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Cerebral Toxoplasmosis in a Previously Fit Individual – Pitfalls in Management – A Case Report.

Norly Salleh[1, 2*], **Rusli Zaim**[3], **Siti Rodiah Udin**[1]

1. Department of Surgery, Hospital Segamat, KM 6, Jalan Genuang, 85000 Segamat, Johor, Malaysia.
2. Satellite Clinical Research Centre, Hospital Segamat, KM 6, Jalan Genuang, 85000 Segamat, Johor, Malaysia.
3. Department of Radiology, Hospital Segamat, KM 6, Jalan Genuang, 85000 Segamat, Johor, Malaysia.

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ABSTRACT

Most people with toxoplasmosis infection are asymptomatic. Cerebral toxoplasmosis in HIV patient represents poor prognostic determinant, but treatable if early treatment is initiated. A case of delayed diagnosis of cerebral toxoplasmosis is presented. A 27-year-old man presented with one-sided facial numbness and treated as Bell's Palsy. Later he developed slurred speech. Contrast enhanced CT brain showed left frontal temporal hypodense lesion, leptomeningeal enhancement and perilesional oedema. Steroid was started. Four days later, he presented with decreased responsiveness. Retroviral was detected and diagnosis changed to opportunistic brain infection. He died after 3 days. Toxoplasmosis IgG antibodies turned out positive. In diagnosing cerebral toxoplasmosis, clinical presentation and examination are as important as laboratory testing and radiological imaging.

Introduction

Toxoplasmosis is a disease that results from infection with the Toxoplasma gondii parasite. Infection usually occurs by eating undercooked contaminated meat, exposure from infected cat faeces, or mother-to-child transmission during pregnancy. Most people who become infected with toxoplasma gondii are asymptomatic [1]. Some may have mild flu-like symptoms and lymphadenopathy. However, severe toxoplasmosis may develop in children and immune-compromised individuals.

Cerebral toxoplasmosis was described in immunocompromised individuals in the early epidemic of AIDS [2]. It is fatal if left untreated.

*** Corresponding Author:**
Norly Salleh
Address: *Department of Surgery, Hospital Segamat, KM 6, Jalan Genuang, 85000 Segamat, Johor, Malaysia.*
E-mail: *norlysalleh@gmail.com*

In patients with HIV or AIDS, the occurrence of cerebral toxoplasmosis represents a poor prognostic determinant. Patients with cerebral toxoplasmosis may present with headache, hemiparesis, cranial nerve palsy, altered consciousness or seizures [3]. Diagnosis may be achieved by detection of anti-Toxoplasma IgG and IgM antibodies in blood serum. Cerebrospinal fluid analysis may also be done to detect toxoplasmosis-specific IgG and IgM antibodies or T. gondii DNA. However, in patients with mass effect in the brain, lumbar puncture may increase the risk of brain herniation.

Cerebral toxoplasmosis is treatable if early treatment is initiated. Treatment is recommended for at least 4 to 6 weeks beyond resolution of all clinical signs and symptoms, but may require up to 6 months or longer [4]. A combination of drugs is usually used. Sonneville in 2012 found the use of pyrimethamine-sulfadiazine was associated with improved survival [5]. Alternatives treatment are combination of pyrimethamine-clindamycin or trimethoprim/sulfamethoxazole. Steroids are sometimes used to treat brain oedema [6] but Arens in 2007 found that the use of steroids in these patients may lead to more deaths [7].

We present a case of delayed diagnosis of cerebral toxoplasmosis in a young man.

Case Presentation

A 27-year-old man who was previously fit and healthy presented to the Ear, Nose and Throat Department with complain of numbness on one side of his face. He was diagnosed to have Bell's palsy and was started on steroid. No imaging was done at this point for this patient and he was given an appointment a few months later to review his response to the steroid treatment.

A month later, he presented to the Emergency Department of a district hospital with headache, facial asymmetry and slurred speech of 1-week's duration. On examination, his Glasgow Coma Scale (GCS) was 15/15. Other than slurred speech and right facial drooping, his neurological examination was intact. An urgent plain computed tomography (CT) scan of the brain revealed a large ill-defined mass measuring 4.8 x 5.0 x 5.4 cm at the fronto-temporal lobe. There was surrounding white matter oedema with minimal mass effect into the body of the left lateral ventricle (Figure 1). He was admitted to the general surgical ward.

