Headaches in the Emergency Room

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Disclosures

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• Allergan
• Merz Pharmaceuticals
• Teva
Overview

- Thunderclap headache
  - Helpful clinical signs
  - Predicting probability of SAH
  - beyond SAH

- Treatment in the ER

- Future therapies coming our way
Setting the scene...

68yr ♀, fit & well, smoker

- 5 days prior in Spain:
  - EtOH ++ in last 24hrs
  - Sunbathing at time
  - Headache abrupt & severe, within 10 mins 10/10
    - L occipital → frontal with facial ache
    - Throbbing pain
    - Assoc vomiting, marked photophobia, pre-syncopal

→ Seen A&E: ?migraine and DC with codeine and paracetamol

Presented NNUH on return:

- Unwell with headache
  - On-going & severe at 8/10 → worse in morning
  - Neck pain
  - Vomiting daily; poor oral intake
  - Photophobia
  - Functionally disabled
  - Collapsed with brief LOC

O/E
- Nil focal neuro, fundi N, nuchal rigidity
- Temp 37.8°C; weight reduction of 4kg
- BP 156/105; HR 114; ECG N
68yr ♀, fit & well, smoker

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French guidelines for the emergency management of headache, Moisset et al 2016

- Nil focal neuro, fundi N, nuchal rigidity
- Temp 37.8°C, weight reduction 4kg
- BP 156/105; HR 114; ECG N

Setting the scene…
helpful clinical signs....the eyes have it

A: Ptosis & conjunctival inj
B: Horners
C: 6th Cranial nerve palsy
D: Subhyaloid haemorrhage

1. Sub-arachnoid haemorrhage
2. Cervical arterial dissection
3. Venous sinus thrombosis
4. Cluster headache
5. SOL
6. IIH
helpful clinical signs....the eyes have it

A

Ptosis & conjunctival inj

4. Cluster headache
helpful clinical signs....the eyes have it

2. Cervical arterial dissection
helpful clinical signs....the eyes have it

B

- headache in 60-95% & thunderclap in 1/5
- ipsilateral
- usually headache/ cranio-cervical pain plus [Silbert et al 1995]
  - Horner’s
  - pulsatile tinnitus,
  - amaurosis fugax
  - cerebral ischaemic symptoms
- ipsilateral cranial palsies in 12% [Mokri et al 1996]
  - 5% lower cranial nerve involvement- usually XII
  - 4% palsy of cranial nerve V
  - 3% ocular motor palsies

2. Cervical arterial dissection

Horners

Neema Kasravi et al. CMAJ 2010; 182:E373-E377

Long segment of narrowed lumen

Dissecting hematoma

Hematoma

ICA

CCA

helpful clinical signs....the eyes have it

6th Cranial nerve palsy

Papilloedema

3. Venous sinus thrombosis
6. IIH
5. SOL
helpful clinical signs....the eyes have it

- headache in 90%
  - postural
  - valsalva triggered
  - visual obscuration
- PULSATILE tinnitus
- diplopia (CN6 palsy as false localiser)
- typically progressive but can be thunderclap
- seizures in 40%
- bilateral brain involvement

- risk factors:
  smoker
  COCP & pregnancy/postpartum
  cancer
  dehydration & hyperviscosity
helpful clinical signs....the eyes have it
helpful clinical signs....the eyes have it

- Leakage of blood into CSF space with meningeal irritation:
  85% Saccular Aneurysm, 10% perimensephalic, 5% other

- Headache in isolation in up to 50-70%

- Thunderclap (peaks within mins & lasts at least 1hr)

- ‘Worst Ever’ with:
  - Vomiting in 70% (vs 42% in benign)
  - Meningism; photophobia
  - LOC: typically transient at onset (26-50%)
  - Transient focal neurology (33%)
  - Subhyaloid haemorrhage (17%)
  - Delirium (16%)
  - Epileptic seizures (6-9%)
  - Sudden death (10%)

