Pituitary abscess in an HIV-1-infected patient

SAGE Open Medical Case Reports Volume 5: 1-4 © The Author(s) 2017 Reprints and permissions: sagepub.co.uk/journalsPermissions.nav DOI: 10.1177/2050313X17701374 journals.sagepub.com/home/sco

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Abstract

Objectives: Pituitary abscess is a rare occurrence among pituitary conditions, but one which carries life-threatening potential. An immunocompromised status is a risk factor for the development of a pituitary abscess; however, literature describes only one case among HIV-infected patients.

Methods and results: We present here a case of pituitary abscess in an HIV-1-positive patient, who demonstrated a shock status, disturbance of consciousness and generalized skin rash with laboratory findings of hypovolemia, acute inflammatory reaction and blood electrolyte abnormality. We first diagnosed the dermal manifestation as atypical generalized zoster, however, the other clinical findings could not be explained by VZV infection only. Combination with anamnesis, head magnetic resonance imaging scan and endocrine function test helped us to diagnose pituitary abscess. Although the etiology of the pituitary abscess could not be detected, the patient was successfully treated with antibiotics but followed by panhypopituitarism as sequela.

Conclusion: A pituitary abscess should be considered in HIV-infected patients with endocrinological abnormalities, visual field defects, and central nervous system infection signs or symptoms, regardless of CD4 T-cell counts.

Keywords

HIV-1, pituitary abscess, panhypopituitarism

Date received: 13 December 2016; accepted: 1 March 2017

Introduction

Pituitary abscess is characterized by symptoms and signs particular to central nervous system (CNS) infection, mass effect, and endocrine dysfunction¹ and accounts for <1% of all pituitary conditions.¹⁻³ Multiple infectious organisms including bacteria,⁴ fungi,^{5,6} and mycobacterium¹ have been reported as etiologic agents. Standard therapy includes surgical drainage and antibiotics; resulting hypopituitarism can persist and requires lifelong hormonal replacement.^{3,7} Risk factors for pituitary abscess include a preexisting pituitary lesion, sepsis,¹ paranasal sinusitis,⁸ meningitis, or an immunocompromised status.3 Many studies have reported cases of brain abscesses and/or encephalitis in HIV-infected patients;^{9,10} however, to this day, there is only one report of a pituitary abscess in this category of patients.¹¹ Here, we report a case of an HIV-1-infected patient who developed a pituitary abscess which was successfully treated with antibiotics only.

Case report

A 40-year-old Japanese man with longstanding HIV-1 infection was urgently hospitalized due to high fever, severe malaise, headache, and generalized skin rash (Figure 1(a) and (b)). For the past 9 years, he had been followed closely without treatment because his CD4 T-cell counts were stable at >500 cells/ μ L and he maintained a stable plasma viral load of around 1000 copies/mL. According to the medical interview at admission, he had had mild sinusitis and had received symptomatic treatment a month prior to presentation. His vitals were indicative of fever (39.5°C), shock status (systolic blood pressure, 84 mmHg; pulse rate, 110 beats/min) and mildly altered consciousness, which motivated admission to the intensive care unit. Further physical examination revealed a slight neck stiffness and diminished skin turgor, but no signs of focal neurological deficits or any form of visual disturbances. Laboratory test results showed moderate leukocytosis (11,600/ μ L), elevated

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Creative Commons Non Commercial CC BY-NC: This article is distributed under the terms of the Creative Commons Attribution-NonCommercial 3.0 License (http://www.creativecommons.org/licenses/by-nc/3.0/) which permits non-commercial use, reproduction and distribution of the work without further permission provided the original work is attributed as specified on the SAGE and Open Access pages (https://us.sagepub.com/en-us/nam/open-access-at-sage). C-reactive protein (12.1 mg/L), hyperproteinemia (9.6 g/dL), acute renal dysfunction (creatinine, 1.54 mg/dL), severe hyponatremia (116 mEq/L), and hyperkalemia (7.3 mEq/L). Full-body and head computed tomography (CT) scans detected only moderate swelling of lymph nodes in the cervical area. The levels of total protein, IgG, and leukocytes in the cerebrospinal fluid (CSF) were all slightly elevated at 66.5 mg/dL (normal range: 10–40 mg/dL), 14.6 mg/dL (normal range: 1.0–3.0 mg/dL), and 56/ μ L (neutrophils 1.8%, monocytes 98.2%), respectively. The glucose level in the CSF was normal. Serology for human simplex virus 1 and 2, varicella-zoster virus (VZV), hepatitis B virus, cytomegalovirus, Epstein–Barr virus, and syphilis demonstrated previous infection with these pathogens. After consulting



