged intubation, but it is rare when intubation lasts less than one week (5, 6). In our case, tracheal stenosis emerged following a 9-day intubation period, similar to cases considered rare in the literature. There are a few reported cases in the literature where post-intubation tracheal stenosis took 28 days to 6 months to develop (7). Following endotracheal intubation, it is common for most patients to experience a certain level of stenosis. Interestingly, patients with this condition often do not display symptoms until the tracheal narrowing reaches approximately 70% of its original lumen (8, 9). In a study conducted by Stauffer JL et al., it was noted that 11% of patients subjected to intubation using high-volume, low-pressure cuffed tubes exhibited tracheal stenosis, manifesting as a 50–90% constriction of the tracheal lumen, specifically at the cuff site (10). In our case, there was grade III tracheal stenosis, in which the tracheal lumen reduction is between 71% and 99%, which explains the respiratory symptoms. Diagnosing tracheal stenosis can be challenging, necessitating a multidisciplinary approach to management. Bronchoscopy serves as the gold standard for diagnosis, with computed tomography scans also proving beneficial. Stenosis can typically be prevented by employing low-pressure cuffs. The available treatment modalities encompass surgical tracheal reconstruction, tracheal dilation, stenting, and laser bronchoscopy (3). These therapeutic options are integral to addressing tracheal pathologies and necessitate careful consideration based on individual patient presentations.Our case is significant as it exemplifies Grade III tracheal stenosis despite a brief intubation period. Furthermore, it serves as a compelling example of the critical necessity to promptly address acute respiratory distress, conduct thorough medical history assessments, minimize unnecessary tests, and concentrate on targeted examinations and interventions.

Conclusion

In order to provide optimal patient care, a thorough assessment of any prior intubation history is imperative when patients present to the emergency department with respiratory symptoms. Additionally, it is vital to include the possibility of tracheal stenosis in the differential diagnosis for patients with a history of intubation who exhibit respiratory symptoms upon presentation to the emergency department.

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Case Report

Journal of Emergency Medicine Case Reports

A Case of Takotsubo Cardiomyopathy

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Abstract

Takotsubo cardiomyopathy (TCM), also called stress cardiomyopathy, broken heart syndrome and apical ballooning syndrome, is a heart problem that ocurs after intense stress, sadness or shock. Takotsubo cardiomyopathy usually presents with chest pain and dyspnea and is similar to the diagnosis of acute coronary syndrome. Echocardiography and coronary angiography have an important role in the diagnosis of Takotsubo cardiomyopathy. In this case report, we wanted to investigate Takotsubo cardiomyopathy in a 59-year-old woman who presented to the emergency department.

Keywords: Takotsubo cardiomyopathy, broken heart syndrome, acute coronary syndrome

Introduction

Takotsubo cardiomyopathy (TCM) is a type of cardiomyopathy without severe coronary obstruction characterised by transient systolic dysfunction of the apical and midsegment of the left ventricle, typically in the presence of emotional or physical stress (1-3). The disease is called Tako-tsubo because the ventricular appearance of the heart in stress, sadness or shock resembles a earthen ware pot. The incidence of TCM has been determined to be approximately 2% in all patients presenting with suspicion of acute coronary syndrome (3,4). TCM, which is mostly known as the disease of middle-aged postmenopausal women, can be determined by seriouse motional or physical stress experienced when the anamnesis is deepened (2,3). However, it can also develop in the absence of stress in men under the age of 50 (2). Echocardiography, one of the imaging methods, is very important in detecting left ventricular wall motion disorder with typical apical ballooning in the diagnosis of TCM (2). The definitive diagnosis for TCM is based on the typical appearance and the absence of severe coronary artery disease (2,3). The drugs recommended for treatment are Standard heart failure drugs that contribute to left ventricular remodeling. In this study, we described the case of a 59-year-old elderly female patient who was admitted to the emergency department with complaints of chest pain and was hospitalized with a diagnosis of acute coronary syndrome, but later we detected TCM.

Case

A 59-year-old female patient, who had previously been monitored for diabetes and hypertension, was admitted to the emergency department with chest pain that started 2 hours ago and worsened half an hour ago. In the patient's vitals, blood pressure: 140/80 mm/Hg, finger tip blood sugar: 156 mg/dl, saturation (spO2) 93%, pulse: 67 beats/min. The patient's electrocardiography (ECG) showed elevation in V1-V6, D1-aVL. The patient was consulted by a cardiologist and hospitalised in the coronary intensive careunit with a diagnosis of diffuse anterior MI. ECG was performed again and echocardiography (TTE) evaluation showed apex akinetic ejection fraction: 30% and moderate mitral insufficiency. There was no pericardial effusion. Antiaggregant and anticoagulant treatment was initiated because of the diagnosis of ST elevation MI. The patient was immediately taken to the catheterisation laboratory and coronary angiography showed normal left anterior descending artery (LAD), circumflexartery (Cx) and right coronary artery (RCA). In the light of these findings, the patient was diagnosedas Takotsubo (Apical Ballooning) Syndrome (Figure-1 and 2).

