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Case Report

Tubercular cerebellitis, identified through an expansive process: A case report ☆,☆☆

Kaoutar Maasaoui, MD, Nizar El Bouardi, MD, Amina Hamdaoui, MD, Amal Akammar, MD, Meryem Haloua, MD, Badreeddine Alami, MD, Moulay Youssef Alaoui Lamrani, MD, Meryem Boubbou, MD, Mustapha Maaroufi, MD*

Department of Radiology and Interventional Imaging, CHU Hassan II Fez, Sidi Mohammed Ben Abdellah University, Fez, Morocco

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ABSTRACT

Central nervous system (CNS) tuberculosis is a postprimary form of tuberculosis with high mortality and morbidity rates, even with early diagnosis and treatment. Focal tuberculous cerebritis is extremely rare, typically occurring in patients without AIDS, and often associated with tuberculous meningitis. In endemic regions, it should be a primary consideration when encountering cerebral anomalies suggestive of granulomatous conditions. Its meningeal pseudo-tumor form poses a significant diagnostic challenge.

We present the case of a 26-year-old man who arrived at the emergency room with cerebellar and pyramidal syndrome. Cerebral magnetic resonance imaging (MRI) revealed an expansive lesion in the right hemicerebellum with glove-finger edema and leptomeningeal thickening. Given the MRI findings and associated lung involvement, the diagnosis of tuberculosis was strongly suggested in our epidemiological context. Due to contraindications for lumbar puncture, high surgical risk, and strong clinical and radiological suspicion, antituberculous treatment was initiated in collaboration with neurologists and infectious disease specialists.

The clinical and radiologic manifestations of CNS tuberculosis can mimic other infectious and noninfectious neurological conditions, as seen in our patient. Thus, familiarity with the imaging presentations of CNS tuberculosis among infectious disease specialists and radiologists is crucial for prompt and accurate diagnosis.

Abbreviations: GDs, Granulomatous diseases; CNS, Central nervous system; MRI, cerebral magnetic resonance imaging; TAP CT, Thoraco-Abdomino-Pelvic CT scan; MT, Mycobacterium Tuberculosis; WHO, The World Health Organization.

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* Corresponding author.
E-mail address: kaoutar.maasaoui@usmba.ac.ma (K. Maasaoui).

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Introduction

Granulomatous diseases (GDs) are rare conditions that can be autoimmune, infectious, idiopathic, or hereditary in nature [3,4]. The imaging characteristics of GDs in the head often lack specificity, showing significant similarities with imaging findings associated with other conditions, such as malignancies. In an endemic country like ours, where the incidence of tuberculosis is estimated at 93 per 100,000 inhabitants, tuberculous cerebro-meningitis remains the primary diagnosis to consider when faced with a cerebral anomaly suggestive of a granulomatous condition.

Central nervous system (CNS) tuberculosis is the most devastating form of tuberculosis [5]. Approximately 5%-10% of all patients with tuberculosis and up to 20% of patients with AIDS-related tuberculosis have CNS involvement [6,7]. Its meningeal pseudo-tumor form represents a significant diagnostic challenge. We report a case of atypical clinical-radiological presentation of tuberculous cerebro-meningitis in a 26-year-old patient.

Clinical observation

A 26-year-old man from Morocco presented to the emergency room with cerebellar and pyramidal syndrome. His symptoms began 8 months prior with progressive heaviness in the right side of his body, balance and speech disturbances evolving in a context of fever, sweating, and a general decline in health, including a 9 kg weight loss. Six months before admission, he had experienced a loss of consciousness. The patient had no significant medical history or known tuberculosis exposure.

During the physical examination, muscle strength in the right upper limb was graded as 4 out of 5. The Romberg test showed instability in multiple directions, and the patient had difficulty walking in a straight line. Dysarthria was present, and the finger-to-nose test was impaired on the right side. The patient had a widened base of support, brisk deep tendon reflexes, and bilateral Babinski signs.

A standard biological workup, including a complete blood count (CBC), blood electrolytes, and C-reactive protein (CRP), was normal except for hypochromic microcytic iron-deficiency anemia. A tuberculosis workup showed that 3 sputum smears for acid-fast bacilli (AFB) were negative.

