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Management of Bell palsy: our clinical practice guideline

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Abstract

Bell palsy is a common neurologic disorder defined by an acute mononeuropathy affecting the seventh cranial nerve, presenting with ipsilateral facial weakness. It remains the most common etiology of facial nerve paralysis.

This retrospective study identified 75 cases of Bell's palsy from the medical records of the ENT department of the Regional Hospital of Sidi Bouzid from January 2020 to December 2022.

Our series consisted of 17 children and 58 adults, with an average age of 37 years (range: 3 – 88 years). All patients presented with a unilateral facial asymmetry. According to the House-and-Brackmann grading, the initial assessment found 5 patients with grade I, 16 patients with grade II, 24 patients with grade III, 27 patients with grade IV, and 3 patients with grade V. Treatment was conducted in an outpatient setting in 64 patients. Hospitalization was required for 11 patients. Corticoids were initiated in 64 patients. Antiviral treatment, always in association with corticotherapy, was indicated within 21 patients, with a mean consultation delay of 3 days. All patients were given a vasodilator and vitamin therapy. Rehabilitation facial therapy was initiated in all cases, with a mean delay of 7 days. The objective of this study is to review the therapeutic management guideline of the patients presented with idiopathic facial nerve paralysis in our department compared with the relevant literature.

Keywords: Bell's Palsy; Corticotherapy; Facial Nerve; Physical Therapy

INTRODUCTION

Bell's palsy is a condition that affects the facial nerve, which is mainly controlling the muscles of the facial expression. It is commonly unilateral and acute, with sudden onset involving both the superior and inferior areas of the hemiface [1].

It occurs in 15-30 people per 100,000 in the population each year, with no predilection for sex or ethnicity [2]. It can occur at any age, but the incidence is moderately higher after the age of forty [3]. Its major cause is believed to be an infection occurring in the facial nerve by the herpes simplex virus responsible for swelling and, therefore, a compression in its canal [2].

Therefore, it remains a diagnosis of exclusion

According to studies, the herpes simplex viral genome is identified in the facial nerve endoneurial fluid in 79% of cases.

Bell's palsy is more commonly associated with patients presenting medical history such as obesity, hypertension, diabetes, or upper respiratory conditions (including COVID-19 caused by SARS-CoV-2), immunocompromised patients, and pregnant women [4,5].

A well-conducted ENT and neurological clinical examination is recommended when Bell's palsy is suspected.

When the diagnosis is confirmed, corticosteroid therapy should be implemented early for 10 days as a primary treatment for its potential to reduce swelling and inflammation. Antiviral therapy (Acyclovir or Valacyclovir) should be associated with steroids within 72 hours after the onset of the symptoms, in severe facial paralysis and in Ramsay-Hunt syndrome to eradicate the HSV infection. An isolated antiviral therapy is not usually recommended [5]. An early physical rehabilitation is performed, especially in severe grades of paresis [6].

The course of this disease is spontaneously favorable, with more than 70% complete resolution within 6 months and more than 80% subtotal resolution (House-Brackmann grade I or II) [1]. Recurrence occurs in 7–8% of patients.

This study aims to describe the epidemiologic, clinical, and therapeutic aspects of Bell's palsy in our department with a brief literature review.

METHODS

Our study was a retrospective analysis that collected data from January 2020 to December 2022. All the patients affected by idiopathic facial paralysis who reached the otolaryngology emergency department of the regional hospital of SIDI Bouzid were assessed. Eleven patients were hospitalized in the ENT department of our hospital.

The clinical evaluation of the BP was assessed via the House-Brackmann (HB) Facial Grading System. CT scans and/or MRI were performed when the medical history or clinical symptoms suggested the diagnosis of a secondary facial paralysis.

Patients who presented with a central facial palsy or facial palsy with a well-known etiology were eliminated.

RESULTS

Seventy-five patients affected by peripheral facial paralysis were included in this study. The mean age is evaluated at 37 years (ranging between 3 and 88 years). There was a predominance of adults over children: Seventeen of our patients were children, while fifty-eight were adults. There was a slight female predominance, with a sex ratio of about 0.87.

Risk factors for peripheral facial palsy were found within 38.67% of patients, mainly due to metabolic diseases (diabetes, hypertension), hematologic disorders, and pregnancy (Table 1).

The consultation average delay was about 3 days (ranging between one and 7 days). All patients

presented with acute facial asymmetry as a main symptom.

Table 1: *Medical conditions associated with facial palsy in our series*

Medical history	recurrence in our series(%)
Metabolic diseases	
- Diabetes	21.3
- High blood pressure	18.6
- Dyslipidemia	4
Hematological diseases (Biermer's disease, B-thalassemia, splenectomy)	4
Neoplasy	5.3
Pregnancy/ Puerperal period/ Pregnancy-induced hypertension	4
Facial palsy history	2.6
Others: hypothyroidism, epilepsy.	4

The left side incidence of palsy was noted in 60% of cases, whereas the right side was observed in 40% of cases.

In the first physical examination, 60% of patients presented with mild-to-moderate facial palsies (grades I-III), while moderate-to-severe palsies (grades IV–VI) was observed in 40% of cases (Figure 1).

CT scan was assessed for 17.34% of patients and were completed with cerebral Magnetic Resonance Imaging for 4% of patients. Imaging examinations were indicated in the presence of other neurological symptoms (peripheral paresthesia), in case of predominance of the palsy in the inferior territory, a negative Charles-Bell sign, and in the presence of central involvement risk factors.

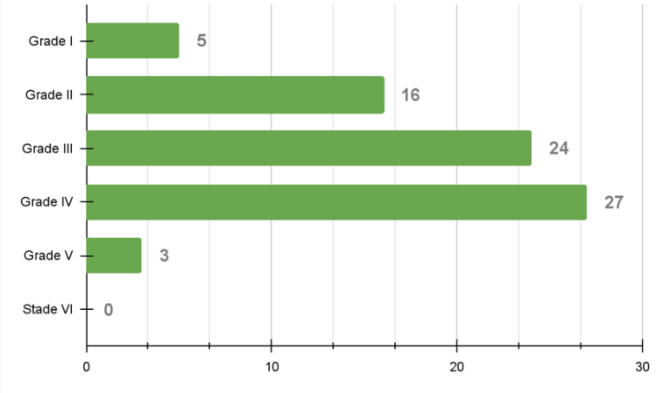

Figure 1: *Distribution of the severity of the facial palsy according to the House-Brackmann facial grading scale at the initial clinical examination.*

Audiometry and impedance audiometry were performed for five patients with the result: loss of stapedial reflex. Most patients were treated on an outpatient basis (85.3%), while 14.7% were treated in a hospital setting. Most patients cared for in hospitals were adults. Hospitalization was indicated in cases of severe initial presentation: House and Brackmann grades III - VI or in the presence of tares with a higher risk of decompensation (Diabetes within 45.45 % of patients) (Table 2).

Table 2*: Detailed hospital setting treatment with molecule, mode of administration, and number of patients treated (total of 11 patients)*

Treatment	Molecule/ Dose	Recurrence in our series%
Corticosteroids only (IV)	Prednisolone or méthylprednisolone 1 mg/kg/d * 7-10 days	27.2
corticosteroids + Antivirals (IV)	Prednisolone or méthylprednisolone 1mg/kg/d * 7-10 days Valaciclovir 1g *2-3/d	81.8
Physical therapy + vitamin therapy		100

81.25% of the patients treated on an outpatient basis received corticosteroids associated with antivirals within 18.75% of the cases who have consulted with severe grades of palsy within 72 hours on the onset of the symptoms (Table 3).

Table 3: *Detailed outpatient-based treatment with molecule, mode of administration, and number of patients treated (total of 64 patients)*

Treatment	Molecule/ Dose	percentage %
Corticosteroids + vitamin therapy + Physical therapy per os	Prednisolone or méthylprednisolone per os*** 1mg/kg/d * 7-10days	62.5
Corticosteroids + Antivirals + vitamin therapy+ Physical therapy per os	Prednisolone or méthylprednisolone 1mg/kg/d * 7-10days + Valaciclovir 1g *2-3/d ***per os	18.7
Vitamin therapy+physical therapy		100

Antivirals were prescribed for 28% of patients in our series. Those patients consulted in our emergency department within 71 hours after the onset of symptoms.

Physical therapy was initiated within all patients with a mean delay of four days. The final evaluation of patients after 6 months of regular follow-up at the consultation showed full recovery in 67% of cases.

DISCUSSION

Bell's palsy is the most common form of peripheral facial palsy with sudden onset involving both the superior and inferior areas of the hemiface and affecting voluntary, autonomic, and emotional motricity [1].

It can be preceded by some prodromes such as auricular or retroauricular pain, dysgeusia, and/or hyperacusis [6,7]. Diagnosis is by elimination. Clinical examination should be complete with a detailed ENT and neurologic examination.

Audiometry seems compulsory for an ipsilateral conductive component screening. Tympanometry is performed to screen the stapedial reflex, which can be useful for the outcomes and localization of the lesion [8].

Assessing neural involvement is important in the initial management of Bell's palsy to enable the grading of severity and to monitor its progression. The most widely used classification in the initial phase is still House and Brackmann (HB) [9].

Studies did not recommend the necessity of any biological examinations in the management of Bell's palsy. However, glycated hemoglobin (HbA1c fraction) may be useful in case of the presence of diabetes to control its balance and prevent a relevant decompensation under high-dose corticosteroids [1,4].

Imaging examinations are known to be less contributive in Bell's palsy when clinical and audiometric assessments are normal. However, Bell's palsy is a diagnosis of exclusion, and imaging may be requested in the presence of signs that may suggest a differential diagnosis, especially if a central cause of the palsy is suspected [1,10].

MRI remains the gold standard when imaging exams are indicated [10,11].

Therefore, Emergency imaging is not necessary if symptomatology is typical of Bell's palsy but should be indicated if necessary to rule out before confirming the diagnosis of Bell's palsy disease.

Medical treatment for Bell's palsy has been debated as progression may be spontaneously favorable, with more than 70% of complete recovery within 6 months and more than 80% of subtotal recovery (HB grade I or II) [1,5].

According to studies, all the patients that were diagnosed with Bell's palsy (unless in the case of a serious contraindication to steroid therapy) were prescribed a standardized oral pharmacological treatment with prednisone 1 mg/Kg for ten days. Antivirals may be considered for patients with severe paresis with a dose of (Valacyclovir) 1g 3 times/day for ten days within 72h after the onset of the symptoms [1,12]. According to the literature, intravenous therapy proves to be more efficient, especially in severe palsies or in the presence of serious medical history [1].

Physical therapy is applied in Bell's palsy, especially in severe grades, to hasten recovery and limit sequelae. However, facial muscle rehabilitation improves facial function when Bell's palsy fails to resolve, particularly when it is assessed sooner after the onset of the symptoms [3,4].

Acupuncture was reported to be relatively superior to drug treatment in a Chinese meta-analysis, but this finding is dubious due to considerable bias induced by the diversity of techniques. Evidence is lacking for isolated acupuncture [13].

Hyperbaric oxygen therapy can be proposed as complementary treatment as its fundamental concept is to spread oxygen within the tissue, thus reducing facial nerve hypoxia, which enhances recovery [1,5].

Clinical follow-up should be continued for several months to ensure against complications, in particular ophthalmic complications: keratitis, corneal ulcer, and panophthalmia [5].

While approximately 80% of patients may experience full recovery, some patients may present residual deficits such as eyebrow droop, eye closure difficulty, asymmetric smile, trouble eating, or nasal breathing dysfunction [7].

Patients with incomplete eye closure should be given eye protection with lubricating eye drops, especially at night, to prevent corneal damage [4].

Long-term complications can include residual facial weakness, facial synkinesis, facial contracture, and facial spasm [8,9,14].

The benefit of surgery for Bell's palsy remains controversial at present. Demonstrating the benefits of decompression may be challenging due to various factors influencing the relevant outcome, such as patient selection, the timing of surgery, and the chosen method and approach. However, although efficacy cannot be proved, we consider that surgery should be proposed as an alternative treatment. Consensus appears to exist regarding the specific part of the nerve that would

be decompressed, guided by radiological and surgical findings [4,10].

CONCLUSION

While spontaneous recovery is common among Bell's palsy patients, improvement in facial weakness can significantly impact their quality of life. It is crucial to accurately confirm the diagnosis and to prevent overlooking other treatable conditions. Optimal recovery opportunities can be achieved by selecting appropriate treatment options for relevant eligible patients. Therefore, the recovery may be complete or partial, and it can affect a patient's long-term quality of life, adding to depression and psychological distress.

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Specific Features of Chest Pain in Young People Treated in Prehospital Care

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Abstract

Background: Chest pain is a frequent cause of emergency department (ED) visits and prehospital interventions. While cardiovascular etiologies are prioritized, many cases have non-cardiac causes. Young patients (<45 years) represent a unique subgroup requiring targeted evaluation to optimize prehospital care.

Objective: This study describes the epidemiological, clinical, electrocardiographic, and therapeutic characteristics of patients under 45 years treated for chest pain in prehospital settings, with a focus on acute coronary syndrome (ACS).