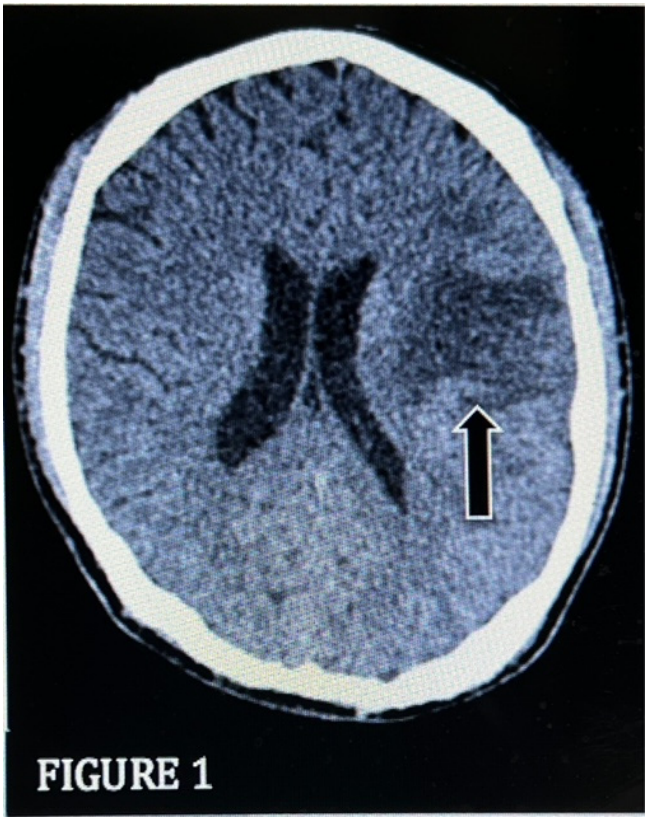

Fig. 1. Plain CT brain shows solitary ill-defined area of hypodensity (black arrow) involving the left fronto-parietal region causing minimal mass effect onto the adjacent of body lateral ventricle. No haemorrhagic component.

Upon discussion with the neurosurgical team in a tertiary hospital, he was started on high and tapering dose of steroid. Prophylactic anti-epileptic medication was also given. The next day, a contrast enhanced CT (CECT) brain was ordered. The scan showed a left frontal temporal area of hypodensity, predominantly white matter with evidence of leptomeningeal enhancement and surrounding perilesional cerebral oedema (Figure 2). This finding is most likely to represent cerebritis which could be inflammatory or infective in nature. However, primary brain tumour is another possibility. A follow up CT or Magnetic Resonance Imaging (MRI) was suggested. There was also a sign of early hydrocephalus. An MRI brain was arranged for him and the date was given in 12 days' time in another tertiary hospital. He was discharged home whilst waiting for his appointment.

Four days later, he presented with decrease responsiveness. According to his sister, he complained of neck stiffness prior to that and was not tolerating orally. On examination, his GCS was E4 (blank stare), V1 and M5. His pupils were equal and reactive to light. He was then subjected again to another CECT scan of the brain with IGS protocol upon request from the neurosurgical team. There was increasing area of previously seen left frontotemporal white matter hypodensity with locoregional mass effects, cerebral oedema, midline shift and uncal herniation. Furthermore, there is evolving hydrocephalus with new changes of acute subependymal CSF transudation with faint area of hyperdensity within the lesion, suggestive of haemorrhagic component (Figure 3). No significant changes or enhancement appreciated in contrasted CT brain study (Figure 4).

A rapid retroviral screening test was taken and it turned out to be positive. HIV Antigen test also turned out to be positive. As patient was unable to give any history at this time, one of his family member was called out to further enquire about patient's past but not much new information was learned as he used to live away from his family for a few years. Later on one junior doctor confirmed that during the first admission patient confided that he used to be promiscuous, and up to 2 years ago he had past history of intravenous

