

# TRANSIENT LOSS OF CONSCIOUSNESS {TLOC}

## BLACKOUTS TO THE POINT

كلنا بنشوف حالات الغيبوبه بشكل يومي وإلى حد ما عارفين اسبابها الشهيره زي نزيف المخ والأسباب الأخرى المتنوعه ... لكن لما مريض يغمى عليه بشكل مؤقت لفترة زمنيّه محدوده ثم يستعيد وعيه مره اخرى .. ده بنسميه ايه ؟ ده عَرَض شهير جدا ده بنسميه فى الطب 📌

TRANSIENT LOSS OF CONSCIOUSNESS {TLOC} or BLACKOUTS (fainting attacks)

والمريض بيكون 📌📌📌

The patient is initially unconscious then he recovers after a while

يا ترى ايه الأسباب ؟ ▶▶

✓SYNCOPE

✓SEIZURES

✓HYPOGLYCEMIA

✓PSYCHOGENIC PSEUDOSYNCOPE (CONVERSION DISORDER)

✓TIAs (rare cause of TLOC)

🔥N.B there are other causes 📌

- **Traumatic brain injury** ده يخص الجراحات

- **Metabolic disorders** e.g. Hypoxia and Hyperventilation with Hypocapnia.

- **Intoxications**

## ما معنى كل سبب من دول ؟

▶ SYNCOPE = Transient loss of consciousness due to insufficient blood supply to the brain (Global cerebral hypo perfusion)

▶ SEIZURES = Sudden, uncontrolled body movements and changes in behavior/conscious Level that occur because of abnormal electrical activity in the brain. Symptoms include loss of awareness, loss of muscle control, and shaking  
التشنجات

▶ Hypoglycemia

When Blood glucose is low ↓ and often < 50 mg/dl, the patient will develop the so called NEUROGLYCOPENIC SYMPTOMS, and he will develop ↓ conscious level until BG is corrected by IV Glucose, the conscious level improves

انخفاض السكر في الدم . ده اهم للـ TLOC metabolic cause

▶ PSYCHOGENIC PSEUDOSYNCOPE (CONVERSION DISORDER)

-- » Unexplained TLOC without evidence of any organic cause and after exclusion of other causes ... السبب النفسى مهم وبتشوفه ...

▶ TIAs:

TIAs commonly lead to transient Focal neurological deficit (often motor like hemiparesis, Sensory (hemihypothesis) and visual (Amaurosis Fugax), but it rarely leads to TLOC

وده لو حصل هيجصل مع الـ Vertebrobasilar insufficiency و هو عبارة عن قصور مؤقت فى الدوره الدمويه الخلفيه داخل المخ

## ✍ Syncope ✍

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🔵 Syncope: transient loss of consciousness due to global cerebral hypo perfusion.

N.B.

The patient might develop Pre-syncope symptoms before the onset of SYNCOPE!!

### ✍ Pre-syncope 🔄

dizziness, blurring of vision, sense of impending fainting plus other associated features related to each cause of SYNCOPE

وده أحد أسباب الدوخه وده احنا شرحناه قبل كده فى بوست الـ dizziness (راجع البوست)

## Syncope is divided into 3 types:

*A: cardiac syncope*

*B: Reflex syncope*

*C: Orthostatic syncope*

### *A: cardiac syncope*

Cardiac syncope is a brief loss of consciousness (from a few seconds to a few minutes), that is characterized by rapid onset and spontaneous recovery. It is caused by decreased blood flow to the brain.

#### ● Criteria:

blurred vision, diaphoresis, nausea, dizziness, Patients appear pale at the start then lose consciousness, Syncope is not related to certain posture like standing

ايه أسبابه؟ 🌴🌴

**Structural heart disease \*\* Brady arrhythmias \*\* Tachyarrhythmias**

## I) Structural heart disease

Particularly causing Left ventricular outflow tract obstruction (LVOTO)

like 🚩🚩🚩🚩🚩

▶ HOCM, aortic stenosis

N.B: in HOCM, syncope might occur after exercise مهم

كمان ال HOCM غالبا فيه positive family history وبيجي فى الصغيرين وبيعمل voltage criteria

Dx: Initial test: ECHO و للتشخيص فى ال ECG

## II) Brady arrhythmias:

Second degree third degree (complete heart block and sick sinus syndrome).

