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Case Report

Massive bilateral paraclinoidal subdural empyema and parenchymal temporopolar abscess with anatomical infection pathway in a chronic inhaling cocaine-addicted patient: A case report and literature review

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ABSTRACT

Background: Focal suppurative bacterial infections of the central nervous system (CNS), such as subdural empyemas and brain abscesses, can occur when bacteria enter the CNS through sinus fractures, head injuries, surgical treatment, or hematogenous spreading. Chronic cocaine inhalation abuse has been linked to intracranial focal suppurative bacterial infections, which can affect neural and meningeal structures.

Case Description: We present the case of a patient who developed a cocaine-induced midline destructive lesion, a vast bilateral paraclinoidal subdural empyema, and intracerebral right temporopolar abscess due to cocaine inhalation abuse. The infection disseminated from the nasal and paranasal cavities to the intracranial compartment, highlighting a unique anatomical pathway.

Conclusion: The treatment involved an endoscopic endonasal approach, followed by a right frontal-temporal approach to obtain tissue samples for bacterial analysis and surgical debridement of the suppurative process. Targeted antibiotic therapy helped restore the patient's neurological status.

Keywords: Cocaine addiction, Cocaine-induced midline destructive lesions, Subdural empyema, Temporopolar abscess

INTRODUCTION

Hematogenous spread, traumatic head injuries with skull and scalp discontinuity, and surgery are the primary causes of focal suppurative bacterial infections of the central nervous system (CNS).[8,15] Diffusion of the suppurative process may cause various clinical manifestations, including fever, headache, and focal neurological deficits.[6] Typically, the treatment of these lesions relies on multidisciplinary management between neurosurgeons, infectiologists, and microbiology laboratories. Primarily, treatment is highly dependent on the spreading pattern of the suppurative process toward the intracranial compartment and the number, localization, and size of the lesions.[1] The main

objective of surgery is to achieve brain decompression in cases of intracranial hypertension. However, patients with several abscess localizations are often immunocompromised due to chemotherapy, pre- and post-organ transplant treatments, acquired conditions, or narcotic drug abuse.[11] We report the case of a patient with bilateral paraclinoidal subdural empyema and right intraparenchymal temporopolar abscess due to a 10-year-long history of cocaine inhalation abuse. There are many studies on cocaine-induced vasoconstriction and its erosive action on the hard palate and nasal and paranasal cavities, but there are few reports on its organic damage to the structures of the CNS.[5] Moreover, magnetic resonance imaging (MRI) helped describe the peculiar anatomic pathway of the phlogistic process from the nasal and paranasal cavities to the intracranial compartment. We performed the surgical approach in two steps: the endoscopic endonasal route and the transcranial right frontal-temporal route. Microbiological culture allowed us to identify *Streptococcus anaerobium* in the intraoperative samples and eradicate the intracranial infectious process, obtaining a complete resolution of clinical and neuroradiological anomalies. To the best of our knowledge, this is the first report of intracranial lesions related to long-term cocaine inhalation abuse.

CASE REPORT

A 63-year-old male with a 10-year history of cocaine abuse presented with hyperpyrexia, seizures, and left hemiparesis. On admission, blood tests showed significant neutrophilic leukocytosis (28×10^3) and high C-reactive protein levels (346 mg/L). Once vital functions were stabilized and seizures were arrested, he underwent contrast-enhanced computed tomography (CT) of the head, which showed a right intra-axial temporopolar lesion (35 × 33 mm) with non-homogeneous contrast enhancement associated with digitiform perilesional edema and another large extra-axial bilateral frontal-basal lesion, which also presented non-homogeneous contrast enhancement. Moreover, extensive remodeling of the nasal and paranasal cavities, recognizable as cocaine-induced midline destructive lesions (CIMDL), and a hyperdense collection in the posterior aspect of the right maxillary sinus, spreading beyond the sphenopalatine foramen and pterygopalatine fossa through the orbital cavity and intracranial compartment were detected [Figure 1]. Subsequent contrast-enhanced MRI of the head confirmed the presence of a round lesion on the right temporal pole, with central necrosis and peripheral ring