Discussion

TCM is considered a newtype of cardiomyopathy, occurring in most women over the age of 58 (2,5). Previous studies have

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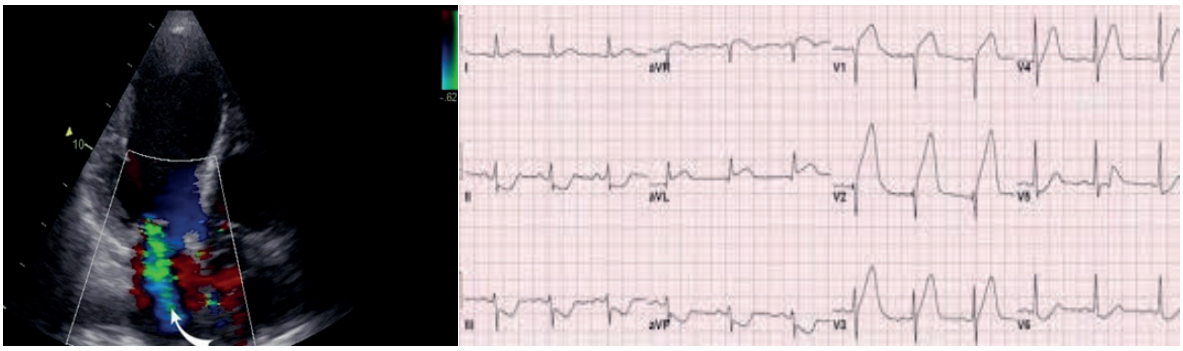

Figure 1. Transthoracic echocardiography and electrocardiography of the patient taken in the cardiology department

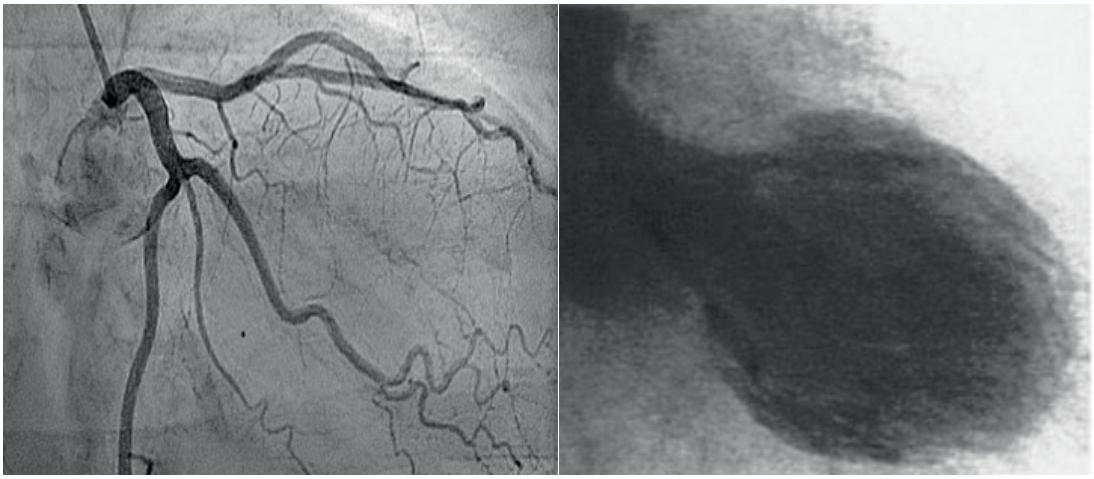

Figure 2. Typical apical ballooning seen on ventriculography

shown that 5% of women suspected of having a heart attack actually have the condition (5). Although the prognosis of TCM is generally good, the mortality rate has been reported to vary between 2% and 4% in different countries (6). Electrocardiograms (ECG) of patients presenting with chest pain and dyspnoea usually show ST elevation, T negativity or ST-T changes in precordial derivations consistent with acute coronary syndrome (2,3). The most common ECG finding in TCM is ST elevation in precordial derivations. However, this elevation is not as high as in acute myocardial infarction. Normal ECG or non-specific ECG changes can be seen in approximately 17% of patients (2). Therefore, transthoracic echo-cardiography (TTE) plays a very important role in the diagnosis, detection of complications of TCM and shaping the treatment (2). Although the pathophysiology of TCM is not known with certainty, catecholamine discharge and microvascular dysfunction are currently the most accepted theories (7). Catecholamine levels of patients diagnosed with TCM were 2-3 times higher than those of patients with myocardial infarction (7). It has been reported that this may be related to excessive catecholamine secretion due to stress (8). Estrogen has an important role in all eviating oxidative stress, affecting sympathetic neuromodulation, decreasing the sensitivity of β-receptors and improving endothelial function (9). Therefore, the reason for the common occurrence of TCM in female patients may be related to the decreasing estrogen level due to the post menopausal period.