Subhyaloid haemorrhage

1. Sub-arachnoid haemorrhage
helpful clinical signs...the eyes have it

Ptosis & conjunctival inj

Horners

6th Cranial nerve plasy

Subhyaloid haemorrhage

Cluster headache

Cervical arterial dissection

6th Cranial nerve plasy

Venous sinus thrombosis

IIH

SOL

Sub-arachnoid haemorrhage
helpful clinical sign?.... meningeal irritation

Thomas et al 2002 - evaluating Kernig’s & Brudzinski sign in suspected meningitis:
sensitivity 5%
specificity 95%

→ not helpful when negative

↑ temp even when no CNS drive can cause headache
↑ temp will occur in both bacterial & chemical meningitis

high frequency of pyrexia in SAH but typically delayed and prominent 72 hrs on
Threshold of suspicion....

- Index of suspicion
- Vulnerable group: age > 50
- Tempo
- Landscape
- Triggers
- Change
- Systemic symp
- Neuro signs

Threshold of suspicion....
Threshold of suspicion.....

Ottawa (adult) SAH rule Perry et al 2013, JAMA

Alert with non-traumatic severe headache peaking within 1 hr w/o deficit

Investigate if ≥1 high-risk variables present:
1. Age ≥40 y
2. Neck pain or stiffness
3. Witnessed loss of consciousness
4. Onset during exertion
5. Thunderclap headache (instantly peaking pain)
6. Limited neck flexion on examination

Not for patients with new neurologic deficits, previous aneurysms, SAH, brain tumors, or history of recurrent headaches (≥3 episodes over the course of ≥6 mo)

Negative Predictive Value 100%;

high sensitivity ~100%
but very poor specificity
(15% reducing to 7% on validation testing_Bellolio et al 2015, Am J Em Med)
Tempo: abrupt, 1st & worst with a progressive course
CT v CSF debate in the diagnosis of Subarachnoid haemorrhage

<table>
<thead>
<tr>
<th></th>
<th>&lt;6 hrs</th>
<th>&lt;12 hrs</th>
<th>&lt;24hrs</th>
<th>&lt;1wk</th>
<th>&lt;2wk</th>
<th>3wk</th>
<th>4wk</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CT</strong></td>
<td>&gt;98%</td>
<td>95%</td>
<td>90-93%</td>
<td>50%</td>
<td></td>
<td>poor</td>
<td>poor</td>
</tr>
<tr>
<td><strong>CSF xanthochromia</strong></td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>~100%</td>
<td>~100%</td>
<td>70%</td>
<td>40%</td>
</tr>
</tbody>
</table>

Timeline of Xanthochromia in SAH: Vermeulen, Van Gin, JNNP 1990; 53; 365-372 & JNNP 1989; 52; 826-828
Reliability of CT alone if done immediately?

CT for SAH in patients with acute headache stratified by timing of scan _Perry et al BMJ 2011

<table>
<thead>
<tr>
<th>Time from onset to scan</th>
<th>N</th>
<th>% sensitivity (95% CI)</th>
<th>% specificity (95% CI)</th>
<th>PPV (95% CI)</th>
<th>NPV (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>3132</td>
<td>92.9 (89 to 95.5)</td>
<td>100 (99.9 to 100)</td>
<td>100 (98.3 to 100)</td>
<td>99.4 (99.1 to 99.6)</td>
</tr>
<tr>
<td>≤ 6 hrs</td>
<td>953</td>
<td>100 (97.0 to 100)</td>
<td>100 (99.5 to 100)</td>
<td>100 (96.9 to 100)</td>
<td>100 (99.5 to 100)</td>
</tr>
<tr>
<td>&gt; 6 hrs</td>
<td>2179</td>
<td>85.7 (78.3 to 90.9)</td>
<td>100 (99.8 to 100)</td>
<td>100 (96.3 to 100)</td>
<td>99.2 (98.7 to 99.5)</td>
</tr>
</tbody>
</table>

—but need real world DGH reporting to insure can extrapolate

CT angio to avoid LP?

retrospective, n729 (88 in house)_Alons et al, 2018

CTA for severe acute non-traumatic headache + normal exam +normal CT
7.4% vascular abnormality but only 1.6% thought to be causative
SAH: need for improved management_NCEPOD 2013

care unsatisfactory in 42% of cases [177/427]; 30% requiring improvement in clinical approach

