

Alien v/s predator: battle for time

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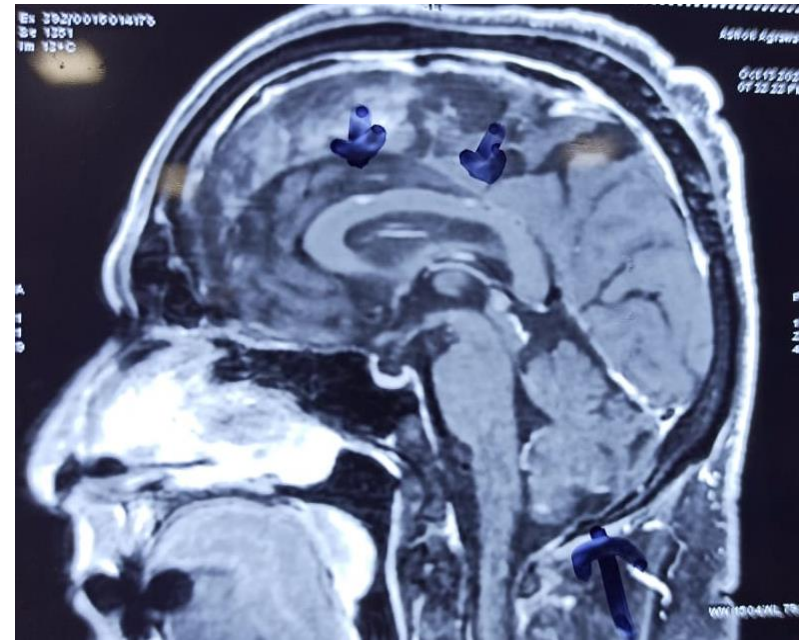
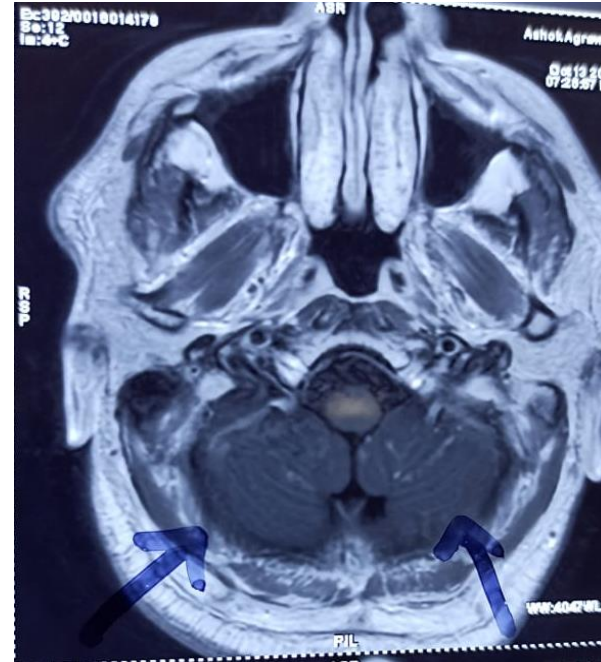
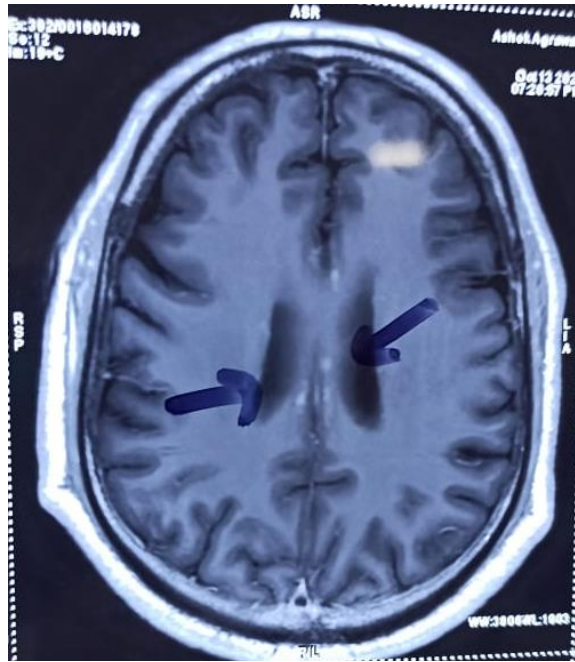
Surat

CASE

- 62/M
- Newly diagnosed DM
- No travel history or other comorbidities
- R/O of surat -> stayed in Mumbai for last few yrs