Conclusions

There has been a significant increase in the number of TCM cases in our country in recent years. In particular, the importance of early diagnosis has increased significantly due to the fact that elderly patients may present with very different symptoms, can easily be confused with acute coronary syndromes, and may lead to death and other complications. Evaluation with TTE of patients with ST segment elevation detected on ECG in the emergency department before emergency coronary angiography allows the detection of diseases that may be confused with acute coronary syndrome such as TCM. In addition to the typical TCM appearance, the detection of intraventricular thrombus and the ability to evaluate right and left ventricular functions increase the importance of TTE.

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Case Report

Journal of Emergency Medicine Case Reports

Post-Myocardial Infarction Complications: A Case of Cardiac Tamponade Secondary to Dressler's Syndrome

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Abstract

Cardiac tamponade, a rare but life-threatening complication following myocardial infarction (MI), can manifest acutely and necessitate urgent intervention. In this case report, we present the clinical course of a 47-year-old male who developed cardiac tamponade secondary to Dressler's syndrome three weeks after a non-ST-elevation myocardial infarction (NSTEMI). The patient presented with epigastric pain, a syncopal episode, and hypotension. Bedside echocardiography revealed a massive pericardial effusion with signs of tamponade physiology. We promptly performed emergency pericardiocentesis, which led to significant clinical improvement. Early recognition and management are crucial to preventing life-threatening complications.

Keywords: Cardiac tamponade, dressler syndrome, pericardiocentesis

Introduction

Dressler syndrome, also known as post-myocardial infarction syndrome, is characterized by aseptic pericarditis that develops following a documented cardiac injury (1). Historically, the occurrence of Dressler syndrome with acute myocardial infarction (AMI) varied between 3% and 4% before the widespread use of coronary revascularization. However, with advancements in early reperfusion therapy and immune-modulatory drugs, the incidence of Dressler syndrome has decreased significantly to less than 0.1% (1, 2).

Cardiac tamponade, although uncommon in the context of Dressler syndrome, can have lethal consequences if not promptly recognized and treated. The accumulation of pericardial fluid can lead to obstructive shock and cardiovascular collapse. Therefore, maintaining a high index of suspicion, conducting thorough clinical assessment, and utilizing ultrasound in emergency settings are essential for timely diagnosis. Emergent pericardiocentesis remains a life-saving intervention.

Only a few case studies of Dressler syndrome complicated with cardiac tamponade have appeared in recent years. This case report highlights the clinical presentation, diagnostic challenges, and therapeutic approach for a patient who developed cardiac tamponade due to Dressler syndrome post-NSTEMI, emphasizing the importance of vigilance in post-MI care.

Case

A 47-year-old Malay male with pre-existing hypertension and ischemic heart disease was transferred from a private hospital to our emergency department for acute myocardial infarction (AMI) at midnight. He presented to the ED after three hours of epigastric pain, accompanied by a syncopal attack. Three weeks prior, he had a history of NSTEMI. During the previous admission, an echocardiogram revealed a left ventricle ejection fraction of 45% with apical septal and anterior hypokinesia but no left ventricle thrombus or pericardial effusion. A subsequent coronary angiography conducted two weeks ago revealed single-vessel disease with chronic total occlusion of the mid-left anterior descending artery.

During the presentation, the patient experienced severe, crushing epigastric pain while at rest, radiating to both shoulders and accompanied by diaphoresis and dyspnoea. His wife discovered his syncopal episode while he was using the toilet and promptly took him to a private hospital. He regained consciousness after 20 minutes. Aside from these

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symptoms, he reported no palpitations, lower limb swelling, headaches, dizziness, fever, upper respiratory tract infection symptoms, or gastroenteritis symptoms. He was treated with antiplatelets for AMI because of ST elevation in ECG leads V2-V6 and was referred to our ED for further management.

