NAAMA Clinical Cardiac Electrophysiology Specialty Symposium

- NAAMA 24th International Medical Convention
- Inaugural Meeting ARAB HEART RHYTHM ASSOCIATION
- SATRURDAY JUNE 26TH, 2010
- BEIRUT

SYNCOPE ETIOLOGY &MANAGEMENT

A REAL WORLD APPROACH

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Definition

- Syncope is defined as transient reversible loss of consciousness typically associated with loss of motor tone .This can be associated with falls and injury .
- The diagnosis of syncope is typically one of the top 10 admitting or ER diagnoses in most countries

Differential Diagnosis

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- Transient Loss of consciousness is also associated with Neurologic syndromes such Seizures and various forms of epilepsy

Differential Diagnosis

- Transient Loss of consciousness is also associated with Neurologic syndromes such as Seizures and various forms of epilepsy ,
- TIA / RIND syndromes ,Vertebrobasilar insufficiency and subclavian steal syndrome ,
- hypoglycemia (+or – associated Whipple's triad) , pheochromocytoma ,
- encephalopathy (various etiologies – Infectious , metabolic, toxic , medication related) .
- Severe sleep apnea /Pickwickian Syndrome can mimic syncope especially while driving .
- Non syncope i.e. severe vertigo due to vestibular neuronitis or 8th nerve compression

Differential Diagnosis

- The major differentiating feature is the presence of associated neurologic symptoms preceding and delay/ prolongation in recovery of consciousness , abnormal motor activity , post ictal states with altered sensorium after the event .

Types of Syncope

- Three types of syncope are well described
- Syncope with structural heart disease
- Syncope without SHD (expected to include benign causes of syncope)
- Syncope with arrhythmias(extrapolated to include channelopathy)

Syncope with SHD and Channelopathy / Arrhythmia

- 1-Aortic Stenosis
- 2-Cardiomyopathy ischemic and dilated
- 3-IHSS/HOCM
- 4-ARVD
- 5-Acute coronary syndrome (IMI and CHB)
- 6-Bradyarrhythmia – Sinus node dysfunction , pauses , heart block different levels
- 7-Tachyarrythmias – VT , SVT
- 8-LQTS , Brugada Syndrome , PMVT , Short QT
- 9-Preexcitation Syndromes WPW +/- atrial fibrillation

Syncope in special circumstances

- Hypoxic syncope – massive PE with RV strain or Tension Pneumothorax (associated rarely with post tussive syncope in COPD emphysema)
- Aortic Dissection
- Carotid or Vertebral artery dissection
- Pericardial Tamponade

Syncope without SHD

- 1-Neurocardiogenic Syncope-
  - Vasodepressor Syncope
  - Cardio inhibitory Syncope
- Mixed variants
- Not associated with cardiac arrest or SCD

Syncope in special circumstances (reproducible clinically)

- 1-Post Tussive syncope
- 2-Post Micturition syncope
- 3-Post deglutition syncope / cold water syncope
- 4-Post defecation syncope

Evaluation

- The single most useful “test” in the evaluation is the history .
- Associated signs and symptoms can point the way and avoid a “million dollar workup” . However if the pattern does not fit classical Neurocardiogenic syncope then such a workup is indeed necessary .
- Ultimately the goal is to risk stratify your patient – high likelihood of SCD/ malignant arrhythmia requiring device therapy or NOT

Clues about “benign” NCS

- Recurrence especially at an early age (however does not rule out channelopathy or HOCM)
- Prodromal symptoms preceding weakness , warmth , flushed sensation , nausea , vomiting , positional dependence , pallor or ashen gray color ,diaphoresis (doesn’t exclude endocrine or coronary syndromes) , other hypervagotonic features .

Clues to Benign NCS

- Sensation of blackout and blood “rushing out of the head” .Rapid Activation of protective reflexes so patient falls but typically without injury .
- Immediate recovery of consciousness upon supine position with passive restoration of cerebral blood flow Patient is able to converse and move with good recollection of events , usually denies total loss of consciousness and is fully aware of surroundings .Pale coloration usually recovers rapidly , diaphoresis resolves rapidly .