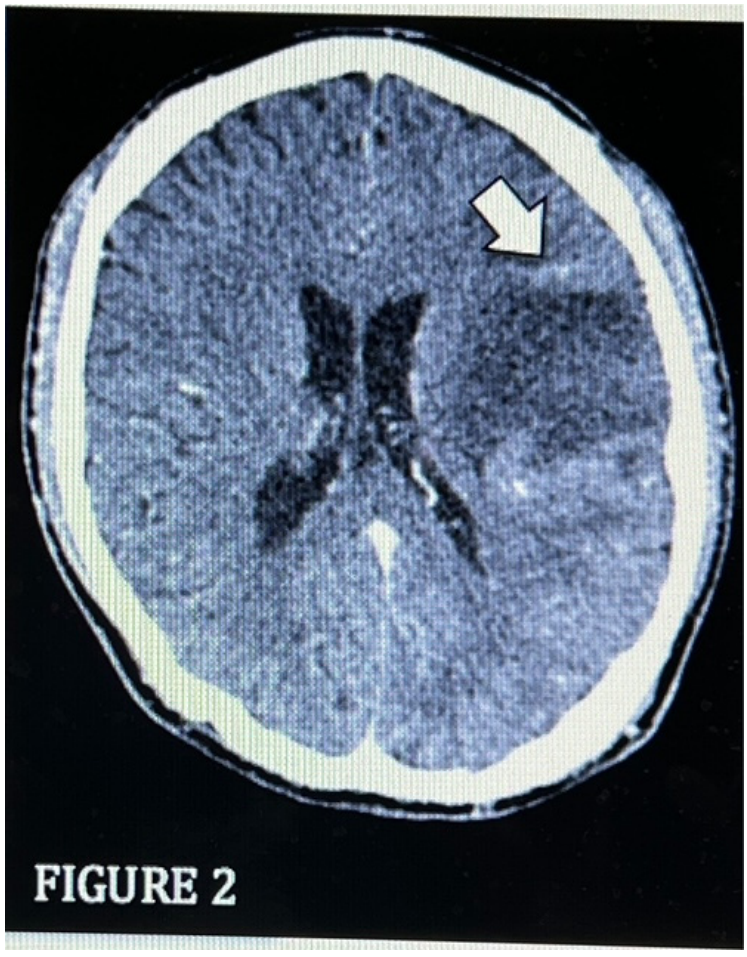

Fig. 2. CECT brain shows minimal regional leptomeningeal enhancement (white arrow). Otherwise no focal or concentric enhancing lesion seen.

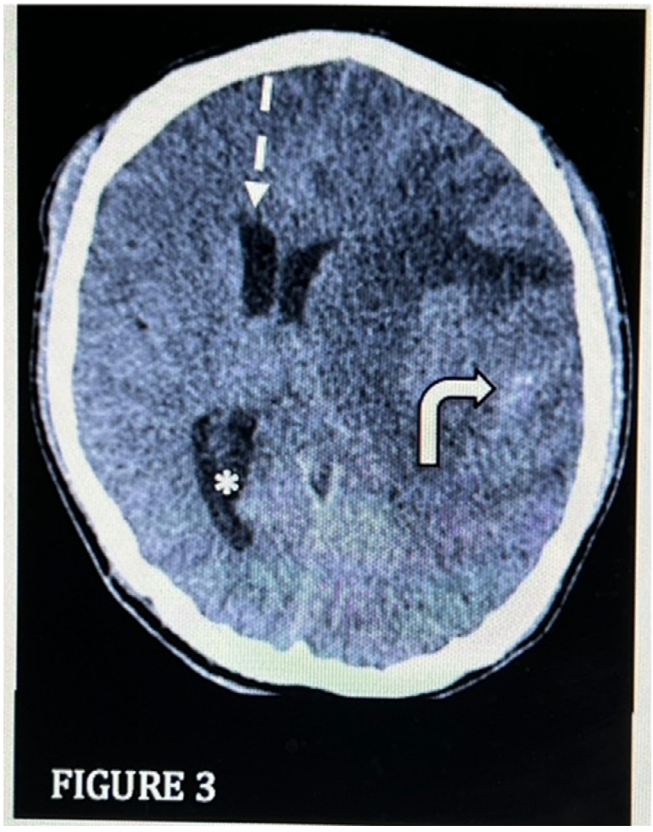

Fig. 3. Subsequent Plain CT brain (4 days later) shows presence of new intralesional haemorrhage at the left fronto-parietal region (curved arrow) with worsening area of hypodensity, causing mass effect onto the 3rd ventricle, with worsening hydrocephalus (*) and periventricular acute sub ependymal CSF seepage (dotted arrow).