Upon arrival at our ED, the patient was alert and conscious, experiencing moderate pain, rated at four on the pain scale, and had no signs of tachypnoea. Physical examination revealed a blood pressure of 100/60 mmHg without inter-arm discrepancy, a heart rate of 150 bpm characterized by moderate pulse volume and irregularly irregular pulse, SPo2 of 95% on room air, and a temperature of 37 °C. The cardiovascular examination revealed a muffled heart sound with a dual rhythm, no murmurs, pericardial rubs, or prominently distended neck veins. Other systemic examinations were unremarkable.

The cardiac monitor detected paroxysmal atrial fibrillation, with an initial ECG showing a fast ventricular response and aberrant conduction or ventricular premature complexes. Subsequent ECGs revealed sinus tachycardia, low voltage limb leads, Q wave and ST elevation over anterior leads (V1–5), and T wave inversion over precordial leads (V2–6) (Figure-1). Bedside cardiac ultrasound identified an aortic root measuring 3.3 cm with no intimal flap seen, massive pericardial effusion (maximum thickness of 3 cm), and right ventricular collapse during diastole (Figure-2). Due to persistently low blood pressure and evidence of cardiac tamponade, an emergency bedside ultrasound-guided subxiphoid pericardiocentesis was performed, draining 100 cc of haemorrhagic pericardial fluid.

Post-pericardiocentesis, vital signs improved, with blood pressure reaching 111/63 mmHg and the heart rate reducing to 83 bpm. A chest X-ray revealed cardiomegaly and an urgent chest CT angiography (CTA) confirmed the presence of a large pericardial effusion measuring 2.8 cm in maximum thickness, along with the collapse of the right ventricle relative to the left ventricle, suggestive of the tamponade effect (Figure 3). Laboratory tests showed a borderline high white blood cell count, thrombocytosis, elevated infective and cardiac markers, transaminitis, and acute kidney injury. However, pericardial fluid analysis ruled out infection and malignancy.

The cardiology team admitted him to the CCU with a diagnosis of pericarditis-induced pericardial effusion post-NSTEMI, likely Dressler syndrome. A formal inpatient echocardiogram revealed global pericardial effusion with clot formation seen over the left ventricular apex, hypokinesia over the apical wall, and a moderate ejection fraction (40–50%). Treatment included anti-inflammatory drugs (Aspirin 750 mg TID, Colchicine 0.5 mg BID) and oral anticoagulants (Warfarin). The patient remained hemodynamically stable during the two-week hospitalization with no bleeding tendencies or other active complaints. Serial bedside echocardiograms demonstrated a reduction in the size of the pericardial effusion. The patient was discharged with nine months of dual therapy (Aspirin plus Warfarin) and scheduled for follow-up in the cardiac clinic as an outpatient. Written informed consent was taken from the patient.