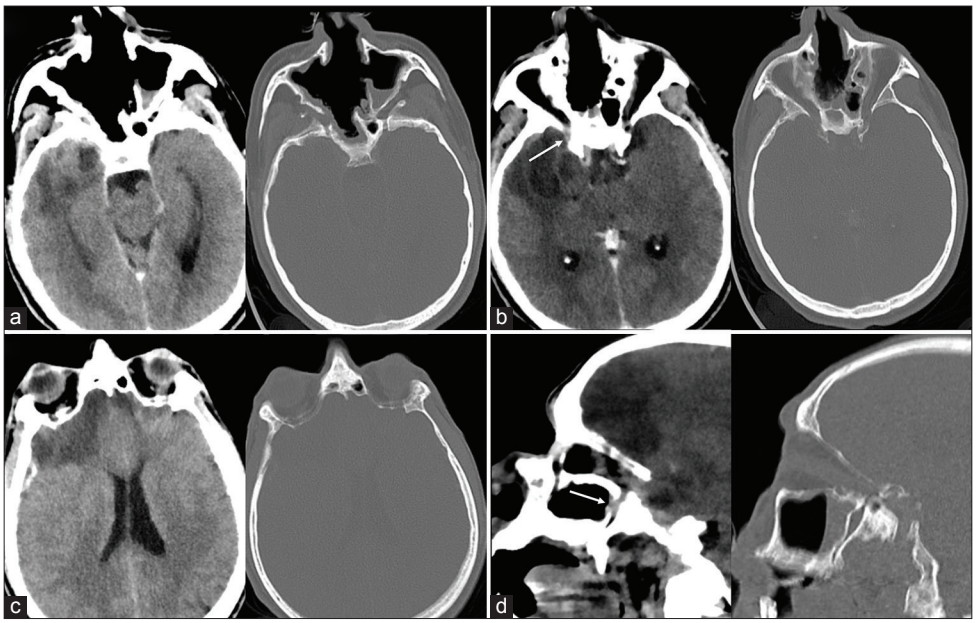

Figure 1: Preoperative head contrast-enhanced computed tomography scan ("Parenchyma and soft structures" window, left; "Bone" window, right). (a) Axial sequence: There is evidence of extensive remodeling of the nasal and paranasal cavities, configuring the cocaine-induced midline destructive lesions setting, consisting of a whole nasal cavity, bilaterally fused with ethmoidal cells and maxillary sinuses. (b) Axial sequence: A right intraparenchymal temporopolar abscess (35 × 33 mm), with central hypodensity and peripheral contrast enhancement and massive perilesional edema; at the level of the posterior aspect of the orbital cavity, there appeared a contrast-enhancing collection, apparently spreading of the infective process (white arrow). (c) Axial sequence: There is a vast bifrontal hypodense extra-axial lesion with subdural empyhematous collection. (d) Sagittal view: A contrast-enhancing collection within the pterygopalatine fossa extends towards the posterior aspect of the orbital cavity and within the intracranial compartment, describing the presumed dissemination pathway of the infective process (white arrow).

contrast enhancement [Figure 2]. Spectroscopy sequences showed a choline spike, altered choline/creatine ratio, and elevated N-acetyl-aspartate and lactate levels. These findings were suggestive of the temporopolar abscess.

Furthermore, a vast subdural empyema was detected in the bilateral frontal-basal and right paraclinoidal regions. The images also showed an infective process pathway through the intracranial compartment and right orbital cavity. There were no appreciable bony defects in the middle basicranium or clinically clear rhinoliquorrhea. The hyperintensity of the right pterygopalatine fossa and posterior aspect of the right orbital cavity on MRI suggested direct spreading of the suppuration. Indeed, the hypothesized dissemination pathway of the inflammatory process started from the maxillary sinus, proceeding through the sphenopalatine foramen toward the pterygopalatine fossa, and then upward through the inferior orbital fissure toward the posterior aspect of the orbital cavity and, lastly, spread posteriorly, and superiorly, through the superior orbital fissure, toward the middle cranial fossa [Figure 2]. Blood cultures showed no bacterial infection in the bloodstream, consequentially osmotic and empiric antibiotic therapy was administered. Endoscopic endonasal exploration was performed, which confirmed almost total erosion of the nasal septum and turbinates, destruction of medial walls of maxillary sinuses, and absence of ethmoidal cells; thus, during the procedure, nasal cavities were cleaned with abundant and repeated H_2O_2 and antibiotic washings. Osseous erosion toward the middle cranial fossa was not observed. Two days later, a second surgical intervention was performed using the frontal-temporal approach. Yellowish liquid from the underlying subdural compartment was detected above the dura mater and collected for microbiological and cultural examination. As a plausible consequence of this inflammatory reaction, the dura mater appeared taut, thick, and yellowish. The right frontal basal side of the outer membrane of the subdural empyema was opened, revealing an organized purulent collection. The anterior and basal portions of the lesion were removed in fragments. They presented parenchymatous consistency and yellowish color and were tenaciously adherent to the underlying tissues. Therefore, minimal temporal corticectomy was performed, and the abscess capsule content was aspirated [Figure 3]. Cultural examination revealed the presence of *S. anaerobium* susceptible to meropenem and levofloxacin. The postoperative course was regular, and the patient was free from further complications. A contrast-enhanced postoperative MRI scan demonstrated resolution of the bilateral subdural empyhematous collection and reduction in the diameter of the right temporopolar abscess. Targeted therapy reduced the contrast-enhanced lesion in the right temporal pole and remaining phlogistic tissue within the pterygopalatine and orbital cavities. Regression of radiological signs and clinical symptoms related to cerebritis in the temporal and right frontal areas has been previously reported.