Methods: We conducted a longitudinal observational study over six months (July–December 2022) in the Emergency Medical Services (EMS) center of Eastern Tunisia. We included patients under 45 years presenting with acute anginal chest pain requiring Emergency Medical Response Team (EMRT) intervention or managed via medical regulation. Data collection included patient demographics, pain characteristics, vital signs, ECG findings, prehospital management, and angioplasty outcomes.

Results: Among 74 cases, the mean age was 35.5 years (±8.5), with 85.1% male. First medical contact was an EMRT physician in 8.1% of cases and an ED physician in 91.9%. Typical chest pain was reported in 45.9%, predominantly retrosternal (62.2%) and radiating to the left arm (23%). ACS was more frequent in men (p=0.004), smokers (p=0.001), and those with cardiovascular risk factors (p=0.015). ACS cases had higher pain intensity (p=0.001).

Conclusion: Smoking and cardiovascular risk factors strongly predict ACS in young patients. Improved prehospital triage and risk stratification tools are needed to enhance early ACS diagnosis and management.

Keywords: Chest Pain, Acute Coronary Syndrome Prehospital Care Young Patients Risk Stratification

INTRODUCTION

Chest pain is a frequent reason for emergency department visits. It accounted for approximately 5% of annual emergency department visits in the United States in 2020 (1), making it the second most common complaint. It is also a frequent reason for calling emergency medical services (EMS), with studies reporting up to 20% of calls (2,3) and up to 16% of their intervention activities (4). Patients present with a range of signs and symptoms reflecting numerous possible etiologies. Cardiac, aortic, pulmonary, esophageal, gastric, Mediastinal, pleural, and abdominal visceral pathologies can all cause chest pain. Thus, the treating physician evaluating a patient presenting with this complaint must always consider the most fatal etiologies, primarily cardiovascular diseases, which cause approximately one-third of deaths worldwide (5),

while taking into account the patient's underlying condition. Acute coronary syndrome represents one of the most formidable cardiovascular pathologies, alone responsible for 1.8 million deaths worldwide in 2020 (5). In addition to acute coronary syndrome, other severe etiologies, such as pulmonary embolism, aortic dissection, and pneumothorax, must not be overlooked (6). These multiple and severe pathologies mean that patients suffering from chest pain tend to be systematically overestimated, increasing the use of ambulance resources and contributing to the overcrowding of emergency departments. Moreover, most patients suffering from chest pain transported by ambulance can ultimately be diagnosed with a transient non-cardiac illness. Indeed, according to studies, only 5 to 10% of patients complaining of chest pain have acute coronary syndrome (4,7,8), and up to a quarter of the population may present with non-cardiac chest pain (9). Especially in young subjects, several benign diagnoses, such as gastro-esophageal reflux disease (GERD), musculoskeletal causes, and psychiatric causes, increase the risk of non-cardiac chest pain (10).In this context, our work aims to describe the epidemiological, clinical, electrocardiographic, and therapeutic characteristics of patients treated for chest pain in Prehospital care and the particularities of chest pain related to acute coronary syndrome occurring in young subjects under 45 years of age

METHODS

Study Design: This is a descriptive longitudinal observational study conducted by the emergency medical services center in Est of Tunisia (03) over 6 months, from July 1, 2022, to December 31, 2022.

Study Population: we have included in this study, patients under than 45 years old, managed in the Prehospital setting for acute anginal chest pain requiring intervention by EMRT(Emergency Medical Response Team), either as a primary transfer(Including Prehospital interventions and those in peripheral hospitals lacking advanced technical facilities); Chest pain cases managed by the medical regulation 03 without EMRT intervention, with indication for another type of intervention and transport (civil protection, type B ambulance) due to unavailability of resources and patients who presented with acute coronary syndrome (ACS) confirmed by ECG and/or troponin assay. Exclusion Criteria was essentially secondary interventions for patients presenting with ACS.

Data Collection: This is an exhaustive study in which data collection was carried out using a pre-filled form for all patients meeting the inclusion criteria, as they were identified, based on data from the intervention and regulation forms as well as the EMS 03 chest pain registry. Follow-up until angioplasty was performed for each patient (when possible) to monitor the evolution and complete the forms. The measured variables are: General characteristics of the population, Characteristics of chest pain, Clinical data after the first examination by the EMRT or the first-contact physician (evaluation of vital signs, ECG: 12 or 17 leads, initial prehospital management, Evolution, thrombolysis (success/failure), angioplasty, and culprit artery and Complications presented by the patient.

Operational Definition of Variables (11): ST-elevation myocardial infarction (STEMI): New ST-segment elevation at the J-point in at least two contiguous leads; Non ST elevation coronary infarction, NSTEMI (Patients with NSTEMI were classified by risk stratification according to ESC 2023 recommendations) (12); STEMI equivalent (Wallens syndrome, Winter syndrome, New-onset left bundle-branch block, ST sub-shift > 1 mm in

6 or more leads with ST+ limited to RV and/or V, Atypical ST elevation); Successful thrombolysis (12) is defined as decrease in ST-segment elevation by at least 50% at 60 - 90 min of thrombolysis, occurrence of reperfusion arrhythmia and disappearance of chest pain.

Data analysis was performed using SPSS software. For descriptive study:

- The normal distribution of variables was verified by the Kolmogorov-Smirnov test.
- Continuous variables following a normal distribution are expressed as means and standard deviations.

Continuous variables not following a normal distribution are expressed as medians and interquartile ranges [25%-75%].

Discontinuons variables are presented in proportions.

For univariate analysis, means were compared using the independent samples Student's t-test.

Ethical considerations: This study was conducted in accordance with the ethical standards for research, ensuring the anonymity and confidentiality of data. The confidentiality of medical records was strictly maintained. The results of this study will be utilized solely for scientific purposes.

RESULTS

We included 74 calls and interventions by an EMRT for a young subject under 45 years old (Figure1)

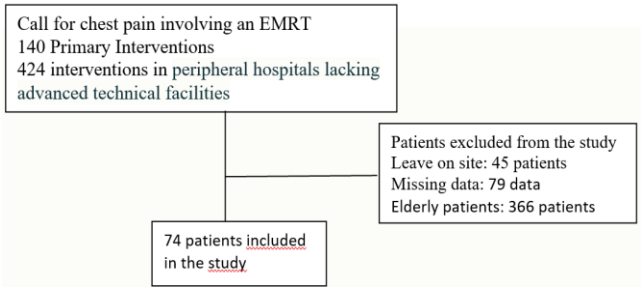

Figure 1: *Study Flowchart*

Socio-demographic characteristics: The mean age of our population was 35.5 years (±8.5), with extremes ranging from 10 to 45 years old. Among our patients, 63 (85.1%) were men, and 11 (14.9%) were women, with a male/female sex ratio of 5.7. (Figure 2)

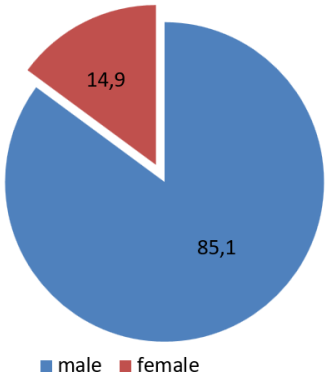

Figure 2: *Distribution of Patients by Gender*

First Contact Physician: The first contact physician with the patient presenting with chest pain was the EMRT intervention physician in 6 cases (8.1%) and the emergency department physician in a healthcare facility in 68 cases (91.9%). (Figure 3)

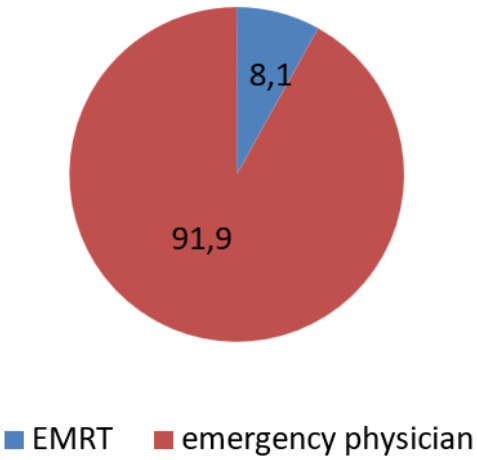

Figure 3: *First Contact Physician*

Medical History of Patients: Among our patients, 31 (41.9%) had medical histories considered cardiovascular risk factors. (Table 1)

Table 1: *Medical History of Patients*

Medical history	**N=74 (%)**
COVID19 Vaccination	11 (14.9)
Hypertension	5 (6.8)
Dyslipidémia	2 (2.7)
Diabetes	2 (2.7)
Ischemic Heart Disease	7 (9.5)
Established Vascular Disease (Stroke, PAD...)	5 (6.8)
Overweight	1 (1.4)
Active Smoking	31 (41.9)
Family History of Coronary Artery Disease	0
Sedentary Lifestyle	1 (1.4)
Pulmonary Embolism or Venous Thrombosis	1 (1.4)
Active Cancer	0
Recent Surgery or Immobilization	0
Coronary Artery Disease	6 (8.1)
Aspirin Use in the Last 7 Days	0
Heart Failure	2 (2.7)
Renal Failure	1 (1.4)
Respiratory Failure	3 (4.1)
Cardiovascular Risk	
Low	64 (86.4)
Moderate	7 (9.5)
High	2 (2.7)
Very High	1 (1,4)

Characteristics of Chest Pain: The chest pain in our study population is often typical (34; 45.9%), retrosternal (46; 62. 2%) radiating frequently to the left arm (17; 23%), described as a burning sensation (34; 44.6%), generally occurring at rest (35; 47.3%), often neglected by the patient and potentially lasting up to 15 hours. The patient is often in pain during the consultation (65; 87.8%), with persistent pain that can last up to 2 hours. Associated signs are discreet (7; 9.5%), predominantly dyspnea (3; 42.6%). The characteristics of the chest pain presented by our patients according to the age category are summarized in the following (Table 2).

Table 2: *Characteristics of Chest Pain*

Characteristics	N= 74 (%)
Chest Pain	
Typical	34 (45.9)
Atypical	40 (54.1)
Location	
Retrosternal	46 (62. 2)
Left Precordial	4 (5.4)
Right Précordiale	0
Basilar	6 (8.1)
Lateral	5 (6.8)
Epigastric	2 (2.7)
Poorly Specified	11 (14.9)
With Radiation	21 (28.4)
Radiation location	
Left Arm	17 (23)
Both upper limbs	1 (1.4)
jaw	1 (1.4)
right upper limb	1 (1.4)
scapular	1 (1.4)
Pain type	
Burning	34 (44.6)
Constricting	11 (14.9)
Oppressive	16 (21.6)
Tingling	4 (5.4)
Palpitation	9 (12.2)
Pain changes	8 (10.8)
Pain changes with	
Respiration	1 (12.5)
Palpation or Arm movement	7 (87.5)
Pain present at consultation	65 (87.8)
Onset time (hours)	15 [9 – 20]
Duration of chest pain (hours)	2 [1 – 7]
Pain onset	
At rest	35 (47.3)
On exertion	6 (8.1)
Not specified	33 (44.6)
Associated signs	7 (9.5)
Type of signs	
Sweating	1 (14.2)
Flu-like Syndrome	1 (14.2)
Dyspnea	3 (42.6)
Fever and chills	1 (14.2)
Nausea and vomiting	1 (14.2)

Clinical Parameters on Initial Examination: Patients usually present in a stable clinical state with a median VAS of 5. Table 3 summarizes the clinical parameters on the initial examination of our patients.

Table 3: Clinical parameters

Variable	N= 74, Median [IIQ]
SBP	120 [110 – 140]
DBP	70 [68 – 90]
HR	84 [78 – 97]
RR	18 [16 – 18]
SaO2	98 [98 – 99]
TRC	2 [1 – 3]
GCS	15
GAD	1.2 [1 – 1.6]
VAS	5 [3 – 8]

***SBP**: systolic blood pressure; **DBP:** diastolic blood pressure; **HR** heart rate, **RR**: respiratory rate, **TRC**: skin recolouring time**GAD**: capillary glycemia; **SaO2**: oxygen saturation**, EVA**: pain scale; **GCS:** Glasgow scale; **VAS:** Visual Analog Scale*

First ECG made: An ECG was performed in 71 (95.9%) cases, with a 12-lead ECG in 50 cases (67.6%) and a 17-lead ECG in 16 (21.6%) cases (Table 4).