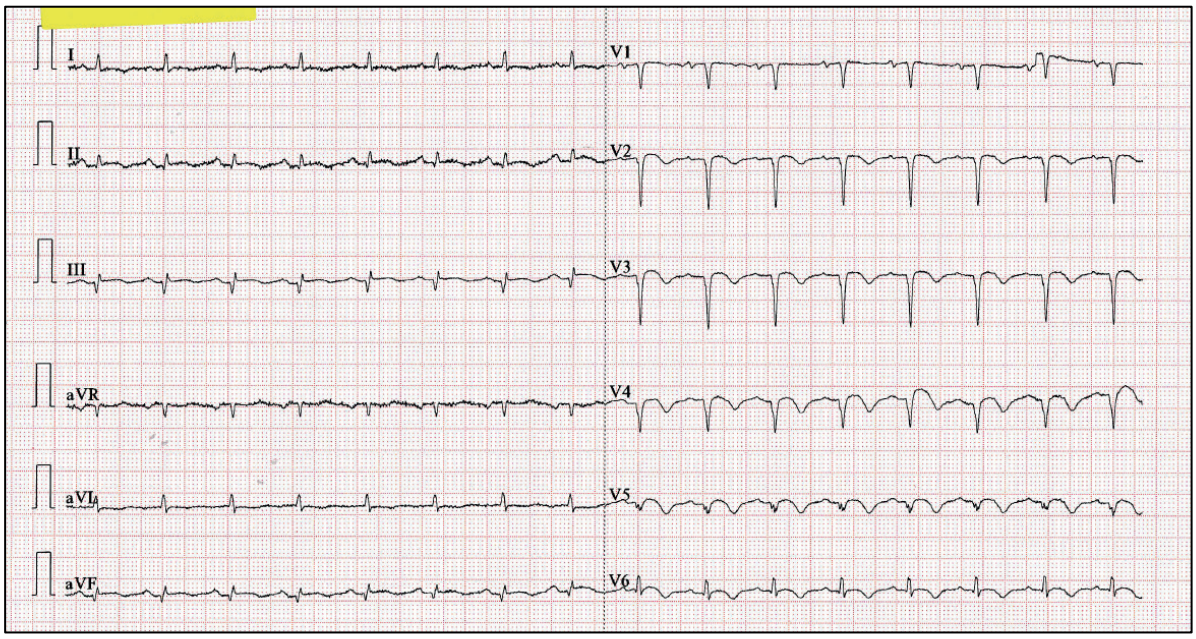

Figure 1. 12 leads ECGs revealed sinus tachycardia, low voltage limb leads, Q wave and ST elevation over anterior leads (V1-5), and T wave inversion over precordial leads (V2-6).

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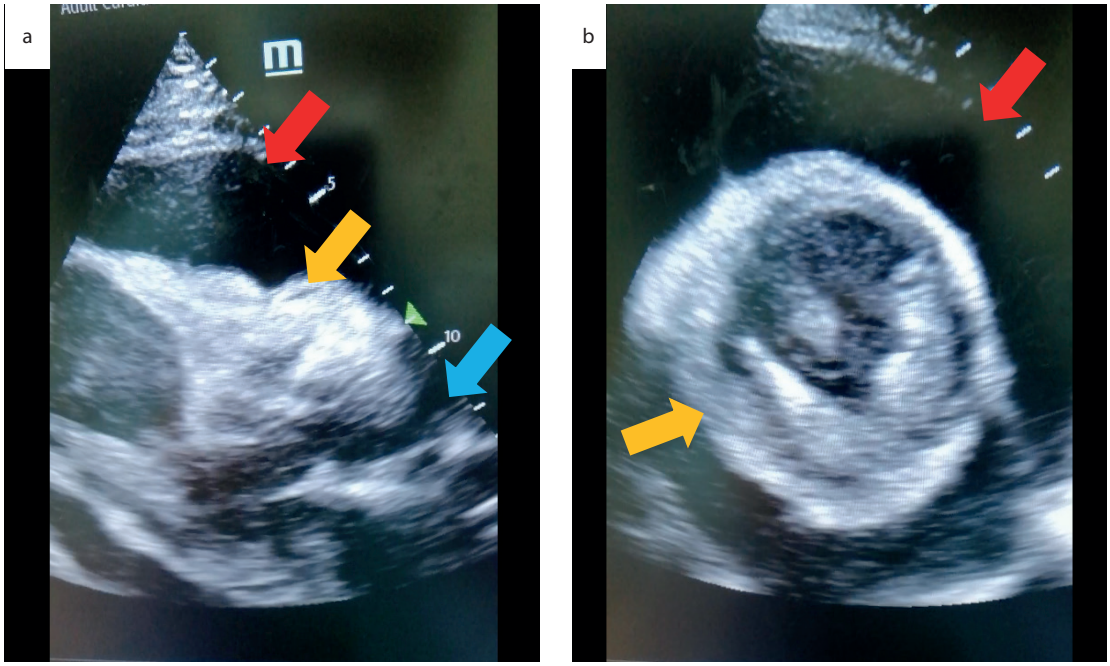

Figure 2. Bedside cardiac echocardiography: (a) Parasternal long-axis view and (b) Parasternal short-axis view during diastole phase identified an aortic root measuring 3.3cm with no intimal flap seen (blue arrow), massive pericardial effusion (maximum thickness of 3cm) (red arrow), and right ventricular collapse (orange arrow).