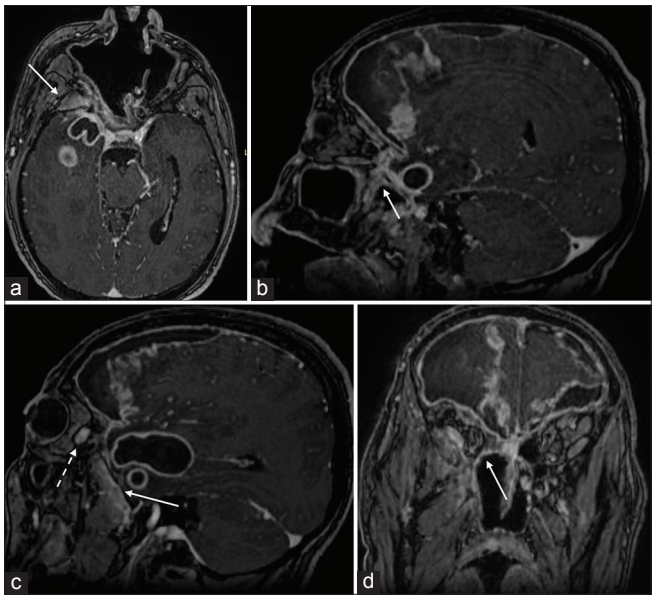

Figure 2: Preoperative post contrast brain magnetic resonance imaging T1-weighted sequences: (a) Axial sequence: A significant contrast enhancement of the right pterygopalatine fossa (white arrow) in the context of the thickened contrasted right maxillary mucosa and the suppurative process extending beyond the right temporal pole. (b) Sagittal sequence, paramedian right: There is a wide subdural empyema in the frontal lobe, with central hyperintensity and peripheral contrast enhancing hyperintensity. It shows the spreading pathway of the infection through the rotundum foramen, which results in an enlarged and hyperintense (white arrow). (c) Sagittal sequence, mid pupillary right: there is an intra-axial temporopolar abscess at its largest diameter in the sagittal plane (40 × 20 mm) and the right side of the bilateral subdural empyema at the frontal lobe. Detail of the hyperintense pterygopalatine fossa in sagittal view (white arrow) and of a hyperintense collection within the posterior aspect of the right orbital cavity (dotted white arrow). (d) Coronal sequence: It shows the subdural empyema at its maximum extension on the coronal plane, characterized by strong peripheral hyperintensity, most pronunciation at the right frontal lobe, and the orbital spreading of the contrast-enhancing collection (white line).

Blood tests on the 1st postoperative day showed a considerable decrease in C-reactive protein blood levels (49.84 mg/L) and white blood cell count (6.81×10^3). Postoperative head CT showed a sharp volume reduction of the extra-axial empyema collections and intra-axial abscess associated with regression of the central necrotic part and decreased intralesional septation. The patient's neurological status progressively improved. Targeted antibiotic therapy successfully resolved the intraparenchymal abscess. The patient was dismissed due to an intact neurological status and an indication to follow targeted antibiotic treatment for the following 6 weeks.

The 1-month postoperative MRI confirmed that the inflammation resolved in the pterygopalatine fossa and orbital cavity and showed a meaningful reduction in diameter on the axial plane of the temporopolar abscess (1.23 × 0.53 cm). Moreover, the subdural empyema located bilaterally in frontal-basal areas showed almost complete resolution, as well as infection of the right pterygopalatine fossa [Figure 4].

