

# A case report of invasive sinus aspergillosis with mycotic aneurysm of vertebral artery and subarachnoid hemorrhage

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## PATIENT HISTORY

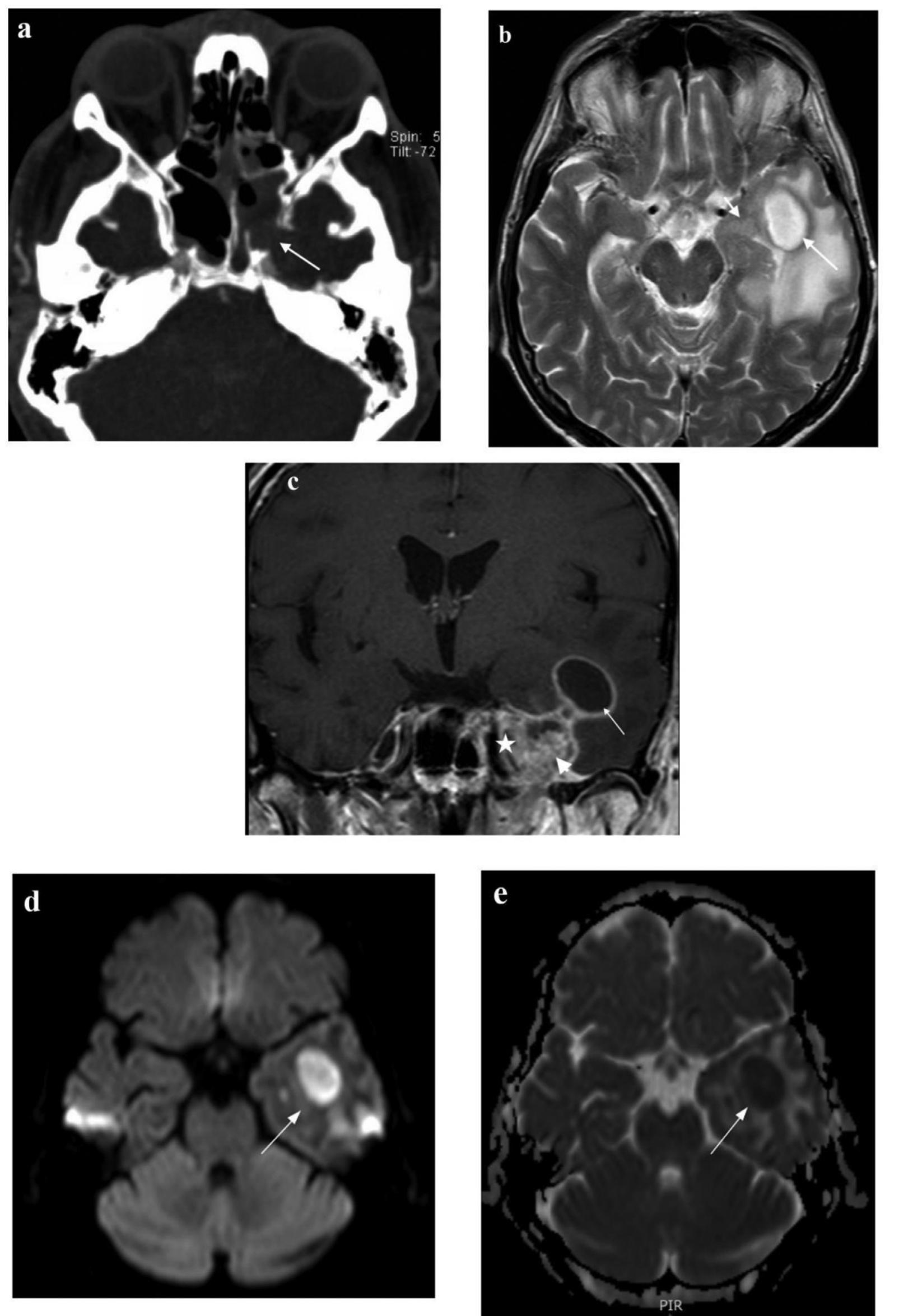
**History of present illness:** 66-year-old man with one-month history of progressive gait instability, cognitive decline, and double vision, accompanied by weight loss for one year and one-year history of symptoms of left-sided trigeminal neuralgia