- C/O: (R) LL numbness & weakness with progressive difficulty in walking & imbalance since 2 years
- history of urinary retention since last 1 yr
- Headache since last few months – TCA taken

- CNS - DTR (R) LL +++, rest NAD
- Usg scrotum – B/L bulky heterogenous epididymis with multifocal calcification & (R) small chronic abscess s/o chronic granulomatous inflammation
- Routine labs – NAD
- CRP & ESR – raised
- MRI spine – NAD
- HIV - negative



Abnormal leptomeningeal enhancement with micronodular pattern predominantly in interfolial space along the inferior cerebellar surface S/O (reactivated) granulomatous meningitis – infective v/s noninfective

- CSF –P- 100, S- 39, wbc- 180(n-12, L-68)
 - Gene xpert MTB – negative
 - India ink – negative
-
- Empirically ATT started with tapering steroids -> Partial decrease in symptoms

Readmitted – 1 month later

- 1/11/21 – history of drowsiness & convulsion
- CSF – P – 119 , S- <2
- Gene xpert – negative (2/2)
- 2nd line ATT started

- Repeat MRI – Previous lesion has marginally reduced **but acute infarct** B/L parasagittal frontal lobe, perisylvian cortex, corona radiate, lentiform nucleus, internal capsule, temporal lobe, cerebellar vermis **represent vasculitis infarct subsequent to meningitis**

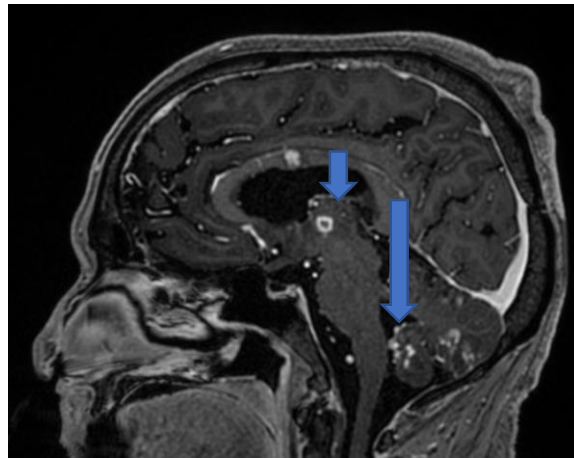
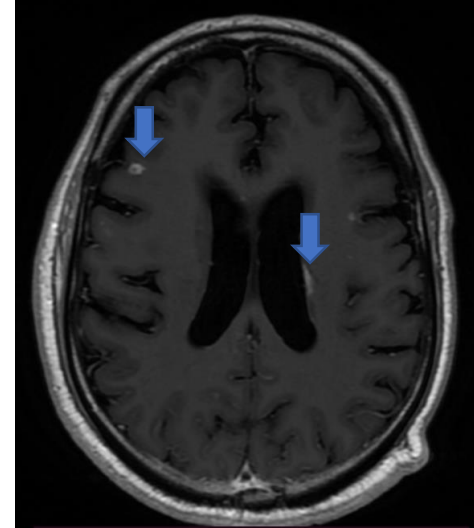
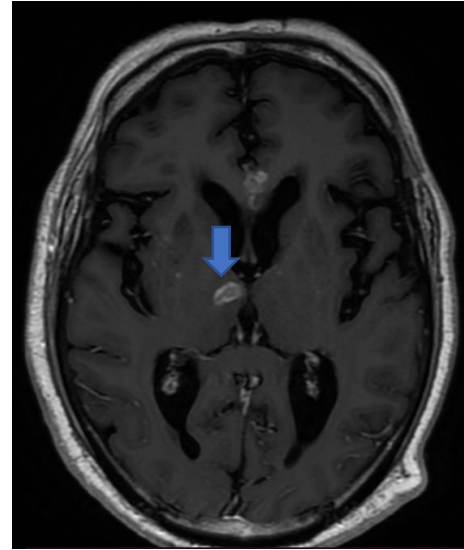
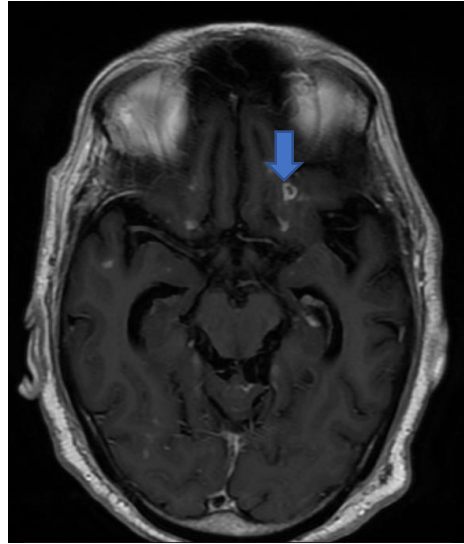
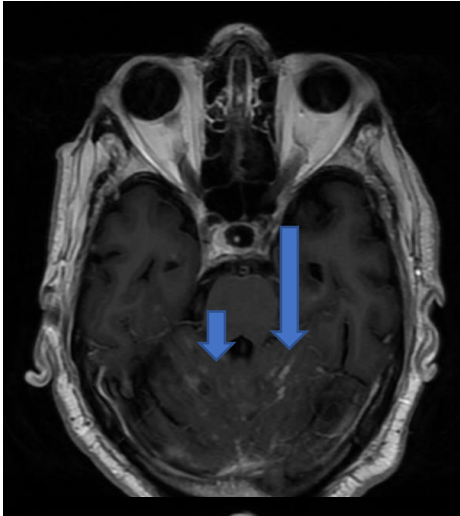
Summary