DISCUSSION

Focal suppurative intracranial collections may cause various neurological conditions. A history of head trauma, prior infective disease, or immunodepletion mainly suggests the correct diagnosis.[14] The patient reported epileptic seizures, hyperpyrexia, and hemiparesis as neurological signs. Seizures have already been described as presenting signs in patients with intraparenchymal abscesses.[3] It has been demonstrated that mortality is approximately 33% when hyperthermia develops along with seizures.[9] Thus, prompt and early diagnosis is critical for appropriate patient management. Moreover, hyperpyrexia and seizures lead to poor prognosis, mainly when they occur together.[2] Seizures may occur after large and continuous cocaine abuse as well as in individuals without a seizure focus. Furthermore, cocaine may exacerbate pre-existing seizure disorders.[13] The challenging management of the patient relied primarily on his history of anamnesis. Anaerobic and microaerophilic streptococci are the most commonly identified pathogens.[4] MRI scans were thoroughly studied in the reported case, assessing the peculiar anatomic corridor through the right pterygopalatine and orbital cavity toward the intracranial paraclinoidal compartment. The devastating effects of cocaine on the splanchnocranium may lead to the continuity between the eroded nasal cavity and middle cranial fossa. However, surgical exploration did not reveal a pathological corridor on the floor of the middle cranial fossa. Remarkably, the devastating vasoconstriction and vasospasm caused by cocaine inhalation may physically affect the inner skull structures even without erosion of the skull bones. To the best of our knowledge, this is not the first report of intracranial involvement resulting from chronic cocaine abuse. Effectively, there is strong evidence of cocaine inhalation effects on the nasal and paranasal cavities and hard palate, but there are limited reports on its organic damage to the CNS.[5] Many studies have described patients with subarachnoid, intraventricular, and intraparenchymal bleeding associated with intravenous cocaine use.[7] Anecdotal cases of cerebral abscesses have been described as a direct consequence of chronic cocaine abuse.[12] Nonetheless, our case showed an impressive subversion of the anatomy in the splanchnocranium and neurocranium as a direct consequence of chronic cocaine abuse. Moreover, the entire nasal cavity anatomy was subverted, with destruction of the nasal septum, bilateral erosion of ethmoidal cells, and medial walls of the maxillary sinuses, a typical CIMDL picture, one of the characteristic manifestations of chronic snorting cocaine abuse. This peculiar setting is caused by vasoconstriction, chemical irritation, ischemia, and collapse of the nasal septum, middle and superior turbinates, lateral wall of the nose, and hard palate, leading to facial pain, dysphagia, and nasal reflux.[10] Recently, a CIMDL was described together with an intracranial space-occupying lesion in a patient with a history of inhaled cocaine abuse, suggesting that the ischemizing, erosive, and destructive action of cocaine on structures of the splanchnocranium can also propagate to the neurocranium.[5] An interesting abscess localized in the pituitary fossa was recently reported.[16] In our case, the cocaine necrotizing and erosive action on the structures of the nasal cavities was massive. In addition, the right maxillary sinusitis spreads posteriorly to the pterygopalatine fossa, upward toward the posterior aspect of the orbital cavity, and finally through the superior orbital fissure toward the right paraclinoidal space. In recent years, in contrast to previously

