EEG WORKSHOP
Nonepileptiform Abnormalities

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EEG reading

Age

Background

Normal

Epileptiform discharges

Non epileptiform discharges

Activation procedure

PDR for age

1 yr 5 Hz
4 yr 6 Hz
5 yr 7 Hz
> 8 yr 8 Hz

Scenario 1

EEG reading

Age

Background

Non epileptiform discharges

Activation procedure

Unequivocally abnormal

Slow activity

• Most common EEG manifestation of focal brain dysfunction
• More reliable when it is:
  - continuous
  - unreactive
  - polymorphic
  - high amplitude
  - unilateral
IRDA: Intermittent Rhythmic Delta Activity

- Varied form
  - Non-specific
  - Structural
  - Infectious
  - Metabolic
  - Epilepsy / Epileptiform Pattern

IRDA

**Adult**
- FIRDA
- TIRDA
- OIRDA (less common)

**Children**
- FIRDA (unclear sig.)
- TIRDA
- OIRDA

OIRDA

- Occurs almost exclusively in children
- Associated with 1st generalized syndrome (childhood absence epilepsy)
- Occasionally seen in encephalopathic children (Salmonella infection, SSPE)

OIRDA

- Typical absence > atypical absence
- OIRDA in absence sz: 3-4 Hz
- OIRDA in localization-related sz: 2-3 Hz
- Most OIRDA < 5 sec
- When: awake and asleep recording
OIRDA

• Occasionally seen in encephalopathic children (Salmonella infection, SSPE)

FIRDA

• 1.5-4 Hz, Mostly in waking adult EEG
  • Previously = deep midline structure, post-tumors, pituitary tumor, subcortical lesion, HC, cerebral edema, IICP
  • Currently = ischemic brain injury, hemispheric brain tumor, with mild to mod metabolic impairment, postictal

OIRDA in atypical CNS Salmonellosis

OIRDA

Absence sz: 3 Hz spike/wave and OIRDA

FIRDA

• FIRDA + normal b/g → structural lesion
• FIRDA + abnormal b/g → encephalopathy

Uncommon epilepsy
FIRDA (+ normal b/g)

Where is the lesion?

FIRDA (+ abn b/g)

FIRDA + abnormal b/g

TIRDA
• TIRDA = Temporal intermittent rhythmic delta activity
• TIPDA = Temporal intermittent polymorphic delta activity
• TIRDA \(\rightarrow\) suggests temporal epileptogenesis

Facts
Temporal lobe epilepsy
• normal awake and asleep EEG
• HV or PS \(\rightarrow\) may or may not induce abnormalities
• sleep deprivation \(\rightarrow\) may induce epileptiform discharges (and also TIRDA)
• TIRDA + epileptiform d/c \(\rightarrow\) temporal lobe epilepsy
Background

Epileptiform discharges

Non-epileptiform discharges

Activation procedure

Age

Scenario 3

Continuous generalized delta slowing

Ex 1

Continuous generalized delta slowing

Ex 2

Bilateral cerebral dysfunction/encephalopathy eg. ADEM, encephalitis, coma, post ictal

Unequivocally abnormal but non specific

Bilateral cerebral dysfunction/encephalopathy
Scenario 4

EEG reading

Age

Background abnormal
Epileptiform discharges
Non epileptiform discharges
Activation procedure

what

Where

Intermittent

eg. Autoimmune encephalitis

NMDA Autoimmune encephalitis

Before Rx

9 days after IVIG treatment

Focal slowing (low voltage + intermittent at baseline) → HV → accentuating the abnormalities

Scenario 5

EEG reading

Age

Background abnormal
Epileptiform discharges
Non epileptiform discharges
Activation procedure

what

Where

Amplitude asymmetry

Intermittent PS → no effect on focal slowing

Amplitude asymmetry

Scenario 5

EEG reading

Age

Background abnormal
Epileptiform discharges
Non epileptiform discharges
Activation procedure

what

Where

Continuous

Amplitude asymmetry

Gen. Focal (F, T, O)

Scenario 5

EEG reading

Age

Background abnormal
Epileptiform discharges
Non epileptiform discharges
Activation procedure

what

Where

Amplitude asymmetry

Intermittent

Gen. Focal (F, T, O)

Scenario 5

EEG reading

Age

Background abnormal
Epileptiform discharges
Non epileptiform discharges
Activation procedure

what

Where

Amplitude asymmetry

Intermittent

Gen. Focal (F, T, O)

Scenario 5

EEG reading

Age

Background abnormal
Epileptiform discharges
Non epileptiform discharges
Activation procedure

what

Where

Amplitude asymmetry

Intermittent

Gen. Focal (F, T, O)
Amplitude asymmetry

1. May occur as normal finding: isolated finding eg. alpha in R-H higher than L-H
2. If amplitude on the Rt is higher than the left for 1½ times = significant asymmetry
3. If amplitude on the Lt is 25% higher than the Rt = significant asymmetry

Which hemisphere is abnormal?

Differences in amplitude

- **Increased** amplitude can be seen in ipsilateral lesions (plus epileptiform discharges)
- **Diminished** amplitude
  1. excess fluid between the cortex and electrodes
  2. abnormalities of cortical gray matter
  3. congenital lesions: SWS, porencephalic cyst
  4. transient b/g attenuation = postictal

**Increased** amplitude + epileptiform d/c

**Increased** amplitude from the skull defect

Excessive fast activity

Breach rhythm
Diminished amplitude

1. excess fluid between the cortex and electrode
2. abnormalities of cortical gray matter
3. congenital lesions: SWS, porencephalic cyst
4. transient b/g attenuation = postictal

2. Abnormalities of cortical gray matter

Rt
Rt

3. Congenital lesion + fluid collection

4. Transient b/g attenuation in postictal

Slow activity and amplitude asymmetry

- abnormalities of gray matter + white matter involvement e.g. ischemic stroke
- diminished amplitude b/g + polymorphic delta activity
Amplitude asymmetry
Continuous + gen (L-H)
Continuous + localized (T)

EEG reading
Scenario 6
Corpus collosum problem

Asynchrony

Background
Epileptiform discharges
Non-epileptiform discharges
Activation procedure

Age
What
Where
When

abnormal
Gen.
Continuous

Corpus collosum problem