***Table 4:** Characteristics of the first ECG made*

Variable	N=71 (%)
Normal ECG	10 (14)
Present Abnormality	
Repolarization	38 (53.5)
Rhythm	8 (11.2)
Conduction	7 (9.8)
Electrical Signs of ACS	36 (40.7)
STEMI	20 (55.5)
NSTEMI	10 (27.7)
STEMI Equivalent	2 (5.5)
Unstable Angina	4 (11.3)
Other Electrical Signs	
T wave Abnormalities	3 (42)
Pathological Q Waves	2 (2.8)
Benign Early Repolarization	4 (5.6)
Rhythm Disturbance	
Atrial Fibrillation	2 (28.5)
Supraventricular Tachycardia	3 (42.8)
ventricular Tachycardia	2 (28.5)
Conduction Disturbance	
Right Bundle Branch Block	5 (62.5)
Left Bundle Branch Block	3 (37.5)
Atrioventricular Block	0
Dynamic ECG changes	7 (9.8)
Repolarization changes	2 (28.5)
Rhythm changes	1 (14.2)
Normalization changes	4 (57.3)

STEMI: ST-Elevation Myocardial Infarction; ACS: Acute Coronary Syndrome; NSTEMI: nonST-Elevation Myocardial Infarction

A normal ECG is found in 10 to 14% of cases, repolarization disorders predominate (38, 53.5%), and STEMI-type (20, 55.5%).

Additional Examinations:

Troponin levels were measured in 19 (25.7%) patients, with serial measurements in 5 cases (26.3%). *(Table 5)*

***Table 5:** Troponin level*

Troponin	Young patients (N=71)
Measured	19 (25.7)
Positive Kinetics	5 (26.3)

A chest X-ray was performed in 7 (9.3%) patients. (Table 6).

***Table 6:** Frontal chest X-ray*

Variable	Young patient (N=7)
Pneumothorax	1 (14.2)
Interstitial Syndrome	1 (14.2)
Bronchial Syndrome	1 (14.2)
Normal	4 (57.1)

Etiology of Chest Pain: At the end of the emergency department evaluation, 57 (76%) of the subjects had cardiovascular etiologies for their chest pain. (Table 7)

Types and territory of ACS presented: Chest pain of cardiovascular origin is essentially related to STEMI (20, 55.6%), with the inferior territory being the most affected (11, 30.5%) (Table 8)

Risk Stratification according to ESC 2023 recommendations: Our patients were classified based on the risk established by the ESC 2023 recommendations. We found that 23 (71.9%) of the patients presenting with ACS were at very high risk, 2 (6.3%) were at high risk, and 7 (21.9%) were not classified as very high or high risk. (Table 9).

Table 7: *Etiology of chest pain*

Variable	**N=74**
Cardiovascular	59(79.7)
ACS	36 (61)
Myocarditis	3 (5)
Aortic Dissection	2 (3.3)
WPW syndrome	1 (1.7)
Supraventricular Tachycardia	1(1.7)
Atrial Fibrillation	1(1.7)
Ventricular Extrasystole	1(1.7)
acute pulmonary oedema	14 (14)
Tetralogy of Fallot	
Benign early repolarization	
Nonspecific repolarization abnormalities	
Pleuropulmonary	1 (1.4)
Pneumothorax	1 (100)
Psychiatric	5 (6.8)
Non specified	9 (12.2)

WPW: Wolff Parkinson syndrome; ACS: Acute Coronary Syndrome

Table 8: Types and territory of ACS

ACS	**(N=36)**
clinical Form	
STEMI	20 (55,6)
NSTEMI	12 (33,3)
Equivalent STEMI	2 (16,6)
Recent LBBB	1 (50)
Not specified	1 (50)
Instable Angina	4 (11,1)
Territory	
Anterior	6 (16,6)
Inferior	11 (30,5)
Lateral	1 (2,7)

LBBB: Left Bundle Branch Block; STEMI: ST-Elevation Myocardial Infarction

Complications of ACS: The main complication of the clinical presentation was the occurrence of a cardio-respiratory arrest during patient management, with 5 (13.8%) of the patients experiencing this complication. The outcome was spontaneous circulation return in 40% of cases and death despite resuscitation efforts in 60% of cases. No patient presented with Acute pulmonary edema (OAP), cardiogenic shock, or rhythm disorders

Table 9 : *Stratification du risque selon les recommandations ESC 2023*

Risk assessment	**(N=16)**
Very high risk	3 (18,8)
High risk	2 (12,5)
Not high risk	11 (68,8)

In the univariate analysis, factors associated with ACS in Young Patients Presenting with Chest Pain were as follows:

Gender: Men presenting with chest pain requiring intervention by a Mobile Emergency and Resuscitation Service (SMUR) team are at a higher risk of having an acute coronary syndrome than women for the same reason, with a statistically significant difference (55.6% vs 9.1%, p=0.004, OR=12.5).

Cardiovascular Risk Factors: Patients with known cardiovascular risk factors are more likely to have an acute coronary syndrome in the context of chest pain, with a statistically significant difference (64.5% vs 35.5%, p=0.015, OR=3.2).

Smoking: Smokers are at a higher risk of experiencing ACS in the context of chest pain, with a statistically significant difference (64.5% vs 35.5%, p=0.001, OR=9).

Characteristics of Chest Pain: Patients with typical chest pain, as assessed by the clinician, have a statistically significantly higher probability of having an acute coronary syndrome (ACS) compared to those with atypical chest pain (67.6% vs 32.4%, p=0.003, OR=4.3).

Duration of Chest Pain: The median duration of chest pain was 2 hours for patients with ACS compared to 6 hours for those without ACS, with no statistically significant difference (p=0.327).

Pain Intensity: Patients diagnosed with ACS and presenting with chest pain had a statistically higher pain intensity score on the Visual Analog

Scale (VAS) compared to those without an ACS diagnosis (7 vs 3, p=0.001).

This analysis highlights various factors associated with Acute Coronary Syndrome (ACS) in patients presenting with chest pain, emphasizing the importance of gender, cardiovascular risk factors, smoking history, and chest pain characteristics, duration of pain, and pain intensity in the evaluation and management of ACS cases. (Table 10)

Table 10: *Factors Associated with Coronary Chest Pain in Young Subjects Under 45 Years Old*

VAS: Visual Analog Scale

Variable	ACS	No ACS	P	OR
Gender			0.004	12.5
Male	35	28 (44.4)		(1.5-
Female	(55.6)	10 (90.9)		103)
	1 (9.1)			
Cardiovascular Risk			0.015	3.2 (1.2-
Factors	20	11 (35.5)		8.6)
yes	(64.5)	27 (64.3)		
No	15			
	(35.7)			
Ischemic			0.004	-
Cardiopathy	7 (100)	28 (42.4)		
yes	0	38 (57.6)		
No				
Active smoking			0.001	9 (2.1-
yes	20	11 (35.5)		38.4)
No	(64.5)	15 (83.3)		
	3 (16.7)			
Typical ischemic			0.003	4.3 (1.6-
chest pain	23	11 (32.4)		11.5)
yes	(67.6)	27 (67.5)		
No	13			
	(32.5)			
Median duration of chest pain in hours	2[1–4]	6[1–48]	0.327	
Average VAS	7[4–9]	3[1–6]	0.002	

DISCUSSION

This study demonstrates that young patients examined by a Mobile Emergency and Resuscitation Service (SMUR) team for chest pain and presenting cardiovascular risk factors have a higher risk of acute coronary syndrome (ACS) compared to those without risk factors. Similarly, smoking patients with chest pain have an increased risk of ACS compared to non-smokers. These findings are consistent with the literature; indeed, young patients with ACS often have multiple classical cardiovascular risk factors, with up to 90% of them presenting at least one risk factor (13). Other factors that may play a role, not addressed in this study, include total cholesterol levels and systolic blood pressure. However, smoking remains a significant risk factor for coronary artery disease; it is the most common and modifiable risk factor among young individuals, with a relative risk of 1.36 for a 10-cigarette per day increase (14).

Furthermore, young patients more commonly present with typical chest pain suggestive of ACS than elderly individuals (15). Several studies indicate that young subjects experience more chest pain and typical symptoms than older subjects (16), which could be explained by sensory function impairment in older individuals (17). However, the use of the term "typical" to describe chest pain depends on the semiological description of pain and the clinician's expertise in evaluating this chest pain, especially in subjects with a non-diagnostic ECG (18).

Chest pain can have various etiologies, with cardiovascular causes being the primary consideration due to their life-threatening nature. In our study, 79.7% of young subjects presenting with chest pain had cardiac etiologies, a higher percentage than reported in the literature (3). This difference could be attributed to variations in emergency medical services across countries. In the United States, the first medical contact personnel are not always physicians, and timely transport to the emergency department is crucial for a comprehensive clinical examination and definitive diagnosis (4). ACS is the primary etiology to consider in chest pain; 48.6% of subjects had ACS, with 55.5% having ST-elevation myocardial infarction (STEMI).

Reviewing the literature suggests that in young subjects with ACS, the diagnosis of STEMI is also common (19,20). These patients often have familial cardiovascular risk factors, are smokers, and have abnormal lipid profiles; the most affected territory is the anterior region, whereas it was the inferior region in our study (17).

Psychological stress is an etiology to consider as a differential diagnosis after ruling out severe conditions (10). Patients experiencing psychological distress often report more chest pain. It is important to note that the etiological diagnosis of chest pain is not always evident in the literature cited series (4).

Typically, as demonstrated, STEMI in young individuals is not high-risk; however, young subjects with ACS can be at high risk of severity or mortality, especially in the presence of risk factors such as active smoking, family history of coronary artery disease, diabetes, hypertension, and dyslipidemia, which have a cumulative effect (21).

Strengths and limitations of the study: Our study established those young patients under 45 years with cardiovascular risk factors, a history of heart disease, and smokers are at very high risk of having acute coronary syndrome as the etiology of their chest pain. Therefore, identifying these risk factors is crucial for the regulating physician and guides the decision to engage an intervention team.

However, this study is limited by its retrospective nature, which restricts data collection to what is available in the patient's medical record. The study also suffers from selection bias, as it includes patients triaged by the regulating physician, who attempts to assess urgency through telephone interrogation to justify the need for engaging a medical team to examine and treat the patient. In-hospital patient follow-up is lacking. Results of emergency department or cardiology investigations for patients transported to hospital services are not always available, preventing an analysis of angioplasty rates, culprit artery analysis in young and elderly subjects, and survival analysis with the available data. Improved data synchronization between services will enhance clinical research.

Recommendations: Based on the study's conclusion, we recommend the following measures: Comprehensive assessment of cardiovascular history and classical cardiovascular risk factors (obesity, dyslipidemia, active smoking, hypertension, etc.) should be conducted for every patient presenting with chest pain, especially when seeking assistance through the emergency number 190, for optimal cardiovascular risk stratification. A diagnostic ECG should be performed within 10 minutes for every patient presenting with chest pain. A 12-lead ECG is mandatory, with additional leads as needed. Stratifying patients into very high-risk and high-risk categories is crucial for better destination management and patient treatment. Patients classified as very high-risk should be transported within two hours to a catheterization room for primary angioplasty, following the 2023 ERC recommendations. Synchronization between pre-hospital and in-hospital medical records is essential to promote scientific research and ensure better patient care.

CONCLUSION

Chest pain in young individuals is a common reason for emergency department visits and calls to emergency medical services. Adequate management, including thorough history taking, a detailed clinical examination, and an ECG, is essential for patient risk stratification. Acute coronary syndrome, one of the most feared etiologies, is prevalent, especially in young

individuals with cardiovascular risk factors, particularly smokers. Multidisciplinary coordination between medical regulation, emergency services, and cardiologists is necessary to ensure care in line with international recommendations.

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Guillain–Barré syndrome after an asymptomatic COVID-19 Infection: a case report

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Abstract

Background: Guillain–Barré syndrome (GBS) is an acute, immune-mediated generalized polyradiculoneuropathy often preceded by several infections. In most cases of GBS linked to SARS-CoV-2 worldwide, the infected individuals developed COVID-19 symptoms. This case report aims to present a case of a GBS post asymptomatic COVID-19 infection discovered in the emergency department of the regional hospital of Ksar Hellal.

Case Presentation: A 55-year-old man consulted the emergency department complaining of lumbar pain treated as a lumbar discal hernia. After 2 days, he developed acute weakness in the lower limbs. Neurological examination revealed a sensory-motor deficit in the lower limbs. The evolution was: absent deep tendon reflexes in the lower limbs, ascending flaccid symmetrical limb paralysis with tetraplegia, swallowing disorder, urinary incontinence, and central respiratory depression. A clinical diagnosis of GBS was made. The patient was transferred to the intensive care unit. He required invasive mechanical ventilation. Cerebrospinal fluid analysis revealed albumin cytologic dissociation. The serology was in favor of a recent COVID-19 infection. The patient was treated with intravenous immunoglobulin at 0.4 g/kg/day for 5 days. His clinical condition improved gradually. He was transitioned out of the intensive care unit after one month.

Conclusion: During the pandemic, all patients with suspected GBS should have SARS-CoV-2 tests to allow eventual rapid isolation. We expect an increase in the rate of GBS without an obvious cause, as long as one can have GBS after an asymptomatic COVID-19 infection. The doctor should be standing by to provide rapid, adequate assistance if required.

Keywords: Guillain-Barré syndrome; COVID-19; Tunisia; Case Report

INTRODUCTION

In December 2019, the novel coronavirus (SARS-CoV-2) was detected in China and has spread worldwide, causing a pandemic: the coronavirus disease-2019 (COVID-19) (1). After an incubation period of 5 days, the respiratory symptoms are the most frequent. Acute renal failure, gastrointestinal, and cardiac damage have also been reported (2). Several neurological symptoms have been related to COVID-19: loss of taste and smell, headache, myalgia, dizziness, and confusion (3).