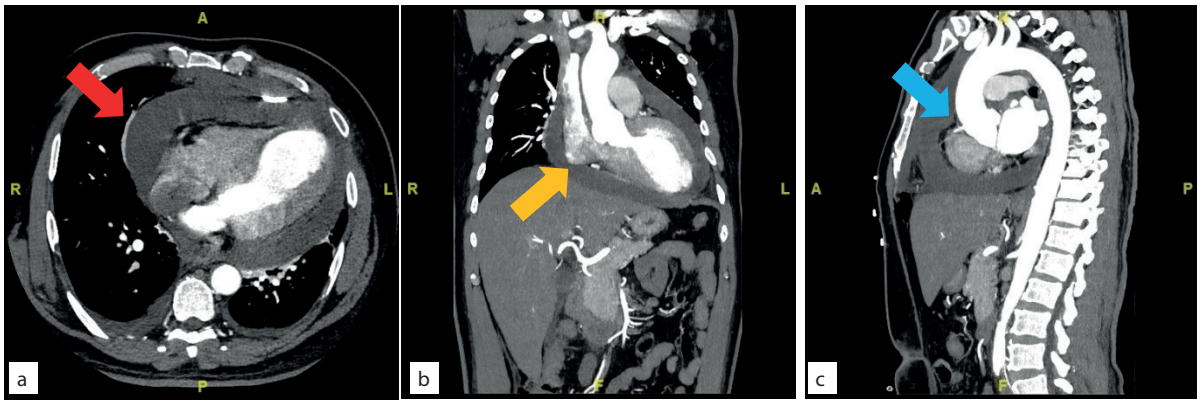

Figure 3. Chest computed tomography angiography (CTA): (a) Axial view, (b) Coronal view, and (c) Sagittal view confirmed the presence of a large pericardial effusion measuring 2.8 cm in maximum thickness (red arrow), along with the collapse of the right ventricle relative to the left ventricle (orange arrow), suggestive of the tamponade effect. There is no evidence of aortic aneurysm or dissection (blue arrow).

Discussion

Dressler syndrome is a form of pericarditis thought to be auto-immune-associated, triggered by cardiac tissue injury following myocardial infarction (1, 3).It typically occurs 2–6 weeks after the infarction(1). Other conditions linked to cardiac injury, such as trauma, post-pericardiotomy, and iatrogenic causes from cardiac procedures, can also drive autoimmune-mediated pericardial inflammation (3). In this case, the patient's recent NSTEMI and angioplasty procedure resulted in Dressler syndrome.

Dressler syndrome is characterized by pericarditis, pleuritis, and pneumonitis(1). On the other hand, the classic triad of chest pain, pericardial effusion, and ECG changes (diffuse ST elevation and PR depression) is not very sensitive, which is present in a minority of patients(4). The patient presented with severe epigastric pain radiating to both shoulders and a syncopal episode, underscoring the variability in myocardial infarction symptoms, which can sometimes mimic gastrointestinal or other non-cardiac conditions, complicating the diagnosis.

Sinus tachycardia, old ischemic changes (Q wave, ST elevation, and T inversion), and low voltage in limb lead on the ECG, suggesting cardiac tamponade. Autonomic sympathetic arousal, secondary to reducing cardiac output, is the cause of tachycardia. ST-segment elevation could be a non-specific finding in cardiac tamponade (5). Despite the initial ED referral for an acute myocardial infarction, the absence of a significant rise in troponin levels and the absence of new left ventricle wall motion abnormalities excluded this diagnosis. Low QRS voltage is due to heart position changes, increased distance from

the current generator to the recording electrodes, and reduced cardiac chamber size and volume (5). Classic pericarditis ECG changes are absent in this case, a finding not uncommon and present in only 20–24% of cases (4).

Dressler syndrome can cause pericardial effusion. Accumulation of pericardial fluid can lead to cardiac tamponade, characterized by hemodynamic instability due to impaired diastolic filling and reduced cardiac output. If the accumulation occurs rapidly, even small amounts (200–300 ml) of pericardial fluid can cause compressive effects(6). In contrast, if the accumulation is slow, these symptoms might not be evident, and the heart may compensate until 1-2 litres of fluid are present before becoming clinically symptomatic (6).The classic triad of hypotension, tachycardia, and muffled heart sounds, along with bedside cardiac ultrasound findings of massive pericardial effusion and right ventricular diastolic collapse, suggest cardiac tamponade. These classic symptoms may be absent in subacute or chronic pericardial effusions(6).

Cardiac tamponade is a clinical diagnosis based on a combination of a suggestive history, unstable vital signs, and physical examinations. The patient's blood pressure was relatively hypotensive (in obstructive shock), considering he was chronically hypertensive, which emphasizes the significance of bedside ultrasound in the rapid assessment and diagnosis of hemodynamically unstable patients in an emergency setting. In cardiac tamponade, echocardiography shows pericardial effusion, right ventricular compression during diastole, abnormal respiratory variation in both ventricular dimensions and tricuspid and mitral valve flow velocities with a plethoric inferior vena cava (6, 7). In this case, the patient exhibited hemodynamic instability, and echocardiography showed massive pericardial effusion with right ventricular collapse during the diastolic phase, confirming the diagnosis of cardiac tamponade.

The primary treatment for cardiac tamponade is pericardiocentesis, which involves draining the accumulated pericardial fluid to decompress the tamponade effect. Advances in medical technology now perform pericardiocentesis under real-time echocardiographic guidance, enabling precise detection of the largest pericardial effusion area, optimal needle insertion location, and prevention of accidental puncture of vital structures(8). In this case, drainage of 100 cc of fluid successfully decompressed the tamponade effect of the surrounding pericardial fluid, as evidenced by the stabilization of the patient's vital signs. The haemorrhagic nature of the fluid and the exclusion of infection and malignancy from pericardial fluid analysis pointed towards an inflammatory aetiology consistent with Dressler syndrome. Additionally, chest CTA is particularly useful to determine the causes of pericardial effusion and cardiac tamponade. The absence of CT evidence of aortic aneurysm or dissection, along with recent myocardial infarction evident by coronary angiography, confirmed the diagnosis and assured further optimization of management.