Clues about Benign NCS

- Immediate recurrence for up to 30 minutes if the patient tries to stand up or is placed in sitting position
- Orthostatic Hypotension with **2 minutes of standing at bedside** needs to be ruled out as this is immediately correctable and does not need workup except to evaluate serum chemistry and anemia
- Normal EKG without QT changes , no axis shift or hemi block or bundle branch block .
- Normal physical exam – including absence or murmur , bruit and equal BP in **both** arms and equal pulses in both upper and lower extremities – BEWARE of Marfan syndrome painless Aortic dissection presenting as syncope

Ominous /Malignant Syncope

- Clues about NON BENIGN Syncope (i.e. SHD or Arrhythmia)
- Age above 65 is usually associated with SHD or arrhythmia
- 1-Presence of risk factor , co morbidity i.e. smoker , hypertension , DM , hyperlipidemia or prior actual history of Coronary , Cerebrovascular or Peripheral Artery Disease
- 2-Preceding Palpitations and preexisting arrhythmias or known preexcitation
- 3-Chest pain of any type or duration , Hypoxia or visible cyanosis

Ominous /Malignant Syncope

- Any EKG abnormality – blocks , pr prolongation , QTC> 450ms with abnormal T waves – broad or notched , or less than 300ms LQTS and SQTS, Brugada Type EKGs , low voltage (effusion) , Delta wave , epsilon wave , RVH , ischemic ST changes
- Frequent ventricular ectopy, monomoprhic or polymorphic especially runs of NSVT
- Syncope while sitting or while in bed

Ominous Syncope-PHYSICAL FINDINGS

- Abnormal physical findings murmur , bruit , pericardial rub , pulsus paradoxus, pneumothorax, unequal pulse and BP in extremities , rash – lyme disease , irregular pulse a fib , pauses , intermittent block or frequent ectopy , neurologic deficits , deafness JLN and Romano Ward chanelopathies , cannon A waves in CHB
- Persistent hypotension or tachycardia especially in a hypertensive patient .
- Any neurologic findings – tremors , Parkinson –Shydrager syndrome, Hemi paresis , cerbellar or sensory signs, CVA
- Anisocoria with cranial nerve involvement –sympathetic ganglion involvement by tumor of skull base or lung

OMINOUS PRESENTATION of Syncope

- Syncope during exertion is a poor prognostic sign . Athletes don't just FAINT ! Suggestive of VF OR HOCM
- SYNCOPE during swimming – LQTS
- Syncope/ sudden death triggered by loud noise or auditory stimuli especially at night LQTS
- Syncope after PPM – VT or VF , pacemaker syndrome , lead dislodgement , PMT, tamponade

Syncope related to Medication

- Brady arrhythmia – Digoxin , Calcium Channel Blockers , Betablockers , dose related
- Hypotension - #1 DIURETICS in older patients also vasodilators and ACE /ARB , nitrates
- SYNCOPE after nitrates- severe AS or even severe left main or 3vdCAD – hypotension resulting in decreased coronary perfusion and ischemia with paradoxical bradycardia with Avblock- RARE but very significant

Syncope related to Medication

- Syncope on antiarrhythmics – very suspicious of proarrhythmia esp. Torsades.
- Enhanced bradycardia effects of flecainide with low sodium, dehydration
- Dronedarone can cause significant AV and SA node inhibition
- Antibiotics – Quinolones and Macrolides can unmask LQTS esp. at high doses –Syncope may be due to Torsades rather than infection

Management

- ECHO important to completely exclude SHD
- EKG careful evaluation of 12 lead ekg preferably with older ekgs for comparison- QT can vary as does BrS
- Holter / Event monitoring/Loop recorder
- Tilt table testing with and without nitroglycerin, tensilon, isuprel only 60-70% diagnostic
- Beta Blocker , Florinef , SSRI, Midodrine
- Postural training
- Vestibular training maneuvers

Management

- Low ejection fraction- SCD heft / MADIT II type patient
- Channelopathy – further risk stratification is needed family history , genetic testing , AICD
- Avoidance of triggers esp. LQTS patients
- Bradyarrythmia – PPM with device and patient specific features
- Medication adjustment