43% of cases [32/75] in 1° care diagnosis overlooked [sig impact on outcome in 23]

18% no formal neuro exam performed or documented in initial presentation in secondary care

In 2° care

- no SAH management protocol = 27%
- no acute severe headache protocol = 32%
Thunderclap beyond SAH

Sudden onset headache

CT head

Normal

Abnormal

CSF studies

Normal

Abnormal

LP: high opening pressure

LP: haemorrhage

LP: elevated WBC

SAH- refer NEUROSURGEON + start Rx

? meningitis- refer ID + start Rx

SAH
Stroke
CVST
SOL etc
seek APPROPRIATE OPINION

LP: elevated WBC

Anderson_manuscript in prep
Sudden onset headache

CT head

Normal
Abnormal

Ipsilateral: cranio-cervical pain + Horner's or other CN

Recent recurrent thunderclap

Postural Valsalva trigger
Visual obscuration
Pulsatile tinnitus
Papilloedema

MR angiogram ± dissection seq

Normal ?Primary

MR venogram ± with contrast

Normal
Abnormal

LP: high opening pressure

LP: haemorrhage

LP: elevated WBC

SAH- refer NEUROSURGEON + start Rx

LP: haemorrhage

LP: elevated WBC

RCVS- refer NEURO + stop driving factor

Dissection- refer NEURO + start anticoag

Ipsilateral:

SAH Stroke CVST SOL etc
seek APPROPRIATE OPINION

MR venogram ± with contrast

SAH- refer NEUROSURGEON + start Rx

LP: haemorrhage

LP: elevated WBC

CVST

SOL

IIH/ SIH

?meningitis- refer infectious disease + start Rx

SAH- refer NEUROSURGEON + start Rx

LP: haemorrhage

LP: elevated WBC

Anderson_manuscript in prep
Acute headache exacerbation in the IIH shunted patient

- Limited evidence of benefit of shunting or stenting for headache alone
- Post shunt ongoing headache in 68% at 6 months and 72% at 2 yrs

Visual led pathway
## Treatment in the ER

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>NNT 2hr pain freedom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paracetamol</td>
<td>100mg po</td>
<td>12</td>
</tr>
<tr>
<td>Naproxen</td>
<td>500mg po</td>
<td>11</td>
</tr>
<tr>
<td>Aspirin</td>
<td>900mg po</td>
<td>8.1</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>400mg po</td>
<td>7.2</td>
</tr>
<tr>
<td>Prochlorperazine</td>
<td>10mg iv</td>
<td>17</td>
</tr>
<tr>
<td>Metoclopramide</td>
<td>20mg iv</td>
<td>17</td>
</tr>
<tr>
<td>Prochlorperazine + aspirin</td>
<td>10mg po + 900mg po</td>
<td>≡ sumatriptan 100mg po</td>
</tr>
<tr>
<td>Metoclopramide + paracetamol</td>
<td>20mg po +1000mg po</td>
<td>≡ sumatriptan 100mg po</td>
</tr>
<tr>
<td>Sumatriptan</td>
<td>50mg po</td>
<td>6.1</td>
</tr>
<tr>
<td>Sumatriptan +naproxen</td>
<td>50mg +500mg po</td>
<td>4.9</td>
</tr>
<tr>
<td>Sumatriptan</td>
<td>100mg po</td>
<td>4.7</td>
</tr>
<tr>
<td>Sumatriptan 6mg s/c</td>
<td>6mg subcut</td>
<td>2.3</td>
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Treatment in the ER _ aim for at least 1 cured out of every 5 treated

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SIGN 155 guidelines _migraine
Treatment in the ER_head to head supports avoiding opiates

- n127 patients enrolled & trial halted by data monitoring committee
- 1mg IV hydromorphone v 10mg IV prochlorperazine
- 1° outcome: patients with mild or no headache at 2 hrs
- significant superiority established for prochlorperazine: achieved in 37 of 62 (60%) v 20 of 64 (31%)

- Conclusions: IV hydromorphone is substantially less effective than IV prochlorperazine for the treatment of acute migraine in the ED and should not be used as first-line therapy.
- Class 1 evidence
Proposed treatment for acute headache in the ER

FRONT DOOR
- IV/IM metoclopramide or prochlorperazine
- IV paracetamol 1000mg
- IV fluid support
- environment

No active vascular concerns
- add SC/PO Sumatriptan

Once haemorrhage excluded
- Regular: PO ibuprofen or aspirin + anti-emetic
- ± PRN: PO/ IV paracetamol
The future is bright.....