Figure 1. (a) Diffuse varicella-like eruption on the ventral and dorsal trunk and extremities. (b) Close observation of the skin lesions highlights a combination of maculopapular rash, vesicles filled with a serous exudate, and erythematous ulcers. These eruptions were accompanied by tenderness.

with dermatologists, a diagnosis of atypical generalized zoster was made; the patient immediately underwent highdose acyclovir therapy (1000 mg/day, for the duration of 1 week), resulting in rapid resolution. Since the clinical and laboratory findings were suggestive of a concomitant bacterial and/or fungal infection, cefepime (4 g/day) and micafungin (100 mg/day) were administered empirically for a week.

The electrolyte imbalance continued to deteriorate the following day despite appropriate infusion therapy; we suspected adrenal insufficiency and performed a comprehensive endocrinological workup (Table 1), which revealed panhypopituitarism. We followed with a head magnetic resonance imaging (MRI) scan on the second day of hospitalization, which identified an inhomogeneous pituitary gland enlargement and disappearance of the T1 hyperintense signal which is normally observed in the posterior pituitary lobe of healthy people (Figure 2(a)), accompanied by sphenoid sinus mucosa thickening (Figure 2(b)). The pituitary lesion had gadolinium ring enhancement, high-intensity signals on diffusionweighted imaging (DWI), and low-intensity signals on apparent diffusion coefficient (ADC) imaging, characteristic findings for an abscess of the pituitary gland (Figure 2(c)-(f)).^{4,12} Cefepime was continued for 1 week before the decision to switch to ceftriaxone was taken (due to better CNS penetration). Then, ceftriaxone (2 g/day) was administered for a total of 3 weeks, with improvement of the lesions on follow-up MRI scans (Figure 3(a) and (b)). Under hydrocortisone and levothyroxine replacement, plasma electrolytes normalized. The patient subsequently developed polyuria (3-8 L/day) with polydipsia; desmopressin stimulation test and hypotonic salt solution test confirmed the diagnosis of

On admission	Result	Unit	Normal range	Combined pituitary function test	Baseline	15 min	30 min	60 min	90 min	120 min
Adrenocorticotrophic hormone (ACTH)	<1.0	pg/mL	7.2–63.3	ACTH (pg/mL)	9.4	17.8	17.3	15.5	10	7.2
Cortisol	1.4	mg/dL	5-15	Cortisol (mg/dL)	2.6	2.5	3	3.5	2.8	2.2
Thyrotropic-stimulating hormone (TSH)	0.018	mIU/mL	0.5–5.0	TSH (mIU/mL)	1.8	-	2.1	2.2	2.3	2.3
Free T3	1.71	pg/mL	2.33-4.00	LH (mIU/mL)	1.6	-	2.8	3	2.9	2.6
Free T4	0.607	ng/mL	0.88-1.62	FSH (mIU/mL)	1.8	_	2.8	3	2.9	2.6
Luteinizing hormone (LH)	<1.0	mIU/mL	1.7–8.6	PRL (ng/mL)	1.2	-	1.8	1.7	1.4	1.4
Follicle-stimulating hormone (FSH)	<1.0	mIU/mL	1.5–12.4	Growth hormone (GH)-stimulation test	Baseline	15 min	30 min	45 min	60 min	
Prolactin (PRL)	<1.0	ng/mL	3.6-16.3	GH (ng/mL)	0.14	1.24	1.26	0.91	0.6	
Somatomedin C (IGF-1)	12	ng/mL	41–272	Hypertonic saline test	Baseline	30 min	60 min	90 min	120 min	
Testosterone	<2.5	ng/dL	3 -87	Serum osmolality (mOsm/kg)	284	294	299	303	305	
Free testosterone	<0.4	pg/mL	7.7–21.6	ADH (pg/mL)	<0.8	<0.8	<0.8	<0.8	0.9	
Antidiuretic hormone (ADH)	<1.2	pg/mL	<3.6							

Table I. Endocrinological examination values.