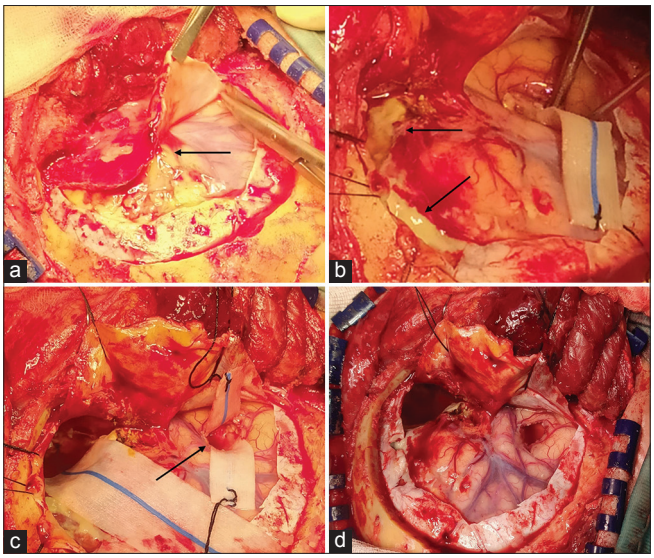

Figure 3: Right frontal-temporal approach: (a) After performing a right frontal-temporal craniotomy, a yellowish fluid collection was sampled for cultural examination. The dura had a thick, reactive, and yellowish aspect, and a solid, parenchymatous lesion was appreciable with a net cleavage plan between empyema and healthy cerebral cortex (black arrow). (b) The empyema had a parenchymatous consistency and was also yellowish (black arrow). A significant phlogistic perilesional reaction was clear. The lesion was gradually and piecemeal evacuated. (c) After evacuating the empyema, a small right temporopolar corticectomy was performed (black arrow), and the content of the abscess capsule was aspirated and sent to the laboratory for cultural analysis. (d) After the piecemeal evacuation of the subdural empyema and aspiration of the temporopolar abscess, nervous structures, and Sylvian vein appeared well decompressed and pulsating. The reactive dura mater was replaced with a synthetic patch.

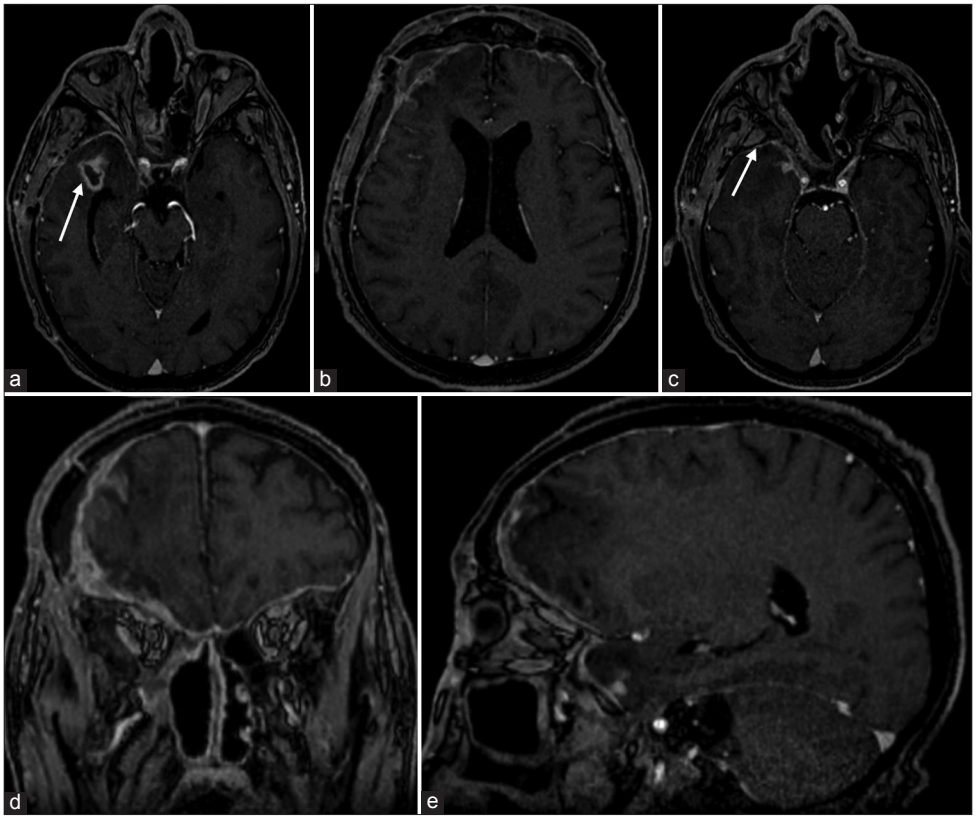

Figure 4: One-month-postoperative post contrast brain magnetic resonance imaging T1-weighted sequences: (a) Axial sequence: 1-month-postoperative imaging showed a critical reduction in the diameter of the temporopolar abscess, in its largest diameter on the axial plane (1.23 × 0.53 cm), due to surgery and subsequent surgery antibiotic therapy (white arrow). (b) Axial sequence: The subdural lesion in the bilateral frontal-basal area showed almost complete resolution in its largest diameter on the axial plane. There was a dural thickening due to the inflammatory reaction in the surgical bed on the right frontal-temporal side. (c) Axial sequence: Antibiotic therapy also successfully resolved the infection among the right pterygopalatine fossa (white arrow). (d) Coronal sequence: The subdural empyema was resolved in the right frontal-temporal dural thickening due to the surgical approach and fibrotic reaction. (e) Sagittal sequence, paramedian right: Net volumetric reduction of infective lesions on the intracranial compartment and within the pterygopalatine fossa and orbital cavity.

described cases, our patient developed both splanchnocranial and neurocranial suppurative involvement exclusively through anatomic pathways.