--> Adam stokes episodes

### 🇸🇦 DESCRIPTION OF STOCKES ADAMS ATTACKS 🇸🇦

👉 It occurs abruptly due to ↓ of cardiac output

👉 The patient initially becomes pale with extreme Bradycardia for few seconds then his face became Flushed after regaining of normal sinus rhythm

Dx: ECG & holter monitoring اهم شئ

## III) Tachyarrhythmia:

Ventricular Tachycardia whether occurring due to 🇸🇦

structural heart disease like CAD etc.

Or

With underlying inherited diseases

like 🇸🇦 channelopathies (congenital or Acquired Long QT syndrome )

Brugada syndrome, arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC)

Next steps:

ECG, holter monitoring, ECHO

EPS (Electrophysiologic study)

## ملحوظات هالاهامه

**100 Cardiac tamponade** might be a cause of acute syncope (clue is the Beck's triad: Muffled HS + hypotension + ↑ JVP); ECHO is diagnostic

**100 Pulmonary Embolism** also might be a cause of acute syncope (clues; other suggesting features of PE like, acute dyspnea, chest pain, ↓ O2 sat, in risky patients (CTPA is diagnostic)

**100 MI** may be a cause of acute Syncope (clue preceding ischemic chest pain in a risky patient; ECG and Troponin in diagnostic)

**100 Aortic dissection** might lead to Acute syncope in some cases (clues; associated chest pain of tearing type; INEQUAL BP: ↑ ↑ BP)

## **B: Reflex syncope (Neurally mediated Syncope)**

### **I) Vasovagal syncope:**

usually occurs in young persons with extreme stress, fear and prolonged standing

العيان هيكون غالبا صغير و هيكون فى وضع الوقوف ولما يتعرض لموقف من المواقف المذكوره دى ..

collapse to the ground 📉📉📉 ثم يحصله sweating , pallor يحصله

و غالبا هي فوق بمجرد رفع الرجلين للأعلى وهو 📉📉 ... in recumbent position (supine)

### **II) Situational syncope**

- cough induced syncope (in some persons) اغماء مؤقت بعد كحه

- micturition induced syncope.

اغماء مؤقت بعد التبول

### **III) Carotid hypersensitivity**

Occurs more in old age. With hypersensitivity of carotids which is triggered by shaving, tight collar, excess manipulation of neck.

or

with certain movement of the neck (turning of the neck or looking upward)

Like in Carotid Sinus syndrome!!

Carotid hypersensitivity has two types

✓ Cardio inhibitory: leading to bradycardia

✓ Vasodilatory: Leading to hypotension.

Next step

📄 Tilt table test for confirmation of the diagnosis

## C: Orthostatic syncope

● Syncope as a result of orthostatic hypotension

Causes 📌

**A) autonomic dysfunction** due to systemic diseases like DM, Amyloidosis, multiple system atrophy (shy dragger syndrome).

primary autonomic failure

Adrenal insufficiency

مشهور جداا فى مرضى السكر الغير منضبط بعد سنوات كثيره .. المريض هيجيله دوخه بمجرد وقوفه سريعا  
من وضع الاستلقاء أو الجلوس .. ويمكن يدخل فى syncope كامل

📌 Measure BP in sitting and standing position (SBP will ↓ by  $\geq 20$  and/or  $\geq 10$  on standing) مهم جداا

**B) Hypovolemia:** مهم جداا

As in any hemorrhage, dehydration

\* drugs like ACEI, Diuretics

📌 Clues: Measure BP in standing and sitting position and ask about bleeding

Examine Signs of hypovolemia and dehydration

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## Clues for SEIZURES

The seizure is often of Grandmal type 🔥

Preceding aura

The person may experience abnormal sensations such as a particular smell, vertigo, nausea, or anxiety

دايخ أو شامم ريحه معينه وكمان بيبقى عارف ان نوبة الصرع هتحصل

Initial TONIC PHASE Tonic contraction of all muscles of the body with cyanosis in some patients due to stoppage of respiratory muscles

Then

CLONIC PHASE, violent rhythmic contraction of all body muscles along with TONGUE PITTING (blood coming from the mouth) and/or urine incontinence

بعد حدوث حركات لا اراديه عنيفه فى كل عضلات الجسم المريض ببعض لسانه وممكن ينزل دم من بقه ،، وفى نفس الوقت بيعمل حمام بول على نفسه .....