Figure 2. (a) Axial T1-weighted image showing a hypointense lesion in the pituitary gland surrounded by a slightly hyperintense rim (arrowhead). (b) Axial T2-weighted image depicting sphenoid sinus mucosa thickening and a hyperintense lesion in the mastoid cell (solid arrow). (c) Sagittal and (d) coronal T1-weighted gadolinium-enhanced images showing a ring-enhancing intrasellar lesion with fluid intensity in its inner part. The pituitary stalk is also enhanced. (e) Diffusion-weighted imaging (DWI) indicating a high-intensity mass in the pituitary fossa. (f) The intrasellar lesion demonstrated restricted diffusion on the apparent diffusion coefficient (ADC) image (open arrow).

masked diabetes insipidus, which was treated with desmopressin acetate.

Because the patient's neurological involvement was minor and empirical medication had improved his clinical status effectively, surgical drainage was not performed. Despite an intensive investigation regarding the etiology of the pituitary abscess, including blood and CSF cultures, immunological tests for diagnosis of fungal infections (beta-D-glucan assay), and polymerase chain reaction for mycobacteria and VZV performed from both CSF and blood samples, we could not determine the causative agent.



Figure 3. Post-treatment MRIs. (a) Sagittal and (b) coronal enhanced TI-weighted scans showing disappearance of cystic lesion and improvement of pituitary gland and stalk enhancement.

At 2 years of follow-up, the patient is doing well but continues to require hormone replacement therapy.

Discussion

A pituitary abscess in an HIV-1-infected patient is a very rare, but potentially life-threatening disease. To the extent of our knowledge, only one other case of pituitary abscess in an HIV-1-infected patient has been reported so far.11 Notably in our case, the pituitary abscess developed while the CD4 T-cell count was within normal ranges. Although we could not detect the causative organism, hematogenous spread from the bacterial paranasal sinusitis was suspected. Our otolaryngologists and neurosurgeons did not suspect a direct spread from the adjacent sphenoid sinus because the sinusitis was mild and there were no osteolytic lesions present at examinations; however, this possibility could not be fully excluded. Another possible etiology is hematogenous spread from bacterial superinfection of VZV lesions with Staphylococcus aureus or group A beta hemolytic Streptococcus. In addition to the absence of literature depicting abscesses due to VZV, acute VZV infection of the CNS would present as meningomyeloradiculitis and would be accompanied by a high viral load in the CSF,13 so we concluded that, for our patient, the probability of VZV causing the pituitary abscess was low. The present case depicts a rare occurrence of a bacterial infection localized in the pituitary gland simultaneous with a generalized herpes zoster viral infection, drawing attention to the possibility of polymicrobial infections in HIV-positive patients. The high rates of bacterial infections in HIV-1-infected people may be the consequence of multiple factors, including qualitative B-cell dysfunction, and impaired macrophage function or numbers.

Interestingly, cases of pituitary abscess presenting with acute severe infectious manifestations are rare.^{3,7} The case presented here follows this pattern regarding the scarce infectious and neurological symptoms to suggest such a pathology. A complete anamnesis revealed the presence of

risk factors (paranasal sinusitis, immunocompromised status) for developing a pituitary abscess, the only hint toward the possible etiology of the infectious process. Even in the absence of any sign of infection, a pituitary abscess should be considered in patients who present with longstanding headache, endocrine dysfunction with or without visual disturbances. Awareness of pituitary abscess as a differential diagnosis in HIV-infected patients with these symptoms can be critical, especially in the case of superinfection. Imaging findings and results of hormonal tests can help rapidly identify the cause of pituitary deficiency.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Informed consent

Written informed consent was obtained from the patient for their anonymized information to be published in this article.

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