Neurological complications such as encephalitis and stroke have occurred after COVID-19 infection (4).

Guillain–Barré syndrome (GBS) is an acute, immune-mediated generalized polyradiculoneuropathy often preceded by several infections: mainly Campylobacter Jejuni, Epstein–Barr virus, Cytomegalovirus, Influenza, Zika, Human Herpes virus, less frequently H1N1, HIV and Hepatitis-E (5). Around 100 cases of a confirmed or suspected GBS have been linked to SARS-CoV-2 worldwide (6, 7). Only a few cases were reported in Tunisia (8, 9). We report a case of a GBS diagnosed in a patient initially presenting without symptoms of COVID-19 in a regional hospital.

CASE REPORT

A 55-year-old man, a retired teacher with no medical condition, presented to the emergency department complaining of acute lumbar pain, tingling, and numbness in the right leg progressing within one day. He was treated for a lumbar discal hernia. After two days, he developed acute weakness in both legs and returned. He denied having respiratory or digestive symptoms. On examination, the patient was afebrile; vital parameters and lung auscultation were all normal. Neurological examination showed a sensory-motor deficit in the distal lower extremities. As his wife was tested positive for SARS-CoV-2 five days previously, the patient was admitted to the COVID ward where he was treated symptomatically with paracetamols. He had a negative PCR test for SARS-CoV-2. Laboratory testing revealed no significant abnormalities. Brain computed tomography revealed the presence of a hypodense area of the left semi-ovale centrum, which could be in line with a lacunar cerebral infarct.

The next day, a physical examination found that the lower extremities' deep tendon reflexes were absent, and their strength was significantly lower than that of the upper extremities. His symptoms progressed, and he developed ascending flaccid symmetrical limb paralysis. On day 6 of admission, the patient manifested tetraplegia, peripheral facial paralysis, swallowing disorder, urinary incontinence, and central respiratory depression with a respiratory rate of 35 cycles per minute and an oxygen saturation of 90 % under 15 L of oxygen. A clinical diagnosis of GBS was made. At biology control, there was an appearance of cytolysis, aspartate aminotransferase (ASAT) at 107 UI/L and alanine aminotransferase (ALAT) at 152 UI/L, at three times normal and biological inflammatory syndrome, CRP at 129 mg/L. The patient was immediately transferred to the intensive care unit, where he required invasive mechanical ventilation. Cerebrospinal fluid analysis revealed albumin cytologic dissociation: Glucose at 4.4 mmol/L, proteins at 1.21 g/L, and chlorides at 123 mmol/L with normal levels of red and white cells at 2 elements /mm^3 in the cytological formula. The diagnosis of GBS was confirmed. A serology test, in favor of a recent COVID-19 infection, explained the etiology of

the GBS. The patient was treated with intravenous immunoglobulin (IV Ig) at 0.4 g/kg/day for 5 days.

His clinical condition improved gradually. On day 27, the patient was extubated and was discharged from the intensive care unit to a rehabilitation facility.

DISCUSSION

We report a case of GBS with a serology suggesting a recent COVID-19 infection. GBS is a rare, serious, post-infectious, immune-mediated disease of the peripheral nervous system. Previous studies have suggested that GBS was not associated with COVID-19 (10). However, the incidence of GBS has significantly increased since the onset of the COVID-19 pandemic. Many reports have described the association between SARS-CoV-2 infection and GBS, which is supported by the chronology of the GBS cases following the same propagation pattern of COVID-19 worldwide (11). Numerous hypotheses have been proposed to explain the association pathogenesis, including immune dysregulation and systemic inflammation (12). Following infection, there is the generation of antibodies against surface glycoproteins or epitopes (spike) of SARS-CoV-2. These antibodies bind the gangliosides present in peripheral neurons due to the structural resemblance of the SARS-CoV-2's epitopes with gangliosides (molecular mimicry) (13). The systematic inflammation theory is explained by the massive cytokine release in infected patients, which may also contribute to amplifying the dysimmune process underlying GBS (14). A mean age of 50 years and male predominance were noticed among patients with the association of both diseases (7, 11).

Inconsistent with most literature data, which reported that respiratory symptoms typically precede the onset of neurologic symptoms (6, 7, 11), our patient was asymptomatic but had evidence of a COVID-19 infection. The median latency period between the COVID-19 infection and GBS was 14 days for most reported cases, supporting the post-infectious immunopathogenesis mechanism.

This duration could be longer than reported, as COVID-19 can initially be asymptomatic (6, 7). Few GBS reported cases had a para-infectious profile[17] as reported with the Zika virus (15 – 18). Nevertheless, the chronology of GBS preceding COVID-19 symptoms, described with few cases, has not been previously reported with other viral agents (6). A systematic review of 73 GBS cases associated with COVID-19 published from January 2020 to July 2020 (11) revealed that only two cases never developed COVID-19 respiratory or systemic symptoms but tested positive for SARS-CoV-2 (19, 20). Our findings were consistent with those of the literature concerning common symptoms of GBS and their mean time to nadir, which was equal to 5 days (range, 1.5–10 days), according to JB. Caress *et al*. (7) and the results of cerebrospinal fluid analysis as albumin cytologic dissociation being the most frequent finding (6,7,11). Like our patient, most reported cases received intravenous immunoglobulin therapy (6). In

terms of clinical outcomes, the admission to the intensive care unit and the requirement of mechanical ventilation were reported respectively in 40 and 33 of 109 cases by M. Aladawi *et al*. (6) JB. Caress *et al*. have reported the response to therapy in 33 of 37 (89%) patients (7).

CONCLUSION

This case report raises several concerns about the few cases of GBS following an asymptomatic COVID-19 infection and brings more attention to the possible atypical association between them. However, the main clinical, radiological, and CFS features of our case report are found to be similar to GBS cases associated with other infectious diseases. Therefore, during the pandemic, all patients with suspected GBS should have SARS-CoV-2 tests to allow eventual rapid isolation. We expect an increase in the rate of GBS without an obvious cause, as long as one can have GBS after an asymptomatic COVID-19 infection. The doctor should be standing by to provide early adequate assistance if required to prevent worse outcomes. Further studies to explore the immunogenicity of COVID-19 in the development of GBS are necessary and should consider the variations between different populations.

Consent

Written informed consent for publication of their clinical details and/or images was obtained from the patient.

Data availability statement

All data underlying the results are available as part of the article and no additional source data are required.

Competing interests

No competing interests were disclosed.

Grant information

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Atrial flutter mimicking ST-elevation myocardial infarction: A case report

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Abstract

The diagnosis of acute ST-segment elevation myocardial infarction (STEMI) is crucial in the emergency department for the rapid initiation of reperfusion therapy. However, many situations of non-coronary obstruction can mimic the ECG findings of a STEMI. These features are confused with myocardial infarction (MI) on the 12-lead ECG and lead to inappropriate activation of catheterization labs or thrombolytic therapy. In this report, we describe a case of STEMI mimicry produced by prominent atrial flutter waves.

Keywords: Atrial flutter; Myocardial infarction; Diagnosis; Management; Prehospital

CASE REPORT

A 45-year-old man with no previous medical history presented to the emergency department of a primary care hospital with palpitations and chest heaviness. Initial ECG showed narrow-complex tachycardia at 170 bpm with infero-lateral ST-segment elevation (Figure 1).

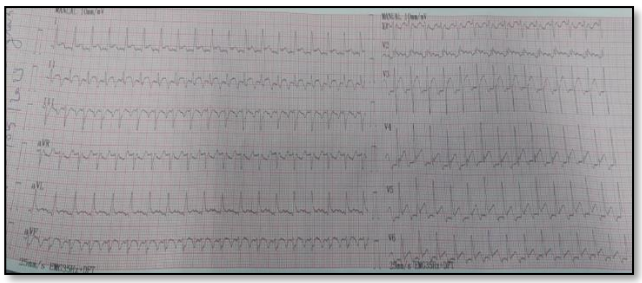


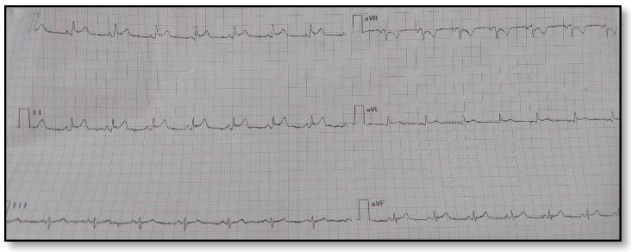
Figure 1: *Initial electrocardiogram*

He received dual antiplatelet therapy (DAPT), unfractionated heparin, analgesics, oral Bisoprolol, and intravenous Amiodarone. The pre-hospital dispatch center was alerted to a STEMI.

The emergency physician found a conscious patient with systolic blood pressure at 120 and diastolic at 80 mm Hg, tachycardia at 180 bpm, and polypnea with crepitant at the base of the lungs. He was treated with 40 mg furosemide, titrated with nitrate, and transferred to the cardiac catheterization laboratory. The coronary angiography was performed and found normal coronary arteries.

On discharge, the patient presented with hypotension and exacerbation of pulmonary edema with persistent arrhythmia. The diagnosis was mal tolerated flutter, requiring electrical cardioversion to sinus rhythm. The ECG showed sinus rhythm with an aspect of early repolarization. (Figure 2 A, Figure 2 B)

A



B

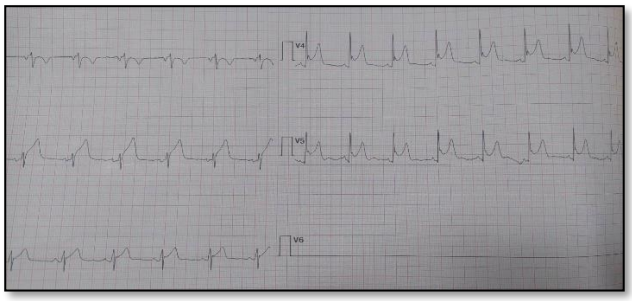


Figure 2: *Post-cardioversion electrocardiogram showing early repolarization in inferior (2A) and anterior (2B) leads*

After electrical cardioversion with confirmation of sinus rhythm, the patient's symptoms resolved, and serial cardiac markers were normal.

DISCUSSION

The 12-lead surface electrocardiogram (ECG) is the cornerstone of prompt acute coronary syndrome (ACS) diagnosis and management, namely ST-elevation Myocardial Infarction (STEMI). The rapid and accurate diagnosis of this critical illness can lead to rapid reperfusion, and it enables the reduction of cardiac ischemic damage and results in improved subsequent outcomes.

However, other conditions aside from STEMI can cause ST-segment elevation on the ECG. Some studies reported that the prevalence of false-positive cardiac catheterization laboratory activations was between 9.2-14%(1)

The ECG remains an imperfect diagnostic tool. Some patients present with classic symptoms and findings; however, around 60-80% of patients with ST-segment elevation on ECG are ultimately not associated with STEMI (1, 2).

Atrial flutter waves, particularly 2:1 atrial flutter, can distort the ST segment in such a way as to mimic a lesion on the electrocardiogram. Flutter waves can mimic ST-segment elevation or depression (3).

However, there have been no previous reports of atrial flutter, masking ST-segment elevation. Atrial flutter has not been included or considered a mimic or confounder of acute myocardial infarction (4,5,6).

Allegedly, the prominent flutter waves distorted the ST segment in this patient and misled physicians into initially considering an inferior STEMI. However, the absence of reciprocal ST-segment changes (ST depression) in the precordial leads could have been a clue. The second ECG ruled out Q waves but created confusion by showing greater ST-segment elevation in precordial leads (V2-V4).

The third ECG confirmed that these changes already existed, although it is still debatable whether there is an additional component of early repolarization changes in the precordial leads contributing to the appearance of a myocardial infarction mimic.

This case demonstrates that atrial flutter waves can mimic ST segment changes, which has been described previously (7)

CONCLUSION

Patients with atrial flutter may present with STEMI due to flutter waves coinciding with ST

segments and Q waves. A repeat ECG with slower atrioventricular conduction during flutter can reveal the diagnosis.

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Bilateral ptosis caused by midbrain hemorrhage: A case report

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Abstract

Ptosis occurring in cases of cerebral ischemic or hemorrhagic stroke, trauma or tumor without involvement of brainstem or oculo-sympathetic mechanism, can be termed as cerebral ptosis. Such eyelid dysfunction can occur with hemispheric involvement of either side. It is associated with a higher frequency of gaze preference to the side of lesion and upgazed limitation compared to patients without cerebral ptosis. Cerebral ptosis is rare. Eliminating an emergency is a real challenge in the face of bilateral ptosis. We report a case of bilateral ptosis occurring after a mesencephalic hematoma. The patient was unable to open her eyes or perform the basic activities. Neurogenic ptosis may improve after treatment of the underlying cause.