### No significant past medical history

**Physical examination:** moderate cognitive impairment (MMSE=18/30 points), slightly constricted left pupil, double vision in all directions of gaze, fasciculations in the lower half of the face, and irregular rest tremor in all four limbs

**Laboratory tests:** minimal elevation of CRP and leukocytosis with relative lymphopenia (relative lymphocyte count 17%); serologic tests for HIV, *T. pallidum*, and *B. burgdorferi* were negative

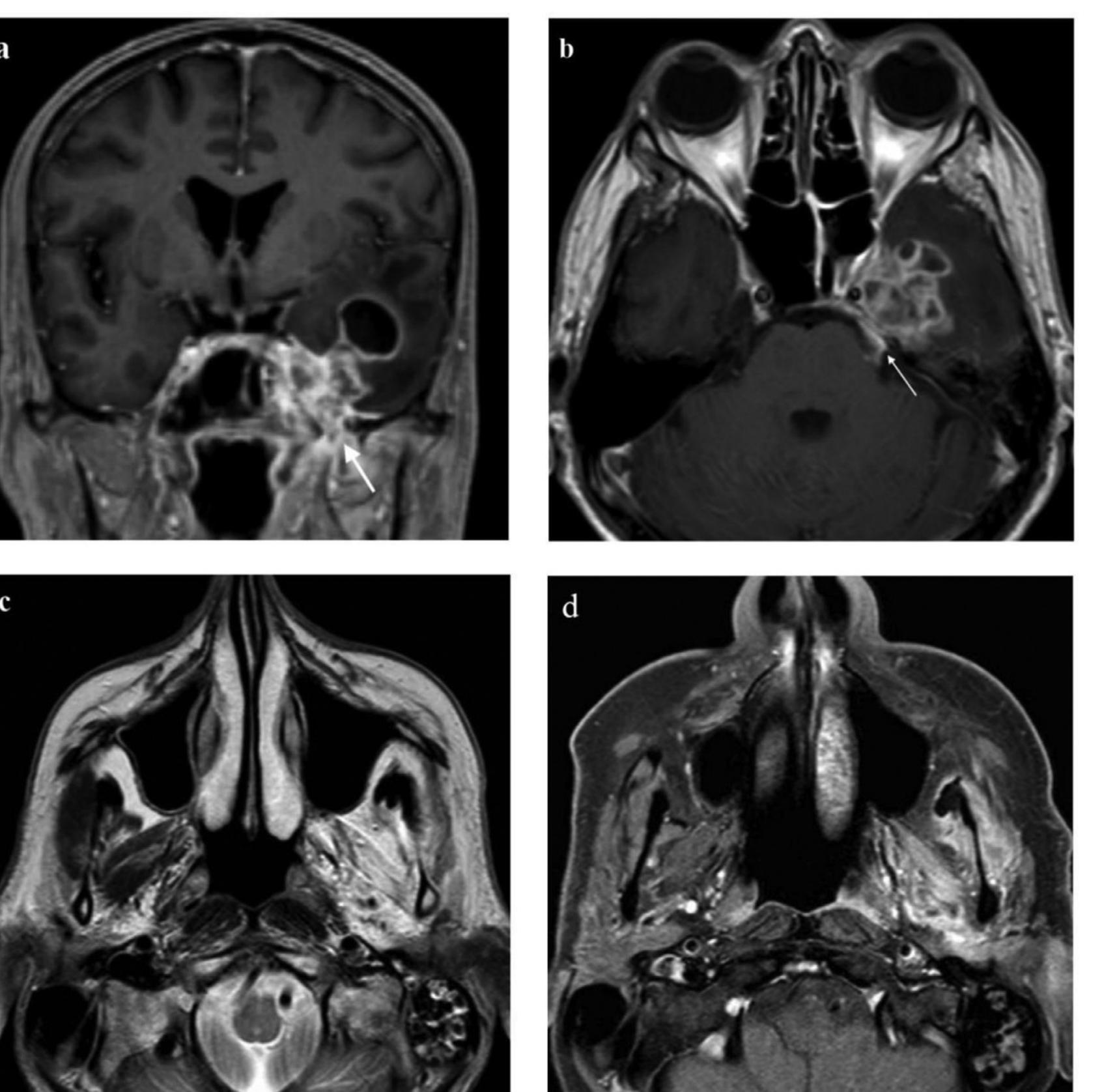
### Imaging:



**Figure 1.** **a.** Head CT in axial plane (bone window) shows thickened mucosa and a defect in the lateral wall (arrow) of the sphenoidal sinus. **b.** The cystic part of the temporal lobe lesion has a T2 hypointense rim (long arrow), and the solid part has a T2 hypointense signal (short arrow). **c.** Corresponding T1 obtained after gadolinium administration showed rim enhancement of the cystic part (arrow) and heterogeneous enhancement of the solid part (arrow head), also affecting the cavernous sinus (asterix). **d.** DWI sequence shows a hyperintense lesion with a slightly hypointense center (arrow). **e.** There is marked restriction of diffusion on ADC maps in the cystic part (arrow).

## CLINICAL COURSE

- dexamethasone and combination of **cefepime, metronidazole, and amphotericin B** (which is later switched to voriconazole)
- surgical debridement of the left sphenoid sinus --> histopathological examination: **chronic ulcerative inflammation of the sinus mucosa caused by Aspergillus fumigatus**
- no immunodeficiency** identified, apart from hypogammaglobulinemia
- transient improvement after pharmacological and surgical treatment
- hospitalization day 16: found unresponsive – resuscitation – head CT showed **subarachnoid hemorrhage (SAH)** and brain edema (*figure 3a and 3b*)
- CTA** of intracranial arteries: fusiform **aneurysm of the left vertebral artery** (*figure 3c*)
- worsening of SAH and cerebral edema
- death on day 18 after admission – **cause of death: ruptured mycotic aneurysm** of the left vertebral artery, caused by *Aspergillus spp*



**Figure 2.** **a.** Contrast enhanced T1-weighted image in coronal plane reveals thickening and enhancement of the third branch of the trigeminal nerve in left foramen ovale (arrow). **b.** Contrast enhanced T1-weighted image in axial plane shows thickening and enhancement of the cisternal part of the left trigeminal nerve (arrow). **c.** Muscles of the left masticator space demonstrate a T2 hyperintense signal and **d.** enhance after contrast agent due to denervation.

## DISCUSSION

### MRI signs of perineural spread:

- thickening and contrast enhancement of cranial nerves
- indirect sign of perineural infiltration: muscle denervation, demonstrated by hyperintense T2 signal and contrast enhancement of muscles [1,2]

### DWI imaging is the most sensitive modality for timely diagnosis:

- DWI hyperintense lesions with reduced ADC values --> restricted diffusion
- another possible DWI finding: target-like lesion with hypointense center, surrounded by hyperintense ring [3], seen in this case

### Aspergillus species are angioinvasive:

- subarachnoid hemorrhage, thrombosis, vasculitis, and fungal embolisms are possible
- intracranial aneurysms as a complication of invasive fungal sinusitis are rare - most occur in the internal carotid artery because of its proximity to sinuses; fungal aneurysm of the vertebral artery is a sporadic condition - only a handful of reports exist in the literature [4-6]
- high mortality of patients with intracranial fungal aneurysms (due to their rapid development and difficulty in diagnosing CNS aspergillosis)

### Immune status in invasive intracranial aspergillosis:

- more common in immunocompromised individuals
- our patient appeared to be immunocompetent, apart from hypogammaglobulinemia - unlikely that hypogammaglobulinemia affected the disease course, as the main defense against fungal infections is cellular immunity [7]
- possible causes for the aggressive nature of our patient's infection:
  - actual tissue penetration of voriconazole may not have been optimal
  - voriconazole metabolism and plasma concentrations can be highly individual [8], were not measured in our patient
  - corticosteroid therapy for cerebral edema, can simultaneously weaken the phagocytic activity of immune cells and in our case probably contributed to the spread of the infection [9]

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