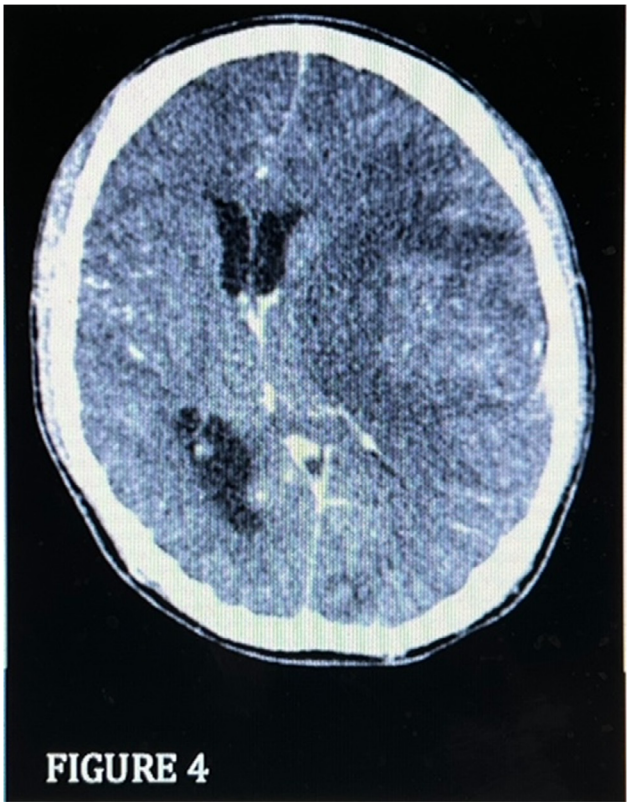

Fig. 4. CECT Brain shows no significant focal or solid mass enhancement

drug usage. None of these were documented in the patient's notes. Our diagnosis was changed to opportunistic brain infection, which includes cerebral toxoplasmosis or tuberculous infection. Empirical treatment of Toxoplasmosis was started. However, 2 days after admission, the patient deteriorated clinically and had to be resuscitated and intubated. He succumbed to death a day later. His toxoplasmosis IgG antibodies result was traced and it turned out to be positive.

Discussion

The first pitfall in managing this patient was when he first presented with one sided facial numbness and was diagnosed to have Bell's palsy. Bell's palsy is the result of a malfunction of cranial nerve VII (facial nerve) which is thought to be due to inflammation. It is a diagnosis of exclusion. Differential diagnosis are herpes zoster infection, stroke or brain tumour. An imaging at this point probably could have pointed us towards a correct diagnosis earlier. Some patients with cerebral toxoplasmosis do present with cranial nerve palsy [3].

The second pitfall is the lack of social history taking or rather lack of documentation. The fact that he was a promiscuous individual with history of intravenous drug abuse would have made us suspected that this is an immune compromised man and the likelihood of opportunistic infection is higher. Cerebral toxoplasmosis is a leading cause of the central nervous system disorders in acquired immune deficiency syndrome (AIDS).

Moreover, our patient had atypical radiological findings on his CT brain. In the majority of cases, radiologically the lesions may show some thin, smooth or faint enhancement, with eccentric nodularity; or no enhancement at all [8]. Our current case had a solitary single lesion rather than multiple lesions like other typical cerebral toxoplasmosis infection. In subsequent scan, this lesion also showed some intralesional haemorrhagic component within. According to Azotvseva et al., single foci in the brain are mostly found in patients with acute clinical symptoms [9]. This contributes to a severe form of toxoplasmosis and a possibly higher mortality [9]. A small study of HIV patient was conducted by Bhagavati et al. which shows 6 out of 11 cerebral toxoplasmosis patients presented with haemorrhagic lesions [10]. In their study, additional one patient was noted to developed haemorrhagic lesions after started on 2-week course of anti-toxoplasmosis treatment [10]. Unfortunately in our case, the previous social history of promiscuity and intravenous drug usage was unknown to the clinician and reporting radiologist. Hence, the differential diagnosis was more towards non-infective or tumoural cause, which includes haemorrhagic metastasis rather than haemorrhagic viral encephalitis. Hence, the patient was given early appointment date for MRI Brain to be done as out-patient.

In patients with equivocal or negative CT scans, a magnetic resonance imaging (MRI) should be obtained [11] as MRI has a sensitivity superior to that of CT scan for radiological diagnosis of cerebral toxoplasmosis [12]. On MRI the lesions are usually multiple with ring and/or nodular enhancement on contrast [9]. Lesions are typically found in the basal ganglia, thalamus and at the cortical/white matter border, with perifocal oedema and mass effect [9]. These lesions had high or mixed signal intensity on T2-weighted and FLAIR images, and low signal intensity on T1-weighted images [9]. Some abscesses may demonstrate the involvement of haemorrhage [9]. However, being a small district hospital, we do not have access to MRI service on site. The nearest is in another hospital which is almost 2 hours away. This probably contributes to the late diagnosis too.

Conclusion

Cerebral toxoplasmosis is diagnosed through a combination of diagnostic methods. Clinical presentation and examination is as important as laboratory testing and radiological imaging. The importance of good history taking in the management of patient could not be stressed enough. As radiological imaging may be variable, it should always be correlated with good clinical history. Despite dealing with solitary lesion, high level of suspicion of infective causes needed to be considered in order to prevent late diagnosis and treatment.

Ethical Approval

Ethical approval is not required at our institution to publish an anonymous case report.

Consent for Publication

Consent was not obtained from the patient as this is an anonymous case report.

Funding

None.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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