Clinical Image

Rhinosporidiosis presenting as an Oropharyngeal Mass

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A 41-year-old male presented with progressive difficulty in swallowing and a foreign body sensation in throat for 10 months. Oral cavity examination revealed a huge pinkish red lobulated, polypoidal, smooth surfaced mass involving full of oropharynx (Figure 1). Nasal cavity examination revealed a rough granular mass arising from posterior 2/3 of inferior meatus extending posterior and inferiorly from choana. Computed Tomography of the Oropharynx showed mass in nasal cavity extending into the oropharynx (Figure 2). Endoscopic wide base excision and cauterization was done and was sent for histopathological examination (Figure 3).The patient was followed up and till now no recurrence for past 9 months.

Rhinosporidiosis is a chronic, non contagious sporadic disease caused by *Rhinosporidium seeberi*.[1] It is a disease prevalent in hot tropical climate of endemic zones like South India, Sri Lanka, East Africa, parts of America.[2] The endospores are transmitted through water and dust into the nasal mucosa by traumatic inoculation where they mature subepithelially and after maturation burst with release of sporangia into the tissues.[3] The disease is characterized by a reddish, friable, polypoidal, hyperplastic mass mostly occurring in the nasal cavity and nasopharynx, however, sporadic occurrence in extra nasal sites is also observed. The most effective treatment is wide excision with a cutting diathermy and cauterization of the base of lesion.[3] Previously antifungal agents were also used but were ineffective. Dapsone has been tried to prevent recurrences. The recurrence rate is high and regular follow up is recommended.

Figure 1: Mass in the oropharynx

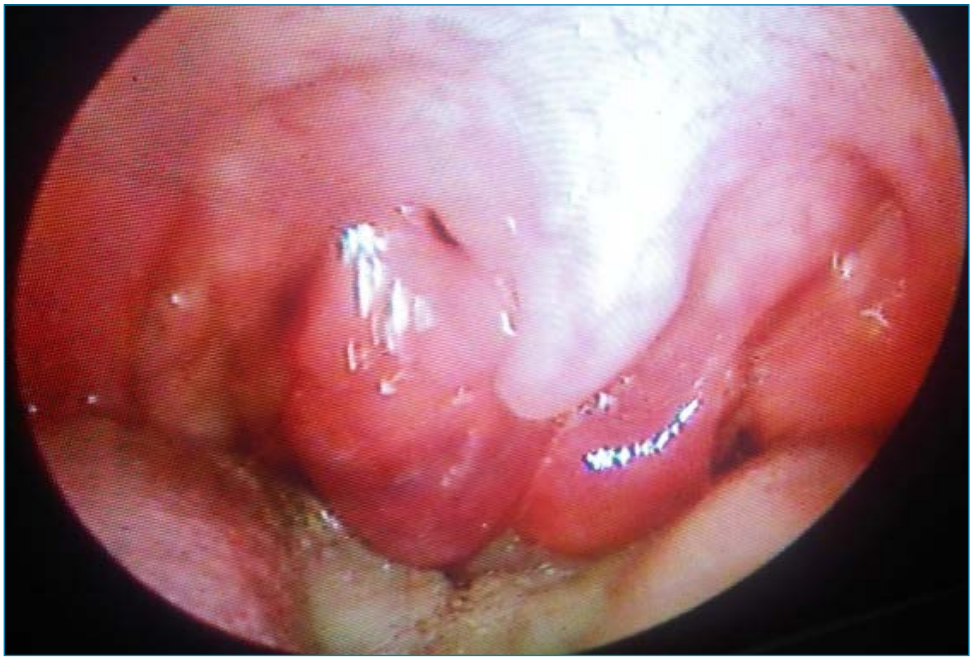

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Figure 2: CT Paranasal sinus showing mass extending into oropharynx

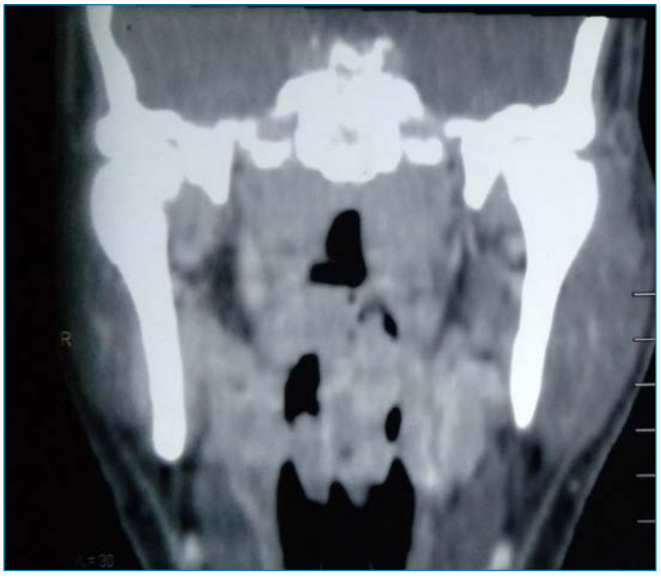

Figure 3: Histopathological examination showing sporangia with sporangiospores [H&E, 10x]

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Excessive Daytime Sleepiness Could be Linked to Alzheimer's

Study Objectives: To determine the association of excessive daytime sleepiness (EDS) and napping with subsequent brain β-amyloid (Aβ) deposition in cognitively normal persons.

Methods: We studied 124 community-dwelling participants in the Baltimore Longitudinal Study of Aging Neuroimaging Substudy who completed self-report measures of EDS and napping at our study baseline and underwent [11C] Pittsburgh compound B positron emission tomography (PiB PET) scans of the brain, an average ±standard deviation of 15.7 ± 3.4 years later (range 6.9 to 24.6). Scans with a cortical distribution volume ratio of >1.06 were considered Aβ-positive.

Results: Participants were aged 60.1 ± 9.8 years (range 36.2 to 82.7) at study baseline; 24.4% had EDS and 28.5% napped. In unadjusted analyses, compared with participants without EDS, those with EDS had more than 3 times the odds of being Aβ+ at follow-up (odds ratio [OR] = 3.37, 95% confidence interval [CI]: 1.44, 7.90, p = 0.005), and 2.75 times the odds after adjustment for age, age2, sex, education, and body mass index (OR = 2.75, 95% CI: 1.09, 6.95, p = 0.033). There was a trend-level unadjusted association between napping and Aβ status (OR = 2.01, 95% CI: 0.90, 4.50, p = 0.091) that became nonsignificant after adjustment (OR = 1.86, 95% CI: 0.73, 4.75, p = 0.194).

Conclusions: EDS is associated with more than 2.5 times the odds of Aβ deposition an average of 15.7 years later. If common EDS causes (e.g., sleep-disordered breathing, insufficient sleep) are associated with temporally distal AD biomarkers, this could have important implications for AD prevention.

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