Keywords: Cerebral Ptosis; Cerebral Hemorrhage; Computerized Tomography

INTRODUCTION

Blepharoptosis (Ptosis) is defined by a drooping upper eyelid, making it difficult to open the eye and narrowing the upper eyelid margin [1-3]. is a common condition that can be congenital or acquired and varies in its timing of onset, duration, severity, laterality, and underlying etiology [4]. Several etiologies are possible, including supranuclear lesions, oculomotor complex lesions, oculocephalic lesions, neuromuscular junction dysfunction, neuromuscular diseases, and mechanical abnormalities of the eyelid [1]. Ptosis occurring in cases of cerebral ischemic or hemorrhagic stroke, trauma or tumor without involvement of brainstem or oculo-sympathetic mechanism can

be termed as **cerebral ptosis (CP) [6-10]**. Such eyelid dysfunction can occur with hemispheric involvement of either side. It is associated with a higher frequency of gaze preference to the side of lesion and upgazed limitation compared to patients without cerebral ptosis.

The vascular supply of midbrain is complex [1,5]. It is ensured by the posterior cerebral artery (PCA), the basilar artery, the superior cerebellar artery and the anterior choroidal artery [5]. The oculomotor nucleus located in the midbrain at the level of the superior colliculus sends efferent fibers to the medial, the superior and the inferior oblique extraocular muscles. The elevator palpebrae superioris muscle is also innervated by the oculomotor nerve. Its origin is from the central caudal nucleus (CCN) [2]. Midbrain lesions involving the CCN have been reported to cause bilateral ptosis [3]. Besides, pure midbrain infarction involving the anteromedial areas can cause bilateral ptosis, in addition to the median longitudinal fasciculus syndrome, and contralateral cerebellar ataxia [3]. The midbrain is often affected in patients with embolic stroke occurring in the posterior circulation, usually with the concomitant involvement of other structures, such as the pons, thalamus, and the cerebellum. Although midbrain infarcts and particularly hemorrhages are uncommon, their clinical manifestations are diverse mainly because the vertical gaze centers and two of three nuclei of the extraocular muscles lie primarily in the midbrain. Consequently, eye movement disturbances are often the hallmark clinical findings in midbrain stroke or hemorrhage [1]. The reported prevalence of pure midbrain hemorrhage varies from 0.7% to 2.3% [1,2]. Ptosis could be caused by oculomotor nerve palsy in the patients with midbrain infarction [1]. In several cases, bilateral ptosis showed the clinical characteristics of midbrain infarction [1,3,4]. We experienced a case of severe bilateral ptosis that occurred after midbrain hemorrhage in which the patient could not open her eyes and was limited in basic activities and mobility. We herein report the case with a review of relevant literature.

CASE REPORT

A 65 -65-year-old woman with a previous history of hypertension was admitted to the emergency department because, suddenly, she could not open her eyes. She did not complain of headache, nausea, fever, or vomiting. Examination revealed pronounced bilateral ptosis, with symmetrical and intermediate pupils. There was marked limitation of adduction, moderate limitation of depression and elevation in the movement of both eyeballs. Abduction was normal. The patient was respiratory stable. Her SBP was 170 mmHg and DBP 100 mmHg. The rest of the examination was normal, her consciousness was clear, her Glasgow coma scale was 15, no motor or sensory deficits, no localization signs and no evidence of other orbital disease-causing ptosis in an ophthalmologic examination. Blood tests and electrocardiography did not reveal any abnormal findings. Brain computed tomography (CT) on admission showed a small

hemorrhage in the medial side of the left crus cerebri without subarachnoid hemorrhage (Figure 2). The patient was initially admitted to the emergency room, where her blood pressure and neurological status were monitored, antihypertensive treatment was started (nicardipine). The outcome was favorable with stabilization of blood pressure. The next day, she was transferred to the department of rehabilitation medicine for intensive rehabilitation management. Participation in the rehabilitation programs was difficult due to continuing inability to open her eyes. We tried taping her lids with Micropore tape and used lubricants to prevent exposure keratopathy. However, this method was less efficient due to blinking. Fixation of the upper eyelid to the supraorbital structures using Eye-putti eyelash glue proved more effective in conjunction with a rehabilitation program. About two months after the cerebral hemorrhage, the bilateral ptosis improved. The patient was satisfied with the rehabilitation program. She performed activities of daily living and mobility with minimal assistance. The ptosis was gradually resolved after five months.

Figure 1: *Follow-up photographs of the patient's eyelids*

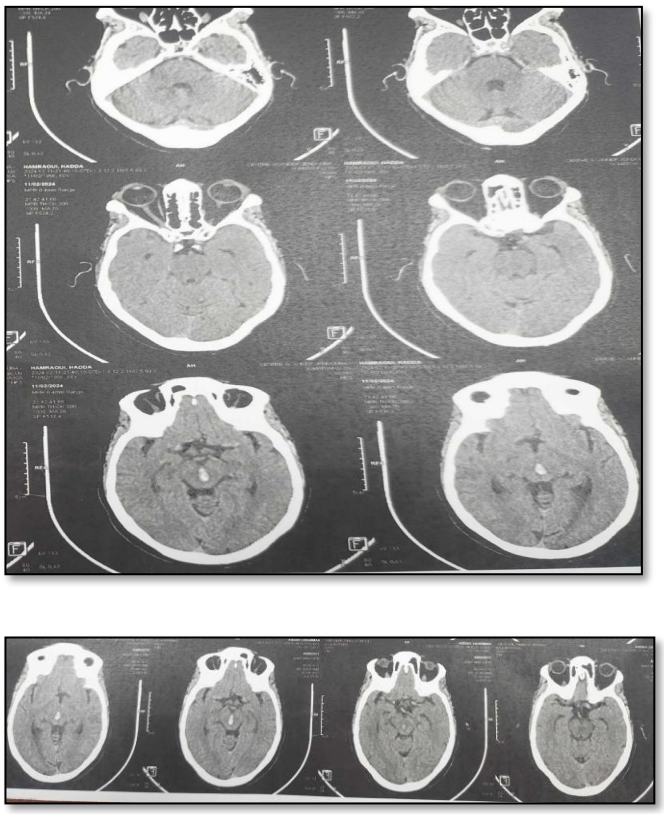

Figure 2: *Computed tomography scan showing hemorrhage in the midbrain tegmentum.*

DISCUSSION

Bilateral ptosis is a rare manifestation and has been reported in 5% of patients with pure midbrain infarction in a previous case series and most of these patients had small lesions in the paramedian or central regions of the midbrain [10-14].

The criteria for the diagnosis of CP have not yet been established. Manconi et al. [10] proposed inclusion and exclusion criteria. The reported inclusion criteria were as follows:

1) sudden bilateral lid drops within 48 hours of stroke or hemorrhage,
2) voluntary, spontaneous, and automatic impairment in eyelid opening,
3) preserved voluntary frontalis muscle

contraction

4) And neuroradiological evidence of supratentorial ischemic or hemorrhagic damage.

The exclusion criteria were presented together to distinguish the following causes of ptosis: apraxia of lid opening (ALO), impaired consciousness, blepharospasm, intrinsic oculomotor dysfunction, neuromuscular disease, brain stem dysfunction, and sub tentorial lesions. In our case, ALO and blepharospasm should be differentially diagnosed [10].

Although the mechanisms and clinical significance of CP remain uncertain, experiments in both animals and humans have shown that opening of both eyelids occurs upon stimulation of

the frontal and occipital lobes [15]. Clinically, it has been hypothesized that eyelid motor control might be lateralized to the right hemisphere in complete CP [9, 10,11]. However, the specific mechanism is not yet fully elucidated, and the above hypothesis is still controversial [5]. The prognosis of CP is variable and depends on the lesion and etiology [5]. Manconi et al. [10] reviewed literature on 75 CP patients, reporting an improvement in ptosis in 70.9%, with an average recovery time of 7.5 days. In most cases of spontaneous midbrain hemorrhage, conservative, supportive treatment is sufficient to ensure a good outcome. Some 25% of patients had no neurologic deficits and 41% of patients had persistent minor neurologic deficits, which related chiefly to cranial nerves III and IV. Only 4% of the patients died [1,10].

In summary, this case demonstrates complete ptosis resulting from a brainstem event. We hypothesize that the patient's oculomotor deficits were secondary to midbrain hemorrhage secondary to hypertension. Additional cases with documented clinical, neuroradiologic, and neuropathologic examination can reinforce our understanding of eye movement disorders and the anatomic organization of the oculomotor nucleus and associated nuclei that mediate conjugate gaze.

Ethical Statement

This research was conducted ethically: Ethics approval was not required by the Ethics Committee because this is a case report. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Funding Sources

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Successful Conservative Management of Penetrating Cervical Tracheal Injury: A Case Report

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Abstract

Penetrating tracheal injuries are rare but potentially life-threatening, often associated with significant morbidity and mortality. This case report presents the successful conservative management of a 21-year-old male who sustained a penetrating cervical injury involving the trachea. The patient was admitted to the emergency department following an assault with a sharp object (screwdriver). Initial clinical examination revealed severe tachypnea, subcutaneous emphysema, and bilateral pneumothorax, confirmed by a computed tomography (CT) scan. The scan further revealed a small tracheal wound (less than 1 cm) and associated pneumomediastinum and pneumopericardium.
This case underscores the importance of early diagnosis and highlights that conservative management, in select cases of tracheal injuries, can lead to favorable outcomes without surgery.

Keywords: Tracheal trauma; Emergency; Management; Conservative management.

INTRODUCTION

Penetrating neck injuries account for 5 to 10% of all traumatic injuries. They can be life-threatening injuries, due to the dense concentration of vascular and neural structures, as well as the presence of the upper aerodigestive tract [1]. The incidence of tracheal injuries in cases of penetrating neck wounds ranges from 3 to 6% [2]. Most of the cervical injuries are associated with mainly assaults, and suicide attempts in fewer cases, involving sharp objects or firearms. Given the severity of injuries in the cervical region, any penetrating neck wound requires urgent medical and surgical management to assess for signs of critical presentation and to conduct surgical management, if necessary.

Initial management must prioritize identifying life-threatening injuries, particularly acute respiratory distress due to upper airway (UA) injury or massive bleeding caused by vascular damage. Recognizing subtle signs of upper airway injury secondary to cervical trauma is crucial for ensuring early diagnosis and prompt intervention, which can improve survival rates and minimize long-term complications. Early detection of tracheal injuries significantly reduces morbidity and mortality [3].

According to the literature, one in three cases of penetrating cervical injuries results in bleeding, and one in ten results in direct injury to the UA, sometimes accompanied by esophageal involvement [4]. It is important to rule out esophageal injury when the UA is affected, as these injuries are commonly overlooked initially and can later manifest as severe septic complications.

The UAs (pharynx, larynx, trachea) are exposed throughout the neck and may be directly injured or compressed by a hematoma [4,5]. The clinical presentation of tracheal trauma varies depending on adjacent structures. Subcutaneous emphysema, pneumomediastinum, and pneumothorax, with or without respiratory failure, are the most observed acute clinical features [6,7]. Pneumomediastinum can further complicate the condition with cardiac tamponade or airway compression [8].

CASE REPORT

A 21-year-old male with no notable medical history was the victim of an assault involving a sharp object (screwdriver) in the anterior cervical region in June 2024 (Figure 1).

Upon initial clinical examination, his Glasgow Coma Scale score was 15/15, blood pressure 120/80 mmHg, and SpO2 92% on room air. He presented with severe tachypnea, a respiratory rate of 40 breaths per minute, with a clear upper airway.

Physical examination found subcutaneous emphysema in the thoracic and cervical regions. Due to respiratory distress, the patient was sedated, intubated, and ventilated.

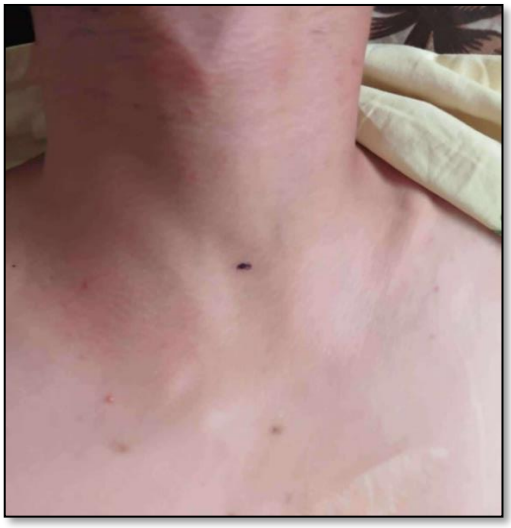

Figure 1: *On-admission presentation of the patient*

A CT scan of the cervical, thoracic, abdominal, and pelvic regions revealed extensive subcutaneous emphysema in the cervical soft tissues, extending into the deep soft tissues of the face. A millimeter-sized ascending anterior cervical wound dissecting the sternohyoid muscle and reaching the anterior aspect of the subglottic larynx was observed, with associated fat densification and a tracheal wound less than 1 cm in size, which was not initially identified during the first review of the scan. In the thoracic region (Figures 2 and 3), a large right anterior pneumothorax caused a collapse of the lung parenchyma and mediastinal shift to the contralateral side. A moderate left anterior pneumothorax, an extensive pneumomediastinum, a large pneumopericardium, and significant subcutaneous emphysema dissecting the deep thoracic soft tissues were also observed.