Cerebral magnetic resonance imaging (MRI), without and with injection, revealed an expansive lesion in the right hemicerebellum with glove-finger edema and leptomeningeal thickening, described as hypointense on T1 and T2, with nodular and heterogeneous contrast enhancement at the late stage and no bone reaction (Fig. 1A-D). Notable compression in the posterior fossa led to the obstruction of cerebrospinal fluid drainage pathways, particularly affecting the fourth ventricle, causing triventricular hydrocephalus with signs of transependymal resorption (Fig. 2). Spectroscopic analysis revealed a pseudotumoral spectrum characterized by elevated levels of creatine choline, free lipids, and lactate (Fig. 3A and B). The imaging characteristics suggested a tumor, but the presence of nodular leptomeningeal thickening, hypointense on T2-weighted imaging with nonrestrictive diffusion, indicated a granulomatous origin, specifically tuberculosis (Fig. 1B).

As part of an assessment of the extension and confirmation of the disease, the patient underwent a thoraco-abdomino-pelvic CT scan (TAP CT) which revealed pulmonary involvement in the form of pulmonary nodules and micro-modules with a peri-hilar infiltrate, associated with necrotic mediastinal lymph nodes (Fig. 4 A and B). In the abdominal area, calcified lymph nodes were found, likely related to tuberculous lymphadenitis sequelae (Fig. 5).

Due to the significant mass effect in the posterior cranial fossa and the associated risk of brain herniation, a lumbar puncture was not performed. Stereotactic and surgical biopsies posed high risks of hemorrhage and neurological sequelae. Therefore, antituberculous treatment was initiated in collaboration with neurologists and infectious disease specialists, based on strong clinical and radiological suspicion and the favorable epidemiological context.

Treatment included HRZE (isoniazid, rifampin, pyrazinamide, and ethambutol) and pyridoxine, along with dexamethasone (0.4 mg/kg/day). Clinical follow-ups at 1 week, 3 months, and 6 months showed significant neurological improvement with the persistence of a non-deficit pyramidal syndrome. Radiological follow-up has not yet been performed. Antituberculous therapy was planned to continue for 1 year.

The provided images illustrate various diagnostic findings in the presented case: The fluid-attenuated inversion recovery (FLAIR) axial image and coronal T2-weighted images (Fig. 1A and B) display hyperintense signal intensity in the right cerebellar hemisphere with associated mass effect. The axial T2 and postcontrast fat-suppressed T1-weighted axial images (Fig. 2C and D) show nodular leptomeningeal thickening, hypointense on the T2-weighted image, and exhibiting nodular and heterogeneous contrast enhancement. The axial FLAIR image (Fig. 2) reveals hydrocephalus characterized by enlarged ventricles and periventricular ooze, indicative of cerebrospinal fluid leakage into the surrounding brain tissue. MR spectroscopy (Fig. 3A and B) demonstrates an elevated choline peak, indicative of increased cellular turnover, along with an increased choline to NAA (N-acetylaspartate) ratio. Additionally, there is a significant increase in lipid and an inverted high lactate peak, suggesting anaerobic metabolism and cellular distress. The CT axial slices in the parenchymal window (Fig. 4A and B) show pulmonary nodules and micromodules in the lung parenchyma, and mediastinal lymphadenopathy with necrotic centers, consistent with tuberculous involvement. The CT axial slice through the abdominal region (Fig. 5) displays a calcified lymph node, likely representing a sequela of previous tuberculous lymphadenitis.

Discussion

Tuberculosis affecting the central nervous system (CNS) represents a severe complication of mycobacterium tuberculosis (MT) infection, posing significant public health challenges worldwide. Diagnosis is often difficult due to the diverse clinical manifestations, nonspecific imaging aspects, and the challenges associated with isolating the MT bacterium [8].