Following stabilization, the patient was managed with anti-inflammatory therapy to address the underlying autoimmune pericarditis. Aspirin, preferred for its dual anti-inflammatory and anti-platelet role, was administered, and colchicine was added for its synergistic effect and to reduce the risk of recurrent pericarditis (9). The patient's stable course over a two-week hospitalization and gradual reduction in pericardial effusion reflected the effectiveness of this treatment approach. Warfarin was also initiated, given the evidence of a left ventricular thrombus in the apex, as recommended by the American Heart Association (10).The patient's outpatient follow-up with aspirin and warfarin aims to prevent recurrent pericardial effusion and thromboembolic events.

Conclusion

Clinicians should maintain a high index of suspicion for cardiac tamponade in post-MI patients presenting with hypotension and other signs of hemodynamic instability. Rapid bedside echocardiography can be a crucial tool in diagnosing this condition. Prompt recognition and emergent pericardiocentesis are life-saving.

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Conflict of Interest statement
None declared.

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Case Report

Journal of Emergency Medicine Case Reports

Phonetic Ambiguities: Risks of Drug Name Confusion in Elderly Patient

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Abstract

An aging population presents significant challenges in pharmacotherapy oversight, with elderly individuals often lacking comprehensive information about their medical conditions. This paper addresses medication errors in the elderly, highlighting a case study of a 68-year-old male who ingested Warfarin® instead of Famodin®, leading to a severe overdose. The incident underscores the risks of phonetic confusion in drug names and the necessity for robust safety protocols. Collaborative efforts among healthcare stakeholders are crucial to ensuring safe medication administration and reducing errors.

Keywords: Drugs&aging, geriatric pharmacotherapy, healthy aging, phonetic ambiguities, warfarin overdose

Introduction

The demographic shift towards an aging population presents an increasingly formidable challenge, necessitating vigilant oversight and evaluation of their pharmacotherapy. Elderly individuals often encounter a dearth of comprehensive or precise information regarding their medical conditions and prescribed treatments, highlighting the critical need for enhanced communication and tailored educational initiatives within healthcare settings (1).A homophone, a fundamental linguistic concept, denotes words that share similar phonetic attributes while harboring distinct semantic or orthographic characteristics. Despite their phonetic resemblance, homophones maintain discrete lexical definitions and may occupy disparate syntactic roles(2). Within healthcare environments, medication errors arising from phonetic confusions in drug nomenclature present formidable obstacles. Of particular concern are the potential hazards engendered by phonetic ambiguities in Turkish medication terminology, which hold the propensity to precipitate significant lapses in medication administration within this susceptible demographic.

This paper aims to contribute to this imperative by presenting a compelling case study featuring a 68-year-old male who, devoid of any clinical indication for anticoagulation therapy, presented to our Emergency Department subsequent to an acute ingestion of warfarin.

Case

The 68-year-old male patient presents with a three-day history of oral bleeding accompanied by abdominal discomfort, which he attributes to gastritis. He denies concurrent symptoms of diarrhea or vomiting and notes no discernible alterations in stool characteristics. Upon examination, vital signs indicate a blood pressure of 160/87 mmHg, a body temperature of 36.3°C, a heart rate of 74 beats per minute, a respiratory rate of 14 breaths per minute, and a blood oxygen saturation (sPO_2) of 97%. Past medical history reveals a diagnosis of coronary artery disease, hypertension, and gastritis, necessitating ongoing pharmacotherapy. However, the patient's current medication list is unavailable, yet he mentions prior use of 40 mg Famotidine (brand name Famodin®), 100 mg Metoprolol succinate (brand name Saneloc®), and 100 mg acetylsalicylic acid (brand name Coraspin®).

The oropharyngeal examination reveals bleeding sites suggestive of gingival origin, while thoracic and abdominal examinations yield no notable findings. Rectal examination demonstrates normal feces. Laboratory analysis reveals a hemoglobin level of 12 g/dL, a white blood cell count of $8.8x10^3$/uL, and a platelet count of $245x10^3$/uL, alongside unremarkable renal and hepatic profiles, electrolyte levels, and an international normalized ratio (INR) of 21.3. Upon further inquiry, the patient confessed to substituting his prescribed medication with a phonetically similar one

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from his spouse's supply due to the depletion of Famodin® over the preceding week. Subsequent investigation into the wife's medication regimen revealed her prescription of warfarin, branded as Coumadin®, for the management of atrial fibrillation and stroke prevention. It became evident that the patient inadvertently ingested his wife's medication, Coumadin®, instead of his prescribed Famodin®.