- Elderly male, immunocompetent
- Focal neurological f/b convulsion
- MRI – granulomatous meningitis with vasculitis
- USG scrotum – chronic granulomatous abscess
- CSF- high protein, low sugar, lymphocytosis
- No response to 1st line ATT

Differential diagnosis??

- CSF – INDIA INK – Positive, CrAg-positive
- L-Amb & 5FC started, ATT withdrawn
- Neurological no major response (on tracheostomy support)
- recurrent tapping done – eye opening +
- L – Amb & 5FC continued for 26 days, no major neurological response

	Protein	Sugar	India ink	CrAg	Culture (KOH)
2/11	119	<2			
9/11	112	<2	+	+	Budding yeast
19/11	100	5		+	Budding yeast
25/11	96	13	+		Budding yeast
26/11			+		Budding yeast



Leptomeningitis- diffuse smooth/irregular thickening -contrast enhancement along the cortical sulci, cerebellar folia, along brainstem

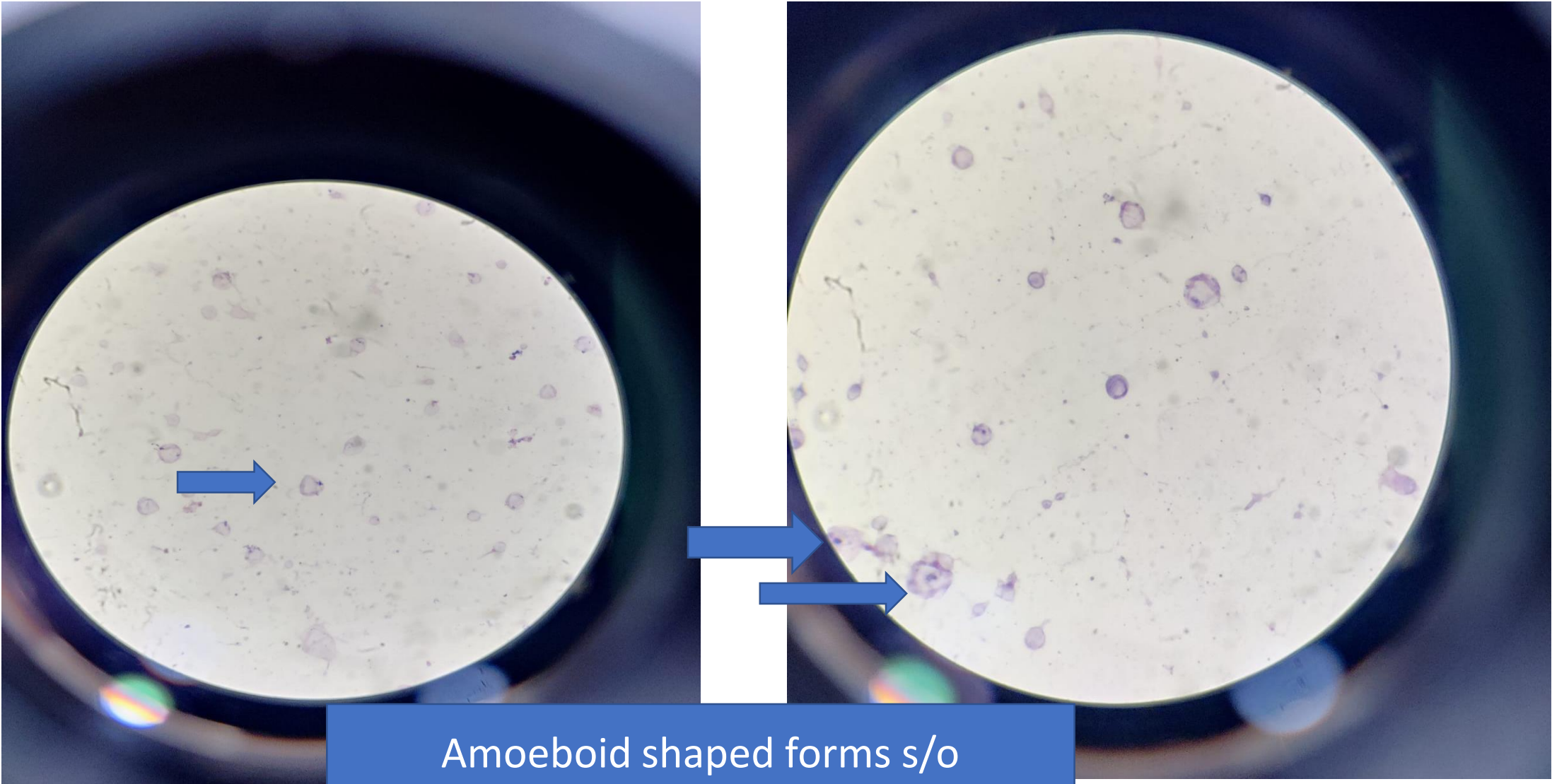
Presence of multifocal granulomatous lesions involving cortex, gray white junction, brainstem, basal ganglia, thalami, cerebellum
Enhancement patterns-ring - solid enhancing- target like – punctate – heterogeneous