Cocaine has a devastating effect on the CNS, both physically and neuropsychologically. Its necrotizing action may also initiate suppuration of the necrotic brain parenchyma. Our case's unity consisted of a peculiar anatomical spreading pathway of the infection. Moreover, this is the first case of subdural empyema and intraparenchymal abscess, both attributable to multiyear cocaine snorting abuse.

CONCLUSION

Inhaling cocaine abuse has devastating organic effects on CNS structures. Our case showed a peculiar spreading pathway of the suppurative process through the splanchnocranial cavities toward the orbital cavity and intracranial compartment, resulting in subdural empyema and an intraparenchymal abscess. Finally, it showed how the necrotizing action of cocaine might cause organic damage to the CNS and physically subvert the splanchnocranial and intracranial compartments.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Busl KM, Bleck TP. Bacterial infections of the central nervous system. Curr Infect Dis Rep 2013;15:612-30.
2. Callaway CW, Clark RF. Hyperthermia in psychostimulant overdose. Ann Emerg Med 1994;24:68-76.
3. Esplin N, Stelzer JW, All S, Kumar S, Ghaffar E, Ali S. A case of *Streptococcus anginosus* brain abscess caused by contiguous spread from sinusitis in an immunocompetent patient. Cureus 2017;9:e1745.
4. Fernández-de Thomas RJ, De Jesus O. Subdural empyema. Treasure Island, FL: StatPearls Publishing; 2020.
5. Garcia-Perez D, Ruiz-Ortiz M, Panero I, Eiriz C, Moreno LM, Garcia-Reyne A, *et al*. Snorting the brain away: Cerebral damage as an extension of cocaine-induced midline destructive lesions. J Neuropathol Exp Neurol 2020;79:1365-9.
6. LaPenna PA, Roos KL. Bacterial infections of the central nervous system. Semin Neurol 2019;39:334-42.
7. Levine SR, Brust JC, Futrell N, Ho KL, Blake D, Millikan CH, *et al*. Cerebrovascular complications of the use of the "crack" form of alkaloidal cocaine. N Engl J Med 1990;323:699-704.
8. Mariniello G, Corvino S, Corazzelli G, Maiuri F. Cervical epidural abscess complicated by a pharyngoesophageal perforation after anterior cervical spine surgery for subaxial spondylodiscitis. Surg Neurol Int 2023;14:102.
9. Marzuk PM, Tardiff K, Leon AC, Hirsch CS, Portera L, Iqbal MI, *et al*. Ambient temperature and mortality from unintentional cocaine overdose. JAMA 1998;279:1795-800.
10. Moreno-Artero E, Querol-Cisneros E, Rodriguez-Garijo N, Tomas-Velazquez A, Antonanzas J, Secundino F, *et al*. Mucocutaneous manifestations of cocaine abuse: A review. J Eur Acad Dermatol Venereol 2018;32:1420-6.
11. Pruitt AA. Central nervous system infections in immunocompromised patients. Curr Neurol Neurosci Rep 2021;21:37.
12. Rana M, Mady A, Alharthy A, Ramadan O, Hashim W, Ashmawi S, *et al*. Brain abscess in a drug abuser with history of cocaine sniffing. Eur Radiol 2014;Case 11786.
13. Sordo L, Indave BI, Degenhardt L, Barrio G, Kaye S, Ruiz-Perez I, *et al*. A systematic review of evidence on the association between cocaine use and seizures. Drug Alcohol Depend 2013;133:795-804.
14. Spennato P, De Paulis D, Bocchetti A, Michele Pipola A, Sica G, Galzio RJ. Spontaneous intracranial extradural haematoma associated with frontal sinusitis and orbital involvement. Neurol Sci 2012;33:435-9.
15. Suthar R, Sankhyan N. Bacterial infections of the central nervous system. Indian J ediatr 2019;86:60-9.
16. Zafar M, Vaughan S, Khuu B, Shrestha S, Porruvecchio E, Hadid A. Cocaine-induced pituitary and subdural brain abscesses and the treatment challenges. Cureus 2021;13:e20821.

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