The patient underwent management according to the warfarin overdose protocol, wherein intravenous vitamin K at a dose of 10 mg was administered initially, followed by the administration of Prothrombin Complex Concentrate (Cofact®) at a volume of 60 ml. The patient's INR levels were monitored, revealing a value of 1.5 at the 3rd hour and 1.4 at the 6th hour. Additionally, the patient's hemoglobin level, assessed at the 6th hour, was recorded as 11.6 g/dL. Following the resolution of active bleeding symptoms and the absence of further decline in hemoglobin levels, the patient, who attained the target INR level during their stay in the Emergency Department, was discharged with personalized recommendations. Therapeutic adjustments were implemented accordingly.

Discussion

Medication misuse is a significant concern, notably among two vulnerable populations: the elderly and preschool children. This misuse encompasses various errors, such as incorrect dosages, missed doses, medication mixing, and off-label use (3). Among the elderly, several factors contribute to medication errors, including polypharmacy, complex treatment regimens, limited awareness of medication schedules, cognitive and physical decline, reduced social acuity, and negative attitudes toward pharmacotherapy (4). Building on this literature, we believe that off-label drug use in advanced age is associated with declines in both social and cognitive faculties.

Medication errors due to phonetic confusions in drug names pose significant challenges in healthcare settings. Studies have shown that similarities in the pronunciation of medication names can lead to administration errors, resulting in adverse patient outcomes. For example, in a study conducted by Hoffman et al., it was found that phonetic similarities between the drugs Celebrex (celecoxib), Cerebyx (fosphenytoin), and Celexa (citalopram) led to medication errors in clinical practice, highlighting the importance of distinct drug nomenclature (5). Similarly, a study by Gentin demonstrated that medications with similar-sounding names, such as Zantac (ranitidine) and Xanax (alprazolam), were frequently confused by healthcare professionals, underscoring the need for heightened awareness and vigilance (6).

Errors in medication names that look alike or sound alike (LASA) pose a significant risk as they can be mistaken for each other, leading to potential harm to patients. For instance, medications like mercaptamine and mercaptopurine share similarities in their names, increasing the likelihood of confusion. LASA errors often occur due to shared linguistic properties between medication names, such as phonetic or orthographic similarities. Moreover, factors like similar packaging, tablet appearance, strength, route of administration, or therapeutic indication further compound the risk of errors. Healthcare providers may inadvertently contribute to these errors through miscommunication or misinterpretation of drug names with similar phonetic characteristics. To address this issue, strategies like the use of Tall Man lettering have been proposed. This method involves capitalizing specific letters in drug names to emphasize their differences, thereby helping to mitigate medication errors related to phonetic confusions (7).

An imperative underscored, particularly in a prior investigation, is the necessity for vigilant monitoring of medication usage among elderly patients. Challenges in adhering to prescribed medication regimens were elucidated among elderly individuals post-hospital discharge (8). As exemplified in the preliminary patient history within our study, the adoption and augmentation of an electronic prescription framework, particularly tailored for individuals with compromised mental and social capabilities, are indispensable. Such systems should be readily accessible to healthcare providers through comprehensive databases to address the multifaceted health concerns associated with medication use among the elderly (9). Moreover, the prioritization of training for personnel involved in elderly care is paramount for the effective regulation of medication utilization among this demographic. Local authorities and healthcare administrators bear additional responsibilities in fostering an environment conducive to the safe and appropriate administration of medications in elderly patients (10).

Conclusion

Proactive measures are urgently needed to address medication misuse among vulnerable populations, particularly the elderly. Implementing comprehensive strategies, such as electronic prescription systems and targeted personnel training, is crucial to mitigate the risks associated with medication errors in this demographic. Additionally, collaborative efforts involving healthcare stakeholders and policymakers are imperative to ensure the safe and effective delivery of pharmacotherapy to elderly patients while minimizing the occurrence of medication errors. Furthermore, phonetic confusions in drug names present a significant challenge in medication safety, especially among the elderly population. Healthcare providers must remain vigilant and adopt strategies to reduce the risk of medication errors stemming from phonetic similarities in drug names. Collaborative efforts between healthcare professionals, medication safety organizations, and regulatory agencies are essential to effectively address this issue and improve patient outcomes.

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