The patient underwent chest drainage on the right side, and an otolaryngology examination was performed, indicating the need for an urgent surgical repair.

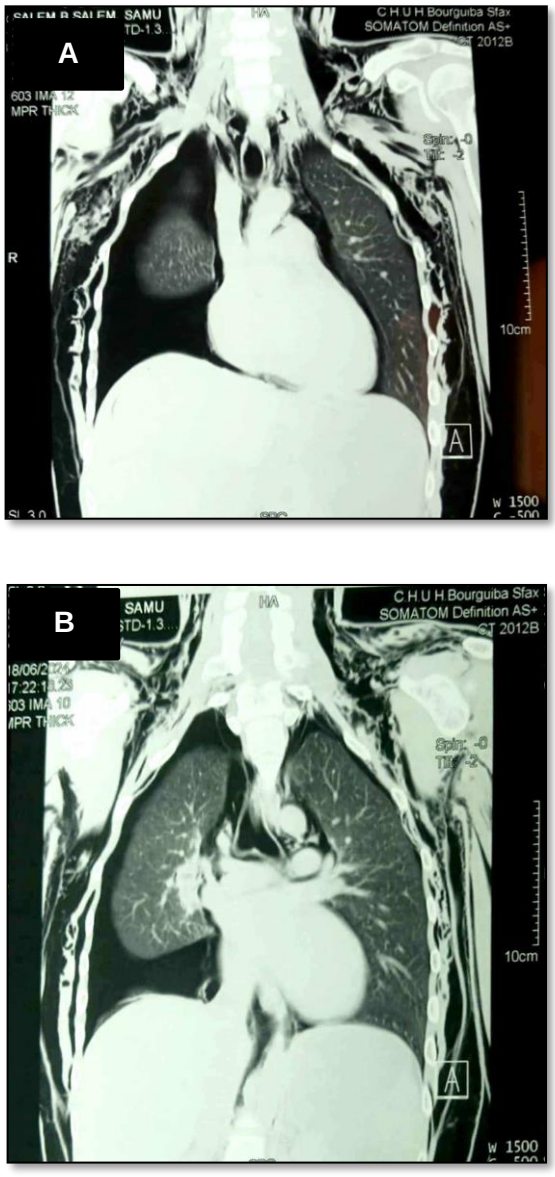

Figure 2: *The CT scan showing an anterior pneumothorax on the right side (A) with a collapsed lung and a deviation of the mediastinum to the left side (B)*

The multidisciplinary decision was to keep the patient intubated for 24 to 48 hours. The decided management plan for the small tracheal wound was close monitoring alongside chest drainage.

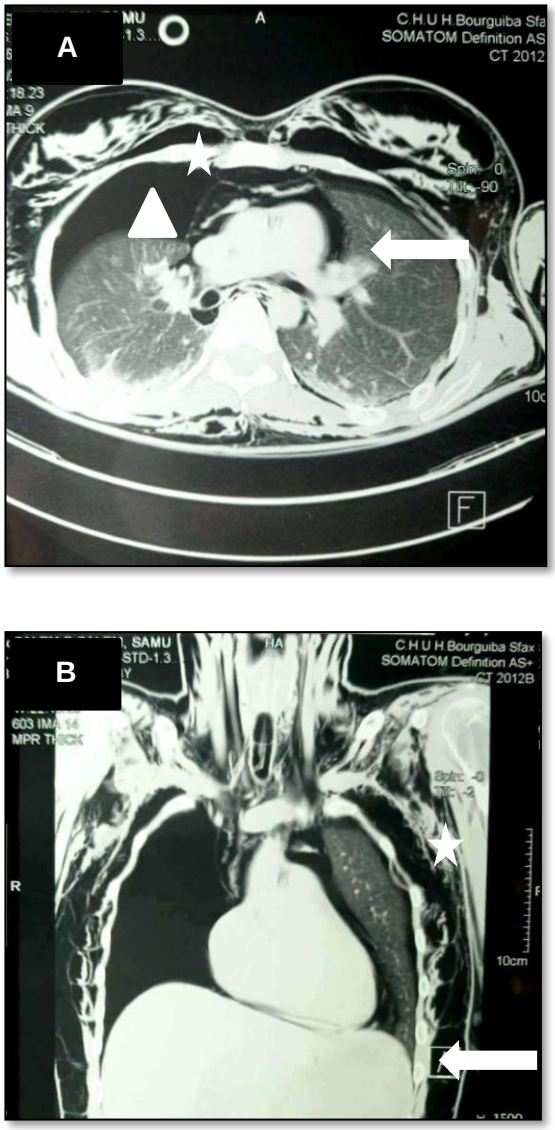

Figure 3: *The CT scan showing a pneumomediastinum (Triangle) with a pneumopericardium (arrow) with an important emphysema (star) in axial (A) and sagittal (B) plans*

The patient was then transferred to the intensive care unit. He sustained hemodynamically stable without the need for catecholamines but with extensive subcutaneous emphysema extending from the chest to the lumbar region and palpebral edema. The therapeutic protocol continued to be conservative including monitoring, proton pump inhibitors, corticosteroids, antibiotics, and

preventive anticoagulation. The outcome was favorable, with a transfer to the thoracic surgery department, and then home discharge.

DISCUSSION

The anatomy of the cervical region is complex with vital structures (vascular, neurological, respiratory, and digestive) within a limited space, surrounded by rigid fascia. This makes cervical wounds, particularly those involving the airway, highly challenging to assess and manage [9].

In the case of cervical injuries, it is crucial to look for expanding hematomas, airway deviation, or subcutaneous emphysema. Physical examination should include carotid pulse palpation, auscultation for bruits or thrills, and a thorough neurological assessment to distinguish between central (e.g., hemiplegia) and spinal cord injuries (e.g., tetraplegia, priapism, anal hypotonia). Clinical signs of tracheal injury, such as subcutaneous emphysema, pneumomediastinum, and pneumothorax, are common [5-7]. However, many patients may present with minimal symptoms, making early diagnosis critical.

Chest radiography is essential for evaluating pneumothorax, pneumomediastinum, or tracheal deviation, which could indicate tracheal damage. A CT scan of the cervical and thoracic regions confirms the diagnosis and provides a complete assessment of associated injuries (e.g., esophageal, pulmonary, or vascular). Bronchoscopy remains the gold standard for diagnosing tracheobronchial injuries but is sometimes avoidable with high-quality CT imaging [10-14].

Management depends on the clinical stability of the patient. Conservative treatment with corticosteroids, proton pump inhibitors, and humidified oxygen is effective for small tracheal lacerations (<2 cm). Larger or unstable injuries require surgical intervention, such as tracheal repair or anastomosis [15-21]. In cases of hemodynamic instability, extracorporeal membrane oxygenation (ECMO) may be considered a bridge to surgery [11, 22]. Postoperatively, patients should be monitored for complications such as tracheal stenosis or dysphonia [23].

In patients with respiratory and hemodynamic stability and moderate tracheal lesions less than 2 cm, conservative treatment with systemic corticosteroids, proton pump inhibitors, and possibly antibiotic prophylaxis, with humidified air, can be initiated. Conservative management may be superior to surgical treatment in well-selected patients with moderate injuries. Small tracheal wounds may close spontaneously within 48 hours [13].

Rehabilitation, including speech therapy, is important to optimize functional outcomes. The risk of high morbidity and mortality associated with tracheal injuries—due to complications like pneumomediastinum or pneumothorax—requires prompt and accurate intervention [24, 25].

CONCLUSION

Penetrating neck wounds are notoriously challenging to assess due to the complexity of the anatomical region. The incidence of tracheal

injuries in the context of penetrating neck trauma is between 3% and 6%.

The clinical presentation of tracheal trauma can vary depending on the involvement of adjacent structures. Subcutaneous emphysema, pneumomediastinum, and pneumothorax are the most commonly observed acute clinical features with or without respiratory distress.

Accurate interpretation of chest X-rays is crucial for the early diagnosis of occult upper airway injuries in stable patients. Cervical-thoracic CT scans confirm the diagnosis and provide a listing of tracheal injuries and surrounding lesions, namely esophageal, pulmonary, or aortic involvement.

This case underscores the importance of early diagnosis and highlights that conservative management, in select cases of tracheal injuries, can lead to favorable outcomes without surgery.

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The challenge in diagnosing human rabies: A case report

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Abstract

Introduction

Rabies is a zoonotic disease caused by a neurotropic virus of the Lyssavirus genus. Human rabies can manifest in either encephalitic (furious) or paralytic (dumb) forms. The diagnosis is still challenging and often delayed. Viral infection must be considered and treated soon after viral transmission; failure to diagnose and intervene will usually result in disease progression and death.

Case presentation

We report a case of a 61-year-old female, with no past medical history, who visited the Emergency Department (ED) initially for sleep disorders, restlessness, anxiety, refusal of food and watery disgust. The investigations were normal, and the patient was discharged with symptomatic treatment. Her condition worsened after 24 hours, and she presented a cardiac arrest. After resuscitation and return of spontaneous circulation, her physical exam was without abnormalities. Laboratory exams and imaging investigations were normal. The diagnosis was a severe septic shock, treated with empirical antibiotics. A multiple organ failure syndrome has rapidly appeared, and the outcome was fatal.

Upon re-interviewing the family, it turned out that the patient was raising around 20 cats and that she was a victim of an accidental bite by one of her cats 3 weeks before admission. No medical advice was sought, and the family reported a completely healed wound. The patient did not receive any vaccination or serotherapy. Rabies serology came back positive. The diagnosis was confirmed after brain biopsy.

Conclusion

Human rabies is a challenging disease, with a complex neuropathogenic mechanism. The infection can be treated after recognized exposures. However, medical management, once the clinical disease develops, has almost universally proved to be unsuccessful, resulting in fatal outcomes.

Keywords: Encephalitis, Rabies, Diagnosis, Challenge.

INTRODUCTION

Rabies is a zoonotic disease caused by a neurotropic virus of the Lyssavirus genus. The virus is transmitted from animals to humans by bite, scratch or direct exposure of mucosal surfaces to saliva from an infected animal (1). Human rabies can manifest in either encephalitic or paralytic forms (2). The clinical stages are incubation, prodrome, acute neurological signs, coma, and inevitable death. The diagnosis is still challenging and often delayed, particularly the paralytic form (3).

Infection with the virus must be considered and treated soon after viral transmission; failure to diagnose and to intervene will usually result in disease progression and death (2).

We report a case of a patient who presented to the Emergency Department (ED) with a .

Observation

A 61-year-old female with no past medical history presented to our ED for sleep disorders, restlessness, anxiety, refusal of food, and watery disgust for two days. On admission, the patient was agitated, and the laboratory investigations were normal. Due to the pandemic context of COVID-19, a PCR test was performed and was negative. A brain and chest CT scans were normal. The patient was discharged with symptomatic treatment.

After 24 hours, her condition worsened; she had abdominal pain, and her consciousness altered. Upon arrival, the patient presented with cardiac arrest resuscitated within two minutes. After the return of spontaneous circulation, the physical examination found a normal temperature, blood pressure was 100/58 mmHg with 1mg/h of adrenaline, no peripheral signs of shock and normal respiratory parameters. No neurological signs were noted. The ECG did not show arrythmias or electrical signs of myocardial ischemia. Initial diagnosis was septic shock. The patient received empirical antibiotic therapy, catecholamine. Cerebrospinal fluid analysis was performed and was normal. Faced with the absence of an obvious cause for the cardiorespiratory arrest, shock, and initial agitation, a toxicological assessment was requested and was negative. On the fifth day of hospitalization, the patient presented multiple organ failure and death.

Upon re-interviewing the family, it turned out that the patient was raising around 20 cats and that there was the notion of an accidental bite by one of her cats 3 weeks before admission. No medical advice was sought, and the family reported a completely healed wound. The patient did not receive any vaccination or serotherapy. Rabies serology came back positive, and the diagnosis was confirmed after brain biopsy.

DISCUSSION

Rabies is a neurotropic RNA virus transmitted to humans through the saliva of infected animals, usually from bites (4). The virus is almost invariably fatal after the onset of neurologic symptoms (2). The rabies virus reaches the brain by centripetal propagation

mediated by retrograde trans-neuronal transfer and the clinical stages of rabies are incubation, prodromes, acute neurological signs, coma, and death (3). The incubation period or eclipse phase can vary from weeks to years but lasts 1-3 months on average (5). The cause of this variation is probably multifactorial including the site of virus entry and the viral load, the species and strain of the infecting virus, and the immunological competence of the host (6). Nonspecific prodromal symptoms include malaise, headache, fever, anxiety, and agitation. Paresthesia, pain, and pruritus are the earliest neurologic symptoms (5). Two classical forms of rabies are generally recognized: furious (also called encephalitic) which develops in approximately 80% of cases, and paralytic, which occurs in approximately 20% of cases. The predicting factors associated with the development of either form remain unclear (7,8). Specific symptoms are described in each form. However, case definition can typically be established with certainty only when the disease reaches the acute neurological phase (6). The paralytic form of disease differs from the encephalitic form in that muscle weakness develops early, whereas progression to coma and death often take longer than with the encephalitic form (3). In our case, the patient presented with signs and symptoms that commonly occur in the encephalitic phase of rabies, including agitation, hyper-excitability, and hydrophobia.