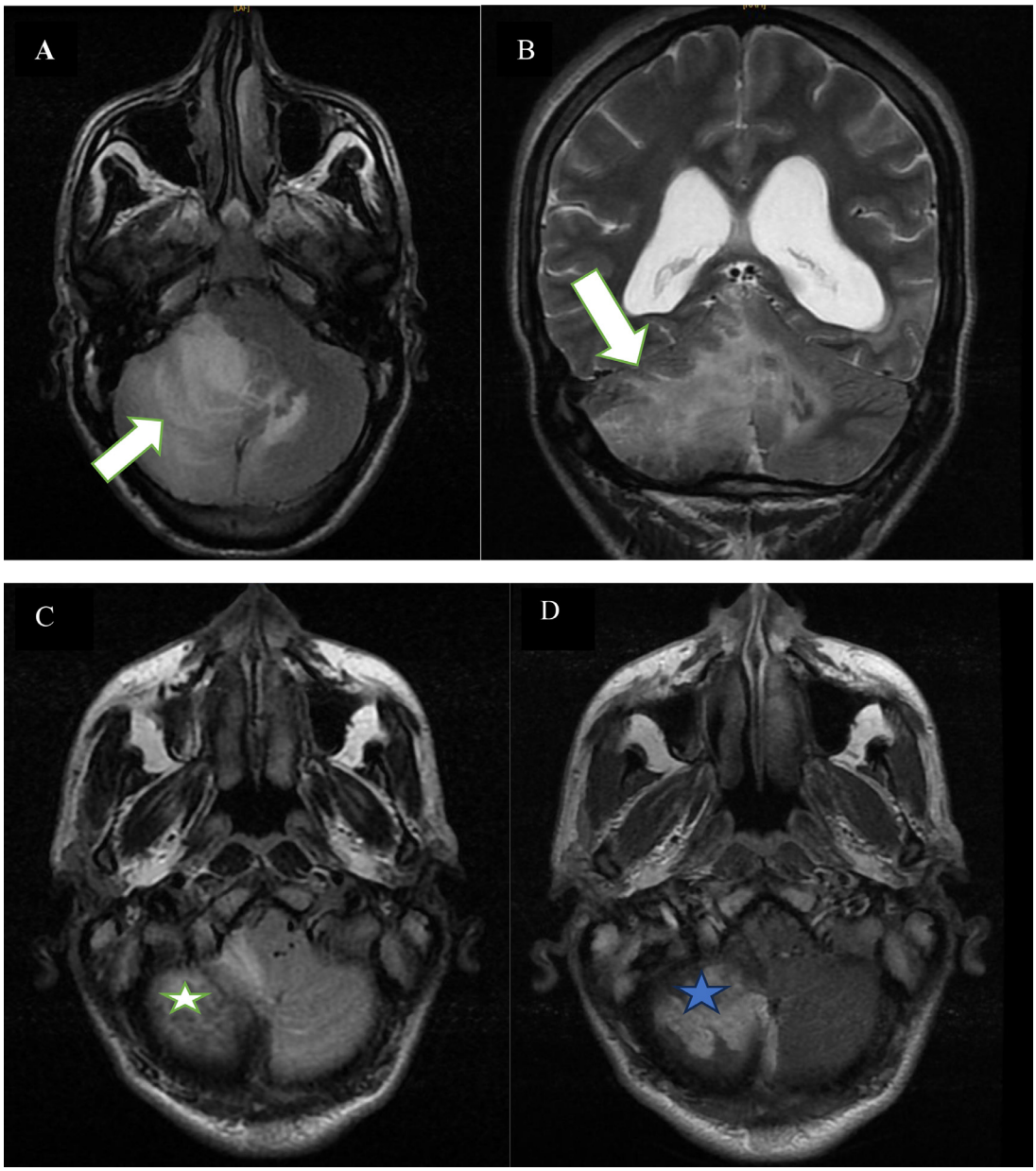

Fig. 1 – (A-D) Fluid-attenuated inversion recovery axial image and coronal T2-weighted showing hyperintense signal intensity involving right cerebellar hemisphere with mass effect (A, B) (arrow); Axial T2 and Postcontrast fat-suppressed T1-weighted axial image showing nodular leptomeningeal thickening, hypointense on T2-weighted (C) White star taking nodular and heterogeneous contrast (D) Blue star.