Then Post-ictal phase: (confusion with ↓ conscious level for a while)

## Clues for hypoglycemia

Initial dizziness, palpitation, sweating (adrenergic symptoms) then ↓ in conscious level if B G is not corrected rapidly

the patient might be diabetic with history of insulin or SU overdose or missing a meal

Hypoglycemia might occur without diabetes (fasting vs post-prandial Hypoglycemia)

Checking of RBG is diagnostic and is a must

## Clues for Psychogenic PSEUDOSYNCOPE

- History of stress
- PH of similar episodes (more common in women; young women)
- Pseudo seizures (side to side shaking or tremors, sounds during episodes)

اصوات او كلام غير مفهوم وحركات غريبه تشبه التشنجات ولكنها مختلفه

## Clinical approach to TLOC

### Take history asking about the following

- ✓ onset of symptoms
- ✓ preceding symptoms
- ✓ description of attacks (what occurs before, during & what occurs after?!
- ✓ You must ask about seizures by their description
- ✓ You must check Blood glucose
- ✓ You should take a collateral history from a reliable witness that was with the patient during the onset of symptoms
- ✓ Asking about color of the face during the onset of symptoms and the posture of the patient while he develops the attack is mandatory.
- ✓ asking about Past History of certain disease of note, is so important like DM, cardiac problems, epilepsy or neurological problems and past history of seizures.

### Clinical pearls that aid in the Dx!

#### The following questions should be answered:

- 🌿 Was loss of consciousness complete?
- 🌿 Was loss of consciousness with rapid onset and short duration?
- 🌿 Was recovery spontaneous, complete, and without complications?
- 🌿 Was postural tone lost?

If the answers to these questions are **positive**, the episode has a high likelihood of being syncope.

While, if one or more answers are **negative**, consider other forms of loss of consciousness prior to proceeding with syncope evaluation.

The clinician should attempt to gather all information with respect to symptoms preceding the syncope.

Symptoms of nausea or diaphoresis prior to the event may suggest syncope rather than seizure when the episode was not witnessed, whereas an aura may suggest seizure

Patients with true syncope do not remember actually falling to the ground.

**👉 Clinicians should specifically inquire as to red-flag symptoms,**

**such as: -**

- ✓ exertional onset
- ✓ chest pain
- ✓ dyspnea
- ✓ low back pain
- ✓ palpitations
- ✓ severe headache
- ✓ focal neurologic deficits
- ✓ diplopia
- ✓ ataxia
- ✓ dysarthria prior to the syncopal event.

**👉 Focused exam:**

- ✓ vital signs
- ✓ test for orthostatic Hypotension after the patient recovers
- ✓ RBG
- ✓ rapid Neuro - exam while in TLOC
- ✓ Thorough cardiac examination

## Dx workup

It will depend on the data that you have gained or gathered from history and exam.

Initial investigations: ✓ECG    ✓ECHO    ✓EEG    ✓Holter monitoring

✓ CT brain (routine) just for exclusion but not for Dx

Then other labs according to certain clues mentioned above.

### Clinical features that suggest a diagnosis on initial evaluation

<b>Neurally mediated syncope:</b>
Absence of heart disease
Long history of recurrent syncope
After sudden unexpected unpleasant sight, sound, smell or pain
Prolonged standing or crowded, hot places
Nausea, vomiting associated with syncope
During a meal or post-prandial
With head rotation or pressure on carotid sinus (as in tumours, shaving, tight collars)
After exertion
<b>Syncope due to OH:</b>
After standing up
Temporal relationship with start or changes of dosage of vasodepressive drugs leading to hypotension
Prolonged standing especially in crowded, hot places
Presence of autonomic neuropathy or Parkinsonism
Standing after exertion
<b>Cardiovascular syncope:</b>
Presence of definite structural heart disease
Family history of unexplained sudden death or channelopathy
During exertion, or supine
Abnormal ECG
Sudden onset palpitation immediately followed by syncope
ECG findings suggesting arrhythmic syncope:
- Bifascicular block (defined as either LBBB or RBBB combined with left anterior or left posterior fascicular block)
- Other intraventricular conduction abnormalities (QRS duration $\geq 0.12$ s)
- Mobitz I second degree AV block
- Asymptomatic inappropriate sinus bradycardia (<50 bpm), sinoatrial block or sinus pause $\geq 3$ s in the absence of negatively chronotropic medications
- Non-sustained VT
- Pre-excited QRS complexes
- Long or short QT intervals
- Early repolarization
- RBBB pattern with ST-elevation in leads V1-V3 (Brugada syndrome)
- Negative T waves in right precordial leads, epsilon waves and ventricular late potentials suggestive of ARVC
- Q waves suggesting myocardial infarction

ARVC: arrhythmogenic right ventricular cardiomyopathy; AV: atrioventricular; LBBB: left bundle branch block; OH: orthostatic hypotension; RBBB: right bundle branch block; VT: ventricular tachycardia.

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