Infection with rabies virus can be difficult to diagnose ante-mortem (3). Hydrophobia is the most characteristic and no clinical signs of disease are pathognomonic for rabies (9). Given that the differential diagnosis for altered mental status is broad (reflecting impairment of affect, behavior, or cognition), the workup should start with reversible and life-threatening causes as recalled by use of the mnemonic “rule-out the WHIMPS.” Each letter of this acronym signifies 1 or more of the following conditions: Wernicke encephalopathy, hypoglycemia, hypoxia, hypoperfusion of the central nervous system, and hypertensive encephalopathy, infections and intracranial processes, metabolic derangements (such as hyponatremia/ hypernatremia, hypocalcemia/hypercalcemia, and hyperammonemia), poisons, and seizures. The workup should start with taking a history, looking for clues to etiology and to temporal relation-ships with symptoms, and then proceed to a thorough physical examination and laboratory testing (an electrocardiogram, a complete blood count, a comprehensive metabolic panel, a toxicology screen, as well as measurement of levels of B12, ammonia, and thyroid-stimulating hormone, and a radiologic examination for intracranial lesions via computerized tomography or magnetic resonance imaging, a chest x- ray, and a urinalysis). Further diagnostic tests (such as electroencephalography and cerebrospinal fluid exam) may also be considered. In the appropriate setting (fever, flulike symptoms, or cerebrospinal fluid inflammation), microbiologic assays (spirochetes) would be recommended. In patients with a longer duration of altered mental

status symptoms, neuro-psychologic testing and functional imaging may be ordered. In addition to pursuing potential medical and neurologic etiologies, psychologic ones must be investigated (5). In our case, there was no reported history of bite or scratch, and the symptoms were not specific.

The management of clinical rabies in nonvaccinated patients is largely palliative, and death is invariably expected (2). The onset of rabies clinical symptoms, and death can be prevented by adequate post-exposure prophylaxis (PEP) including vaccines and, if required, rabies immunoglobulin (RIG) (9).

CONCLUSION

Human rabies is considered as a disease of complex neuropathogenic mechanisms and challenging diagnosis. The infection can be treated after recognized exposures; however, medical management once the clinical disease develops has almost universally proved to be unsuccessful, resulting in fatal outcomes.

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Multivisceral Damage Following Acute Methotrexate Intoxication By Dosing Error

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Abstract

Background: Methotrexate (MTX) is a chemotherapeutic agent commonly used for the treatment of nonmalignant and malignant conditions. It remains the principal medication used to treat rheumatoid arthritis. High-dose MTX is known to cause significant injuries, including acute renal failure, hepatotoxicity, myelosuppression, and multiorgan failure.

Case presentation: Here we report a 65 years old female patient with rheumatoid arthritis who had developed oral and mucocutaneous ulceration after daily ingestion of MTX.

Conclusion: This case report highlights the importance of early follow-up and regular laboratory investigations of patients on methotrexate therapy.

KEYWORDS: Methotrexate; Intoxication; Dosing Error; Multivisceral Damage

INTRODUCTION

Methotrexate (MTX) is a folic acid used to treat autoimmune conditions and inflammatory diseases such as rheumatoid arthritis. Daily accidental ingestion instead of one dose per week is a common cause of acute MTX intoxication. Most of the publications state MTX's side effects, but there are very few revealing mortality (1).

Here we report a 65-year-old female patient with rheumatoid arthritis who had developed oral and mucocutaneous ulceration after daily ingestion of MTX.

CASE REPORT

A 65-year-old female was admitted to the emergency department for mucocutaneous ulceration after daily ingestion of MTX. She had hypertension and diabetes. She was recently diagnosed with rheumatoid arthritis 5 days ago, and she had a prescription for MTX. Upon review of the methotrexate dose administration, she reported that she had wrongly taken 15mg for the first 2 days and then 5 mg on the third day.

On physical examination, the patient was conscious and oriented. She had no respiratory nor hemodynamic distress. General examination noted conjunctivitis (figure 1).

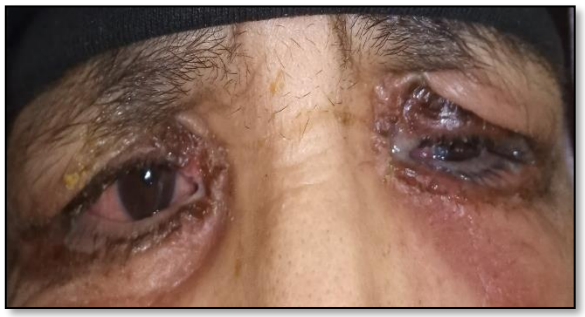

Figure 1: *Bilateral Conjunctivitis related to MTX intoxication*

She was febrile and in reduced general condition. Oral cavity examination showed ulcerated mucosa with bleeding (figure 2). Genital examination

revealed vaginal mucosa with per vaginal bleeding (figure 3).

Figure 2: *Ulcerated mucosa in oral cavity*

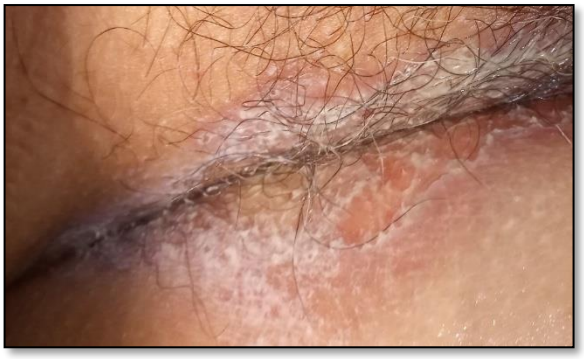

Figure 3: *Genital mucosa (figure 3).*

Blood counts and peripheral blood smear have shown pancytopenia: leucocyte count of 400/uL, Hemoglobin rate of 9.5 g/dl, and platelet count of 79.000/ uL. The liver functions tests showed slightly elevated liver enzymes, and she had acute renal failure (creatinine level of 250 µmol/L). A Biological inflammatory syndrome was detected (Reactive C protein 445g/L). Regrettably, we could not measure the blood level of MTX because of a lack of facilities.

The patient had benefited from hyperhydration and intravenous folinic acid at a dose of 15 mg and was given six hourly (1 mg/kg). Unfortunately, two days after her admission she developed acute respiratory distress for which she was sedated and intubated. Despite aggressive therapy, her blood tests worsened and she developed a multi-organ failure. She deceased four days after admission.

DISCUSSION

This case describes the clinical features of a rare case of acute MTX intoxication. Toxicity from low-dose MTX is uncommon. Most cases are due to failure to follow the prescribed recommendations. (1)

Methotrexate is a chemotherapeutic agent commonly used to treat nonmalignant and malignant conditions. It remains the principal medication used to treat rheumatoid arthritis (2). In therapeutic doses, MTX has anti-inflammatory and immunosuppressive action. Renal excretion is the primary route of elimination, which is influenced by the route of administration and the dosage (3).

As described in several studies, MTX toxicity has an impact on the skin, gastrointestinal mucosa, liver, kidneys, and bone marrow (3,4). High-dose MTX is known to cause significant injuries, including acute renal failure, hepatotoxicity, myelosuppression, and multiorgan failure.

Furthermore, pancytopenia can be one of the complications of methotrexate use whether in low or high doses (5). The mechanism of development of pancytopenia in MTX intoxication is not clear. It is commonly seen in therapeutic doses when the patient presents some risk factors such as dehydration, renal failure, or hypoalbuminemia (6). In the same wavelength, Calvo Romero discovered that pancytopenia caused by a therapeutic dose of methotrexate is more likely to

progress in the presence of renal failure (7). Grissinger reported one such similar case where the patient took the drug daily instead of weekly (8). In our case, with no history of renal failure, our patient had developed pancytopenia. Concerning thrombocytopenia, Paul et al have demonstrated that MTX could promote the apoptosis of platelet and cause mitochondrial damage (9).

Furthermore, our patient developed acute renal failure. It has been proven that the most commonly described mechanism of MTX nephrotoxicity is the crystallization of MTX in the renal tubular lumen (10). This acute kidney injury can be seen in 2% to 12% of patients (10). Renal failure may be also precipitated by concomitant ingestion of some drugs, which are protein bound like sulfonamides, nonsteroidal anti-inflammatory drugs (NSAIDs), and barbiturates. (4)

Skin lesions are reported to be more common in patients with psoriasis. As hyperproliferative psoriatic plaques absorb more methotrexate than normal skin, skin ulcerations caused by MTX toxicity are limited to the psoriatic plaques (11). In our case, we describe oral and vaginal ulceration in a previous normal skin. According to the literature, skin lesions due to acute MTX toxicity are still infrequent and can include ulcers, Stevens-Johnson syndrome, and toxic epidermal necrolysis (12)

In addition to the abovementioned signs, our patient has developed conjunctivitis, which is uncommon. Cases using therapeutic doses usually described symptoms of ocular burning and pruritus (13).

Pulmonary toxicity due to MTX has also been proven. It can be fibrosis, interstitial pneumonitis, or diffuse alveolar damage (14). In our case, the patient has developed respiratory distress.

Regarding the management of MTX intoxication, it is based mainly on hyperhydration, which was used in our case. Folinic acid (also known as leucovorin) is used as an antidote in case of overdoses(15). It has been proven that it competes with MTX to enter cells and allows the replacement of intracellular folate (16). Even though we used it, the outcome was fatal, which can be explained by the delayed consultation.

CONCLUSION

This case report highlights the importance of early follow-up and regular laboratory investigations of patients on MTX therapy. Thus, face-to-face advice and supplemental written information should be provided, especially to elderly patients. Avoiding self-administration of MTX is essential, as is never combining it with another medication without a doctor's approval.

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Perthes syndrome: a case report

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Abstract

The association of cervicofacial petechiae and subconjunctival hemorrhages with neurological manifestations defines Perthes syndrome or traumatic asphyxia syndrome. This syndrome appears after sudden and brief posttraumatic thoracic or thoracoabdominal compression on a hyperinflated chest like a Valsalva maneuver. In general, the prognosis is favorable. The accompanying lesions and the length of the chest compression are predictors of the outcome.

INTRODUCTION

Perthes syndrome, also known as post-traumatic superior cava syndrome, is an uncommon condition. It results from a violent and brief thoracic or thoracoabdominal compression on a hyperinflated thorax. This syndrome associates cervicofacial petechiae and subconjunctival hemorrhage with neurological manifestations. The evolution is favorable if the compression is relieved rapidly and cardiopulmonary resuscitation is initiated early, otherwise anoxic encephalopathy with serious sequelae may develop. We report a pediatric case of Perthes syndrome with a favorable evolution.

CASE REPORT

A 3-year-old child, with no particular medical or surgical history, was admitted to the emergency room following a domestic accident: the father, while reversing the car, hit his son who was trapped under the vehicle with thoracic compression. The family quickly discharged the child and brought him to the emergency department two hours after the accident.

On admission, the boy was drowsy; the Glasgow Coma Scale was at 14/15. He was polypneic at 38 cycles/minute with peri buccal cyanosis. The pulmonary auscultation was symmetric, and the oxygen saturation was at 93% on room air and 98% with 3 l/min of oxygen. His blood pressure was 90/50 mmHg, and his heart rate was 130 bpm. He had thoracic and epigastric abrasions. The abdominal examination was normal. We noted diffuse petechial lesions on the face and the chest and bilateral subconjunctival hemorrhage. (Figures 1 and 2)

The blood count, as well as biochemical analyses, were normal. Three hours after the accident, a body scan was performed and only identified pulmonary contusion. The eye fundus was normal. Neurological symptoms disappeared within the first day of admission. The evolution was favorable within 48 hours; the boy was discharged. Cutaneous petechial lesions and the subconjunctival hemorrhage disappeared progressively.

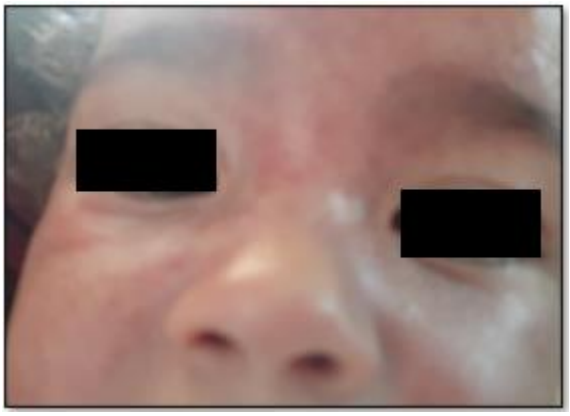

Figure 1: *diffuse petechial lesions on the face*

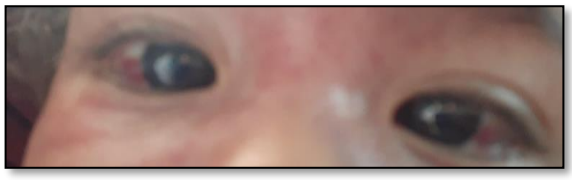

Figure 2: *bilateral subconjunctival hemorrhage*

DISCUSSION

Olliver first described Perthes syndrome, also known as post-traumatic superior cava syndrome, in 1837. It associates petechiae, subconjunctival hemorrhage, and neurological manifestations [1]. The incidence of Perthes syndrome is low. Some authors report an incidence of one case per 18,500 accidents [2, 3]. It is mainly reported in road accidents when the victim is trapped between the seat and the dashboard (40%). The other causes of Perthes syndrome include work accidents, sports accidents, crushing during jostling, and child victims of sexual abuse. Whatever the traumatic cause is, the common mechanism is thoracic compression or crushing [3].