Shifted to surat -> ID Opionion

- GCS- 5 (deteriorating)
- L-Amb & 5FC continued,
- Post Tapping done -> GCS – 6

- Repeat CSF – Protein – 179, S – 19, opening pressure - 30
- India Ink - negative
- Plan -> Biofire – was not affording (for confirming by PCR & for strain of cryptococcus)
- GCS – STATIC for few days

CSF SHOWED



Ameboid shaped forms s/o trophozoites of acanthamoeba spp

- CSF C/S - Negative
- For further confirmation -> CSF was required -> CHARCOL YEAST AGAR (acanthamoeba), fluorescent microscopy

- Meanwhile patient develop abdominal sepsis
- Succumb

QUESTION?

1. Immunocompetent, No history of exposure for crypto, was it cryptococcal meningitis? if yes then which species?
2. If crypto meningitis, then y it was not responding to almost 1 month therapy of L-Amb & 5FC?
3. Would CSF biofire would have been useful for diagnosis?
4. Was CSF CrAg false positive? If, then y?
5. Was it dual pathology → acanthamoeba spp + cryptococcal ?
6. Radiological Difference between 2 pathology?

False positive CrAg

- Use of BBL Port –A-Cul transport vials (52 sample)

-[Deborah A. Wilson et al. J Clin Microbiol. 2011 Feb; 49\(2\): 702–703](#)

- *Trichosporon beigeli* or *Capnocytophaga canimorsus*, presence of a malignancy or rheumatoid factors, and contamination with agar, agarose, or syneresis fluid during laboratory pipetting

- Thomas C. Stoeckli et al. <https://doi.org/10.1086/319212>

MRI difference

	Cryptococcal	Acanthamoeba – 2 form a) Primary amoebic meningoencephalitis b) Granulomatous amoebic encephalitis
Key finding	1) Dilated VR/perivascular spaces 2) Cryptococcoma (immunocompetent > immunosuppressed)	1) Multifocal Granulomatous lesion 2) Intralesional hemorrhage 3) Initial ischemic stroke – trophozoite predilation to VR space
Leptomeningitis	+/-	Basal meningitis +/-
Hydrocephalus	+/-	Occasionally late finding

Lesson learnt

1. Immunocompetent cryptococcal patient are difficult to treat
2. CSF CrAg has high sensitivity & specificity, false positive r reported
3. The mortality rate of granulomatous amebic encephalitis (GAE) due to *Acanthamoeba* exceeds 90%