A first seizure

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EPILEPSY

-Treatment gap: up to 90%
-Loss to follow up about 50%

All pictures with (parental) consent

Neurology in Africa. Howlett 2012
Walker et al., Lancet Neurology 2011
Complications

Photos Prof Howlett, KCMC
The first seizure (“S1”)

2 questions to answer:

Q1. Seizure? Or no seizure (PNEA)?

Q2. Acute symptomatic seizures or possible epilepsy?
S1 symptoms

Q1. Seizure? Or no seizure (PNEA)?

• Really the first seizure ever? (early childhood, FS)
• Eye witness
• Precipitants (lack of sleep, stress, fever, light, substance abuse etc.)
• Chronology (frequency, time of day/night, stereotypical)
• Associated (mental/somatic) symptoms

Smartphone video? Combined with exam and history: 95% sensitive.

“Am I seeing what I am seeing?”

“Am I seeing what I am seeing?”

Top 3 Most Helpful

Seizure:
Abrupt onset,
eyes open/pupils dilated,
postictal confusion/sedation.

Psychogenic Non Epileptic Attack (PMEA):
Preserved consciousness (no amnesia),
eyelid flutter,
influencable from the outside.

Syed et al, Ann Neurol 2011
S1 can present as status epilepticus

Often (plm 50%): history of epilepsy, often unknown at time of presentation (when patient brought in)

Think of: sudden withdrawal ASM (phenobarbital).

Or the many other causes
“Atypical symptoms”

Seizure mimic: pale, sweating, nauseous, dizzy: (pre)syncopal (cardiac, vasovagal, hypoglycaemia?)

Or: PNEA?
- Eyes closed, eyelid flutter
- Motionless posture from onset of attack
- Duration (the longer the less typical)
- Never any incontinence/tongue bite/injury
- Onset long enough ‘to seek comfy position'
- Quick postictal recovery (possible in eg FLE)
- Emotional (during and afterwards)
- Thrashing; groaning; weeping; pelvic thrusting
- Never eye witnessed; solely patient's own account
- 'Secondary benefit' (teenagers, boarders, soldiers etc.)
- No response on adequately dosed ASM
- ‘Coincidence’ factor… some of the fits in the waiting room
S1 additional investigations

Vitals, neuro examination
Neuroimaging: abnorm CT 17%, MRI 23%
EEG: 72h capture chance IED 95%
Laboratory tests (CK better than Prolactin)
LP: CSF
Do S1 additional investigations suggest “acute symptomatic seizure”?

By
- CNS infection
- TBI
- hypoxia
- intoxication (medication/substance)
- eclampsia/HTN/PRES
- ICH etc.
Often: post stroke, trauma, HIE, metabolic, HTN, CNS infections


<table>
<thead>
<tr>
<th>Common Etiologies</th>
<th>Adults</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke, including hemorrhagic</td>
<td>20%</td>
<td>10%</td>
</tr>
<tr>
<td>Low antiepileptic drug levels</td>
<td>35%</td>
<td>20%</td>
</tr>
<tr>
<td>Alcohol withdrawal</td>
<td>15%</td>
<td>—</td>
</tr>
<tr>
<td>Drug intoxication (theophylline, imipenem, isoniazid, beta-lactams, clozapine, bupropion, 4-aminopyridine, cocaine, etc) or withdrawal (benzodiazepine, barbiturate, baclofen)</td>
<td>5%</td>
<td>5%</td>
</tr>
<tr>
<td>Anoxic brain injury</td>
<td>15%</td>
<td>5%</td>
</tr>
<tr>
<td>Metabolic disturbances (low glucose, calcium, magnesium, or sodium level; high glucose level; renal failure; liver failure)</td>
<td>15%</td>
<td>5%</td>
</tr>
<tr>
<td>Infection (meningitis, encephalitis, brain abscess, sepsis)</td>
<td>5%</td>
<td>5%</td>
</tr>
<tr>
<td>Traumatic brain injury</td>
<td>2.5%</td>
<td>15%</td>
</tr>
<tr>
<td>Brain neoplasm</td>
<td>5%</td>
<td>0%</td>
</tr>
<tr>
<td>Febrile seizures</td>
<td>—</td>
<td>50%</td>
</tr>
<tr>
<td>Remote brain injury/congenital malformations</td>
<td>20%</td>
<td>40%</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>5%</td>
<td>5%</td>
</tr>
</tbody>
</table>

*Modified with permission from DeLorenzo RJ, et al, Neurology. © 1996, American Academy of neurology. [www.neurology.org/content/46/4/1029.short?sid=e0855659-4863-46e8-b0c8-0d49dfd06f97](www.neurology.org/content/46/4/1029.short?sid=e0855659-4863-46e8-b0c8-0d49dfd06f97).*
S1 management:  
’One seizure is no seizure’?

Usually no to ASM after S1:
- Neurological examination normal
- Neuroimaging, EEG normal
- Specific trigger eliminated
- Seizure during sleep
- No family history of epilepsy

(Q2: Acute symptomatic seizure: depends)
Occasionally **yes** to ASM after S1 if:
- Only 1 seizure, but raised risk
  (eg TC seizure: recurrence risk 16%-61%)
- Seizures with impaired awareness

(Q2: Acute symptomatic seizure: depends)
If ASM after S1:

Then rational choices, eg.:

Uninsured and/or rural setting: phenobarbital, phenytoin, carbamazepine (TZ)

Focal onset by history, nocturnal: carbamazepine (levetiracetam)

Post stroke seizure: phenytoin (see considerations above), valproate

Childbearing age: lamotrigine, levetiracetam.

(often 2nd-3rd trimester presentation: dose matters!)
S1 management:
Life style rules after S1

Poll:

Raise your hand if your country’s road law has legislation regarding seizures and driving
Life style rules after S1

Fire
Water
Traffic -18% car accidents: driver S1!
Dependants: infants/elderly in their care
Heights

But most activities are possible > prevent stigma + attention to mental health.
Easily overlooked/ignored cause of S1: Alcohol

.....ask!

Consider withdrawal (10% of alcoholics: 7-48h post interrupted intake), intoxication or compounded vascular or metabolic -liver- complications.

SESA entity: ‘Subacute encephalopathy with seizures in alcoholics’.

Uncommon in WKS unless there are cortical lesions.

Missed head injury: cSDH series KCMC: 25% alcohol use

Hence:

• High index of suspicion

• Low threshold to supplement thiamine (/glucose infusion)
Thiamine: the refresher

All figures based on normal gastrointestinal uptake

- Daily baseline requirement: **1-3 mg**
- Body stores: **30 mg**
- A healthy individual can deplete their thiamine in **2-4 days**
- Intestinal resorption of oral thiamine only **5%** BUT SAFE
- IV thiamine in at-risk patients: **100mg IV**
- PO thiamine in at-risk patients: **200mg PO BD**
- IV thiamine in WKS: **500mg IV TDS**
- PO thiamine in WKS: up to **1500mg PO QID!**
- Duration of high-dose treatment: **three** days or until plateau in neurological improvement
- Continue with tapering dose to eg. **100mg PO OD**
“Mr. Osborne, may I be excused?
My brain is full.”