Lasmitidan
- oral
- selective serotonin 5HT$_{1F}$ agonist
- CNS penetrance
- 2hr pain freedom comparable to triptans
- response not dependent on prior triptan response
- no vasoconstrictor effects

![Chemical Structure of Lasmitidan](image)

**Graph:**
- Placebo n=1262
- Lasmitidan n=1258

**Bar Chart:**
- % headache free at 2 hrs
- SPARTAN: Placebo (15%) vs Lasmitidan 200mg (30%)
- SAMURAI: Placebo (20%) vs Lasmitidan 200mg (40%)
The future is bright..... the new target: calcitonin gene-related peptide (CGRP)

Edvinsson et al, Nature rev neuro 2018
# The future is bright... Phase 3 CGRP mAb’s & small molecule antagonist (EM)

<table>
<thead>
<tr>
<th>Target</th>
<th>Fremanezumab</th>
<th>Galcanezumab</th>
<th>Eptinezumab</th>
<th>Ubrogepant (small molecule)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>target</strong></td>
<td>CGRP Receptor</td>
<td>CGRP</td>
<td>CGRP</td>
<td>CGRP Receptor</td>
</tr>
<tr>
<td><strong>Route of admin</strong></td>
<td>SC (mthly)</td>
<td>SC (mthly)</td>
<td>SC (2 wkly or mthly)</td>
<td>PO</td>
</tr>
<tr>
<td><strong>T ½ (days)</strong></td>
<td>21</td>
<td>45</td>
<td>28</td>
<td>31</td>
</tr>
<tr>
<td><strong>EM phase 3</strong></td>
<td>STRIVE LIBERTY (failed 4 prev) ARISE</td>
<td>HALO EM</td>
<td>EVOLVE 1&amp;2</td>
<td>PROMISE 1</td>
</tr>
<tr>
<td><strong>arms</strong></td>
<td>P, 70mg, 140mg</td>
<td>P, 225, 675mg</td>
<td>P, 120mg, 240mg</td>
<td>P, 100mg, 300mg</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>ACHIEVE 1&amp;2 ACHIEVE 1: P, 50mg, 100mg ACHIEVE 2: P, 25mg 50mg</td>
</tr>
</tbody>
</table>
The future is bright..... Phase 3 small molecule antagonist (EM)

Ubrogepant (small molecule)
CGRP Receptor
PO
?
ACHIEVE 1&2

ACHIEVE 1: P, 50mg, 100mg
ACHIEVE 2: P, 25mg, 50mg

% headache free at 2 hrs

<table>
<thead>
<tr>
<th></th>
<th>Placebo n=1436</th>
<th>Ubrogepant n=1327</th>
</tr>
</thead>
<tbody>
<tr>
<td>50mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100mg</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The future is bright.....Phase 3 CGRP monoclonal Ab (EM)

[Graph showing 50% responder rate for different doses and treatments]

PROMISE 1: Maximum Clinical Benefit with Eptinezumab Attained by Day 1 Post-Infusion and Sustained

Immediate and Sustained Response: >50% reduction in percent of patients experiencing migraine within 1 day
<table>
<thead>
<tr>
<th>Event</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| Front door | - IV/IM metoclopramide or prochlorperazine  
- IV paracetamol 1000mg  
- IV fluid support  
- environment |
| Vascular concerns or failed triptan | • add PO Lasmitidan |
| Once haemorrhage excluded | • Regular: PO ibuprofen or aspirin + anti-emetic  
• ± PRN: PO/ IV paracetamol |
| If all excluded and migraine | • Consider CGRP blockers |
Thank you for listening
Reversible Cerebral vasoconstriction syndrome (RCVS)

• majority spontaneous but can be triggered
eg. vasoactive drugs (aka Call Fleming) or post-partum

• monophasic course

• recurrent thunderclap over wks; spontaneously resolves (4-6 wks)

• transient disturbance in the control of cerebral vascular tone

• early complication: cortical SAH, ICH, seizures and PRES

• on cerebral angio ‘beading’ which fully reverses on f/u

Ducros & Bousser_Practical Neurology 2009
Thunderclap headache

- Peaks within minutes - 80% within 1st minute.