In 1900, Perthes elucidated the mechanism of the post-traumatic superior cava syndrome [4]. The victim blocks the thorax in forced inspiration with a closed glottis at the onset of thoracic impact because of a panic reaction [5]. Traumatic compression on a thorax which is already hyper-inflated increases the pressure in the superior vena cava. It results in venous stasis and capillary and venous ruptures in the overlying territories. The fall of the cerebral blood flow and asphyxia secondary to the traumatic thoracic compression induces cerebral anoxia [3]. The absence of venous stasis in the underlying territories can be explained by the collapse of the inferior vena cava within the Valsalva maneuver, protecting the underlying venous territory [6, 7].

The clinical feature of these phenomena is the manifestation of cyanosis, petechiae, and subconjunctival hemorrhage, with respiratory, neurological, and visual manifestations. The duration and intensity of compression influence the occurrence of lesions [8]. These lesions can be associated with other direct lesions related to the trauma itself.

Cutaneous-mucosal lesions, namely cervicofacial cyanosis, cervicofacial petechiae, and bilateral subconjunctival hemorrhage are reported in more than 92% of cases [3]. Our patient presented all these signs.

Ophthalmologic manifestations are always present. The patient can describe a decrease in visual acuity, diplopia, or scotoma. The examination may reveal anisocoria, traumatic mydriasis, and a decrease in the photo motor reflex. The fundus examination is normal in half of the cases. It may show a retinal hemorrhage, exudates, hyperemia, or retinal edema. These disorders are commonly transient, and they regress completely, but slowly [3]. However, some cases of late blindness due to central retinal

vein thrombosis and optic atrophy have been reported [9]. In our case, the fundus examination was normal.

Neurological damage is the most serious and frequently occurs (90%) in Perthes syndrome. The patient may present consciousness disorders, from agitation to severe coma. These neurological symptoms typically disappear within one to two days [3, 10]. In our case, they disappeared within the first day of admission.

In addition to these specific lesions, other thoracoabdominal lesions, which are non-specific, can be present. They reflect the violence of the trauma and may include pulmonary contusion, rib fractures, hemopneumothorax, joint damage, hemoptysis, or diaphragmatic rupture [11, 12]. Cardiac damage is exceptional. Even when there are intrathoracic lesions present, costal bone lesions can be prevented in children thanks to their high thoracic elasticity [7]. Our patient presented only pulmonary contusion.

Hemodynamic instability can be attached to myocardial contusion, or hemorrhage secondary to a lesion of the large vessels [13]. Abdominal injuries may include hemoperitoneum, digestive hemorrhage, laceration, or perforation of abdominal organs [14]. Genito-urinary lesions, tympanic perforation, hematuria, and medullary injuries were also described [15].

The management of Perthes syndrome should be rapid and start at the site of the accident. Prehospital measures aim to release the thoracic compression and start cardiopulmonary resuscitation early to reduce the risk of anoxic brain injury [3, 11].

Perthes syndrome has a generally positive prognosis, with a 90% chance of survival [3]. The occurrence of severe accompanying lesions and prolonged chest compression for more than ten minutes are predictors of a bad prognosis [14].

CONCLUSION

Perthes syndrome, or post-traumatic superior cava syndrome, is rare. The physician should suspect it when there is an association between an ecchymotic mask and neurological manifestations, occurring after a sudden and brief compression trauma of the thorax. The treatment of Perthes syndrome consists of the rapid lifting of the compression and early cardiopulmonary resuscitation to reduce the risk of cerebral anoxia.

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An abrupt cardiogenic shock in a female at the era of the COVID 19 pandemic lockdown

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Abstract

We reported the challenging diagnosis of Takotsubo syndrome TTS in a female, admitted to the intensive care unit, with cardiogenic shock, and we highlighted the impact of Levosimendan use in the salvage of this patient.

INTRODUCTION

Takotsubo cardiomyopathy or Takotsubo syndrome (TTS), also known as stress cardiomyopathy, is a type of non-ischemic cardiomyopathy in which there is a sudden temporary weakening of the muscular portion of the heart (1). It usually appears after significant physical or emotional stressors (2). We reported the case of TTS complicated by Cardiogenic shock during the COVID-19 pandemic lockdown

CASE REPORT

We report the case of a 73-year-old woman with a history of Hypertension treated by lercanidipine/ enalapril 20/20, Paroxysmal atrial fibrillation under bisoprolol 10mg+ flecainide (lp) 100mg/d, and acenocoumarin and Hypothyroidism under Levothyroxine 50ug/d, presented to the emergency unit because of sudden onset of progressive dyspnea without chest pain. On clinical examination, she didn't present any shock signs nor fever. The heart auscultation revealed regular rhythm without murmurs. The pulmonary exam under oxygen therapy with facemask showed tachypnea, Oxygen saturation at 78%, and diffuse crepitations.

On the electrocardiogram, the rhythm was sinus, with a significant ST-elevation in D1 AVL leads, a poor R-wave progression and a normal QT interval 440ms. Rapidly the patient developed a cardiogenic shock.

A Mechanical ventilation and inotropes were initiated, and the patient was transferred to the Cath lab. The coronary angiography revealed a 40% atheromatic

plaque in the distal left anterior descending artery. The laboratory tests showed a slight elevation of Troponin US (first point=0.065 ng/ml, second point 6 hours later was 0.089 ng/ml), an acute kidney injury with a creatinine of 176 µmol/l and a normal CRP.

The Chest X-ray showed a diffuse pulmonary edema.

The transthoracic echocardiography (TTE) showed signs of hypertensive cardiopathy, with low left ventricular ejection fraction (LVEF) at 15% and akinesia of mid-apical myocardial segments associated with hyperkinetic basal segments (apical ballooning) (figure 1). There wasn't a left ventricular outflow tract obstruction (LVOTO) or a systolic anterior motion of the mitral leaflet. There was no pericardial effusion. The left ventricular twisting on 2D speckle-tracking imaging was reduced with diminished left ventricular longitudinal strain (GLS -10%) (Figure 2 A).

Twelve hours later, there was a T-wave inversion in the anterior leads with a prolonged QTc interval (501ms) (Figure 3). Given these electrical modifications, the echocardiographic findings, the low and stable troponin elevation contrasting with severe LV injuries, the Takutsubo syndrome was the most likely diagnosis (The Inter TAK score was calculated at 75 points >70 points).

ECMO wasn't available in our hospital. The decision was to switch to the Ca2+-sensitizer levosimendan as an alternative to the catecholamines. Few hours later, we obtained hemodynamic stabilization. And after 4 days, the LVEF has remarkably improved (LVEF 50%) (figure 2B)

At 1-month follow-up, the patient demonstrated remarkable recovery with New York Heart Association (NYHA) Class I symptoms. She continued with outpatient psychologic rehabilitation. At that time, TTE demonstrated normalization of cardiac function with LVEF of 60% and complete recovery of the wall motion abnormality.

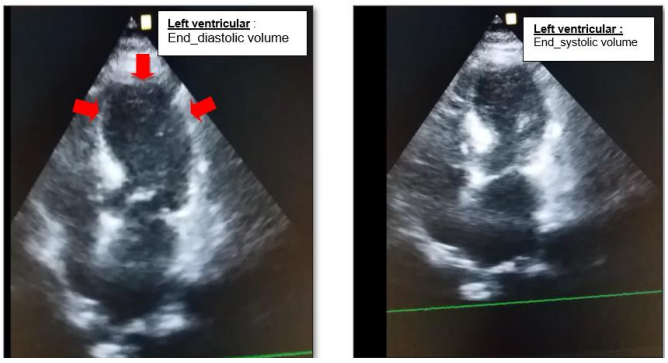

Figure 1: *The echocardiography*

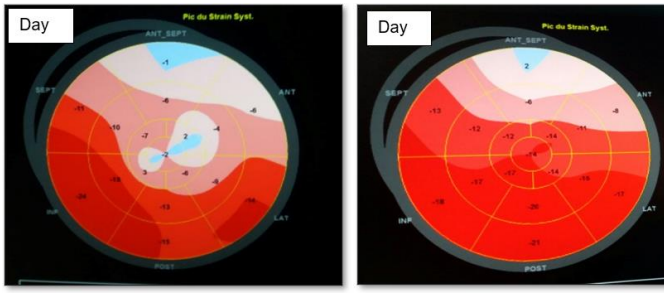

Figure 2: *Left ventricular twisting on 2D speckle-tracking imaging (Day 3-Day 7)*

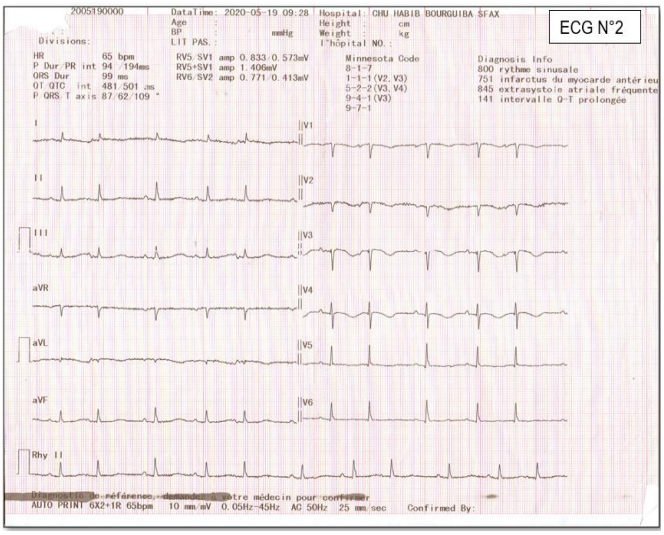

Figure3: *The electrocardiogram*

DISCUSSION

Many cardiovascular consequences of COVID-19 have been described in literature, including myocardial injury, myocarditis, acute coronary syndromes, pulmonary embolism, stroke, arrhythmias as well as heart failure. (1)

This case report illustrated an unusual cause of cardiogenic shock during the first wave of COVID-19, due to the psychological impact of the lockdown.

Takotsubo syndrome is a working diagnosis, and we need many arguments to establish the diagnosis (2).

Initially, we found ST elevation, so it was necessary to practice an emergent coronary angiography to exclude acute myocardial infarction (AMI), and this exploration didn't reveal significant coronary artery stenosis. Few hours later, we noticed progressive T-wave inversion and QT interval prolongation reaching 501ms, which was very suggestive of TTS. The transthoracic echocardiography was very contributive to retain the diagnosis, it showed an Apical ballooning with akinesia of mid-apical myocardial segments associated with hyperkinetic basal segments which extend beyond the distribution of a single coronary artery territory.

Given the findings of coronary angiogram, electrocardiogram, and echocardiography, and in the absence of troponin elevation, TTS was the most likely diagnosis and confirmed after follow-up echocardiography.

In this case, there weren't <red flags> of acute infectious myocarditis (Signs and/or symptoms of viral infections - Elevated

CRP and Pericardial effusion) that's way, cardiac magnetic resonance (CMR) wasn't performed (3). The second challenge in this case report was the treatment.

How did we manage this severe cardiogenic shock?

With Catecholamine (dobutamine and noradrenaline) infusion, the cornerstone in pharmacologic treatment for cardiogenic shock, we didn't notice any amelioration for our patient who presented pulmonary oedema and a refractory shock. Thus, according to the last International Expert Consensus Document on Takotsubo Syndrome, catecholamine should be avoided in TTC, levosimendan is recommended as a safe and effective non-catecholamine inotrope in managing TTS complicated with heart failure.

In our case, levosimendan infusion was well tolerated; and we noted a good clinical improvement.

Levosimendan is a molecule with both inotropic and vasodilator action, with low rates of adverse events. The main mechanism of action is the increase in the troponin C affinity for Ca2+ and the stabilization of troponin C conformation.

The main mechanism of increasing myocardial contractility is based on the increased sensitivity of cardiac troponin C towards intra-cytoplasmic calcium.

The vasodilatory properties lead to a dramatic increase in cardiac output with a concomitant reduction in cardiac filling pressures in the failing heart enabling it to generate more efficient systolic and diastolic functions.

CONCLUSION

Cardiac involvement while the Covid-19 pandemic may manifest atypically. We report the diagnostic challenges with severe cardiogenic shock secondary to the psychological impact of the lockdown on our patient.

This case report highlights the beneficial role of levosimendan in managing TTS with cardiogenic shock as an alternative to mechanical support.

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