In our case, the clinical presentation and imaging appearance were initially suggestive of a brain tumor. However, the diagnosis of the pseudo-tumoral form of tuberculosis was made after thorough clinical and radiological investigations.

According to the World Health Organization (WHO), there were an estimated 10 million new cases of tuberculosis globally in 2018, resulting in 1.5 million deaths. In Morocco, the average tuberculosis incidence was reported at 89 cases per 100,000 inhabitants in 2015. Extrapulmonary tuberculosis forms account for 14% of cases, with only 1% affecting the neuromeningeal system [8,9].

Pathophysiology

Transmission of the tuberculosis bacillus primarily occurs through the airborne route. CNS tuberculosis is caused by the hematogenous spread of the mycobacterium from the lungs to the subpial and subependymal regions, brain parenchyma, and meninges, forming mycobacterium-rich foci called “Rich foci.” These foci rupture, triggering an intense cytokine-mediated inflammatory reaction. Exudates from this inflammatory reaction accumulate in the basal cisterns and meninges [1]. In our patient, the chest CT scan revealed pulmonary involvement. However, the absence of active or

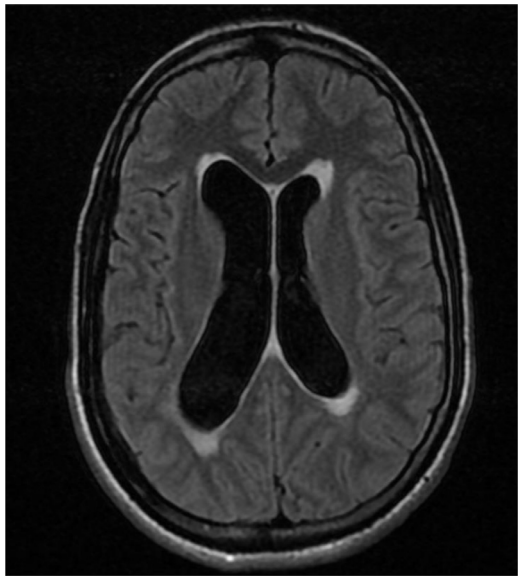

Fig. 2 – Axial fluid-attenuated inversion recovery image showing hydrocephalus and periventricular ooze.

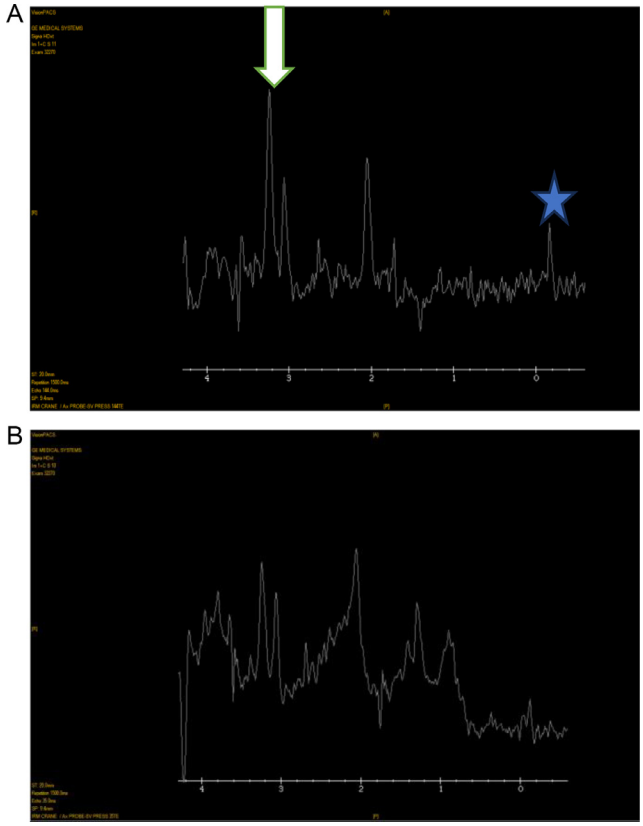

Fig. 3 – (A, B) MR spectroscopy shows elevated choline peak (arrow) with increased choline to NAA ratio, and significant increase in lipid (Star) with inverted high lactate.

sequelae pulmonary tuberculosis on imaging does not exclude the diagnosis of CNS tuberculosis.