1° v 2°: clinically cannot differentiate

- Primary TCH → diagnosis of exclusion
  - SAH → CT/LP earlier or CTA later
  - Arterial Dissection → focal Neuro signs, MRA
  - Venous Sinus Thrombosis → raised CSF OP, CTV
  - Pituitary Apoplexy
  - Spontaneous Intracranial Hypotension
  - 3rd ventricle colloid cyst
  - Reversible cerebral vasoconstriction syndrome (RCVS)

- EMERGENCY Referral required
Cervical arterial dissection

- Headache occurs in 60-95%
- typically progressive but thunderclap in up to 1/5
- ipsilateral
- usually headache plus (develops 4 days post [Silbert et al 1995])
  - Horner's
  - pulsatile tinnitus,
  - amaurosis fugax
  - cerebral ischaemic symptoms
  - ipsilateral cranial palsies in 12% [Mokri et al 1996]
    - 5% lower cranial nerve involvement- usually XII
    - 4% palsy of cranial nerve V
    - 3% ocular motor palsies

- Treatment: anticoagulation/ antiplatelet

CT Angiogram – Dissection ‘flap’
CT Angiogram
CT Angio 3D
MR – T1 fat suppression

Long segment of narrowed lumen
Dissecting hematoma
Hematoma
ICA
ECA
CCA

Q4. differential for severe headache, visual loss, diplopia?

- Pituitary apoplexy
- IIH
- GCA
- SOL; PCOM aneurysmal mass
- Cervical arterial dissection
Cerebral venous sinus thrombosis

Cerebral vein thrombosis
  ▲ Venular and capillary pressure
    ▼ Capillary perfusion
      ▼ Cerebral perfusion
        ▼ Ischemic injury
          ▼ Cytotoxic edema
            ▼ Headache, focal neurological findings, seizures, mental status change

Cerebral sinus thrombosis
  ▲ Intracranial pressure
    ▼ CSF absorption
      ▼ Venous and capillary rupture
        ▼ Vasogenic edema
          ▼ Parenchymal hemorrhage
            ▼ Disruption of blood-brain barrier
              ▼ Increased venous pressure
Cerebral venous sinus thrombosis

have a low index of suspicion especially in progressive states /seizures

→ direct relevant investigations

- look for papilloedema
  - Present in up to 25% of CVT [Crassard et al 2005]
  - n131 papilloedema/?IIH- 10% CVT [Ferro et al 2004]

- >80% will have elevated OP on LP but not always when hyper-acute

- direct appropriate imaging to include venography if suspected
Young female BMI COCP Vit A, tetracyclines,

Idiopathic intracranial hypertension

Idiopathic Intracranial Hypertension

**IIH Diagnostic criteria**

A. Papilloedema
B. Normal neurological examination (except sixth cranial nerve palsy)
C. Neuroimaging: normal brain parenchyma (no hydrocephalus, mass, structural lesion or meningeal enhancement). Venous thrombosis excluded in all.
D. Normal CSF constituents
E. Elevated lumbar puncture pressure ≥ 25cmCSF

**IIH Consensus guidelines** _Mollan et al Jnnp 2017_
Idiopathic intracranial hypertension

Young female
high BMI
COCP
Vit A, tetracyclines,

IIH Diagnostic criteria
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Radiological findings
- empty sella
- dilated optic nerve sheaths
- optic nerve tortuosity
- posterior globe flattening
- protrusion optic nerve head
- narrowing of Meckel's cave
- reduction in the diameter of the cavernous sinus
- venous flow voids/ stenoses

Papilloedema Identified
Record:
1. Visual acuity
2. Formal visual fields
3. Dilated fundoscopy
NB Regular assessment of vision if affected

Check blood pressure and exclude malignant hypertension (ref)

Brain Imaging within 24 hours (CT/MRI) AND venography
No lesions identified.