Diagnosis

Neuromeningeal tuberculosis most commonly manifests initially as a meningitis syndrome. A focal deficit may appear during the course of the disease. Cerebellar syndrome, as in our case, is less common. Altered consciousness, ranging from confusion to deep coma, can also be observed, often associated with signs of tuberculous involvement, notably general deterioration and fever. Numerous studies have elucidated the various aspects of neuromeningeal tuberculosis. It can present in different forms, including meningitis, encephalopathy, miliary tuberculosis, tuberculous vasculitis, and, more rarely, an intracranial expansive process suggestive of tuberculoma or tuberculous abscess [10,11].

Our patient represents one of the rare cases of CNS tuberculosis described in the literature, featuring tuberculous

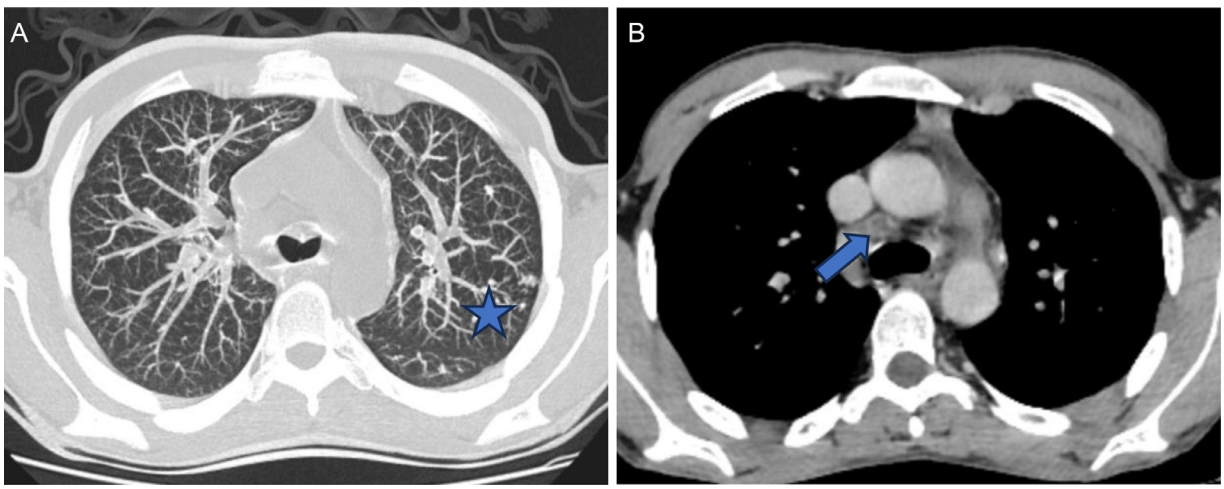

Fig. 4 – (A, B) CT axial slice in parenchymal window showing some pulmonary nodules and micromodules in the lung parenchyma(star), as well as mediastinal lymphadenopathy with necrotic center (arrow).

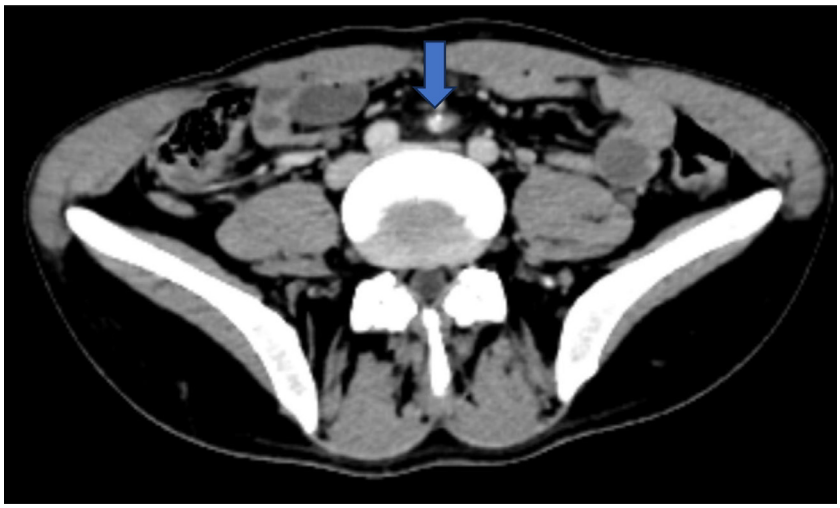

Fig. 5 – CT axial slice through the abdominal region showing a calcified lymph node formation, likely a sequela of tuberculous lymphadenitis (arrow).

cerebritis mimicking tumors with focal meningitis. Initially, conventional MRI findings suggested a tumor. However, the presence of enhanced nodular homolateral meningeal thickening indicated a granulomatous etiology, specifically tuberculous granulomas. The appearance of tuberculomas on conventional MRI varies depending on their stage of maturation. Tuberculomas with a solid caseating center, as observed in our patient, typically appear iso- to hypointense on both T1-weighted (T1W) and T2-weighted (T2W) images, with an iso- to hyperintense rim on T2W images and peripheral enhancement on contrast-enhanced studies. Various enhancement patterns may be observed, including complete ring enhancement, open rings, lobular patterns, or irregular enhancement [12–17,18].

CNS tuberculosis can mimic intracerebral tumors on MRI imaging. Without clinical and laboratory findings suggestive of tuberculosis, it can be challenging to distinguish CNS tuberculosis from tumors such as lymphoma and glioblastoma multiforme [18–21,13,22,23]. Functional imaging helps support the diagnosis of tuberculosis. Metabolic study by spectroscopy can provide clues towards a tubercular origin, characterized by an elevated Cho/Cr ratio, reduced NAA/Cr and NAA/Cho ratios, often accompanied by a prominent lipid peak. A similar spectrum may be observed in cases of neoplastic brain lesions but with a significantly increased Cho/Cr ratio, with or without the presence of lipid or lactate peaks. Perfusion imaging also aids in distinguishing ring-enhancing tuberculomas from metastases. Ring-enhancing or nodular tuberculomas typically exhibit slightly elevated mean rCBV values (2-3.3) [2]. In contrast, the wall of metastases demonstrates substantially higher mean rCBV values (5.43), with a suggested cut-off value of $\geq$3.745 to distinguish ring-enhancing metastases from tuberculomas [24,25].

Confirmation of the diagnosis requires histopathological evidence or a lumbar puncture to search for Koch's bacilli or specific DNA using the GeneXpert method. However, the association with pulmonary and nodal tuberculosis can be highly suggestive of the diagnosis and prompt the initiation of anti-tubercular treatment, especially in a favorable epidemiological context.

Treatment

The primary treatment modality for CNS tuberculosis is medical management. This should include measures to control cerebral edema and increased intracranial pressure (ICP), alongside anti-tubercular therapy and corticosteroids [26]. The current recommendations for treating CNS tuberculosis involve an initial regimen combining isoniazid, rifampicin, and pyrazinamide with ethambutol (or streptomycin or a fluoroquinolone) for 2-3 months, followed by consolidation treatment with isoniazid and rifampicin for up to 12 months. If necessary, the total treatment duration can be extended to 18 months [26].

For patients with drug-resistant tuberculosis, treatment should be guided by the treatment guidelines for drug resistance, selecting appropriate sensitive drugs [27]. In some cases, urgent surgical intervention may be required to alleviate symptoms caused by mass effect from a giant tuberculoma or in cases where there is a paradoxical response to anti-tubercular treatment [28].

Conclusion

The clinical and radiological manifestations of CNS tuberculosis can mimic other infectious and noninfectious neurological conditions, as observed in our patient. Therefore, familiarity with the imaging presentations of CNS tuberculosis, including multimodal imaging, is essential. Additionally, searching for more common tuberculosis infections, particularly pulmonary and nodal, through both biological and radiological

evaluations, is crucial for the prompt and accurate diagnosis of this condition.

Ethics approval and consent to participate

Not applicable.

Availability of data and materials

The data sets are generated on the data system of the CHU Hassan II of Fes, including the biological data and the interventional report.

Patient consent

Written informed consent was obtained from the patient, and legal guardian for publication of this case report and any accompanying images.

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