Lumbar puncture
Opening pressure >25cm CSF; normal contents.

Exclude secondary causes

Idiopathic Intracranial Hypertension
Is the vision at imminent risk?
Is this a woman, of reproductive age, with a BMI<30kg/m²?
Is this person not female, not of reproductive years and with a BMI<30kg/m²?

Fulminant IIH Typical IIH Atypical IIH
Idiopathic intracranial hypertension management

Idiopathic Intracranial Hypertension

Weight management advice for all with a BMI ≥30kg/m² *(Question 2)

No immediate threat to vision

Consider medical therapy with acetazolamide *(Question 8, 9)

Headache assessment:
1. Evaluate the headache phenotype *(Question 11, 12)
2. Eliminate medication overuse elements *(Question 14)
3. Treat *(Question 13)

Ophthalmology assessments *(Question 23, Table 5)

If significant deterioration of visual function, consider diagnostic lumbar puncture

Vision threatened (Fulminant IIH)

Temporising lumbar drain if surgery planned >24 hours

CSF diversion *(Question 4)*

OR

Optic nerve sheath Fenestration *(Question 5)*

If pathologically high (see figure 1)

If not pathologically high (see figure 1)

Re-evaluate

IIH Consensus guidelines _Mollan et al jnnp 2017_
Intracranial hypotension

- CSF volume depletion as a result of leakage:
  - Iatrogenic (post LP, post surgery)
  - Traumatic (skull fracture)
  - “Spontaneous” dural tear (?minor initial trauma)

- At risk: hypermobility syndrome/ Marfan’s

- Clinical:
  - Orthostatic headache
  - Orthostatic component ‘fades’: start hx from the beginning
  - Headache worsens as day goes on
  - Assoc orthostatic: pulsatile tinnitus, nausea & dizziness

- Investigations:
  - MRI + contrast: meningeal enhancement, tonsillar descent, subdurals
  - May need to pursue: MRI spine (FIESTA), CSF isotope study, ICP monitoring

- Treatments:
  - Post-LP: spontaneous resolution with fluids and strict bed rest
  - Epidural blood patching (may require several), IV caffeine
Intracranial hypotension
Temporal arteritis

Critical to identify as 13% permanent visual loss

Ha characteristics unhelpful other than allodynia:
   Headache, scalp tenderness, jaw claudication, weight loss, low grade fever, non-specifically unwell

Clinical suspicion high
   - Consider in >50yrs; mean age 71yrs
   - Most helpful clinical symptoms/signs are:
     - Diplopia
     - Jaw claudication
     - Temporal artery beading/ Palpable enlarged temporal artery
     - Temporal artery tenderness
     - Absent temporal artery pulse

   Excess of vertebrobasilar territory ischaemic events: 7% vestibular symptoms [Caselli 1998]

ESR/Plt and CRP Normal < 1%

Temporal Artery Biopsy as soon as possible— but can be patchy; (?role of US)

**Treatment:** High dose steroids 1mg/kg - initial improvement of PMR by 48hrs and ESR by 72 hrs
   BUT steroids produce non-specific improvement in many types headaches & this does NOT confirm the diagnosis
Temporal arteritis

US of greater sensitivity but not greater specificity cf biopsy - role for proceeding to biopsy in US negative group

TABUL study Luqmani et al at 2016

Color-coded duplex sonography of the temporal artery:
- a) normal branch, longitudinal and transverse views;
- b) transverse views;
- c) in acute temporal arteritis, showing hypoechoic wall thickening (arrows), longitudinal and transverse views

Fragmentation /distortion of internal elastic lamina

Dense inflammatory infiltrate with giant multinucleated cells

Intimal thickening & transmural inflam. Obliteration of the lumen.

Normal H&E

TA H&E

Normal EVG

TA.EVG