Case Report

Encephalitis Lethargica like Illness: Case Report and Literature Review

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Recent studies have revealed that encephalitis lethargica (EL) may not be related to influenza virus infection but more likely to be a post-infectious autoimmune disease. The diagnostic clinical criteria for EL like illness include subacute hypersomnolence and ophthalmoparesis followed by Parkinsonism, oculogyric crisis, neuropsychiatric disorders and central respiratory abnormality. Recently, Magnetic Resonance Imaging (MRI), which depicts hypersignal intensity on T2 weighted, and FLAIR images at midbrain, tegmentum, and basal ganglia, have been very helpful diagnostic tests in EL like illness. Nevertheless, EL like illness has never been reported in Thailand. A 17 year-old man presented with hypersomnolence one week before admission. Physical examination revealed drowsiness and ophthalmoparesis. MRI showed bilateral hypersignal intensity lesions on T2 weighted and FLAIR images at midbrain, basal ganglia and temporal lobes. CSF studies showed normal profiles. CSF-PCR for herpes simplex virus, varicella zoster virus, cytomegalovirus, Epstein-Barr virus, Pan-Enterovirus and Westnile virus were negative. CSF Dengue and Japanese encephalitis virus hemagglutination test were negative. He was treated with intravenous dexamethasone and immunoglobulin. Somnolence and ophthalmoparesis were improved. Two months later, he developed schizophreniform features and Parkinsonism. MRI revealed improvement of midbrain and basal ganglia lesions. CSF studies showed normal CSF profiles while oligoclonal bands were positive. Autoimmune profiles and serological tests for post-streptococcal infection as well as syphilis were negative. Thyroid function tests and serum ceruloplasmin were within normal limits. Levo-Dopa, clonazepam and sodium valproate had been prescribed and the clinical syndrome was gradually improved.

Keywords: *Encephalitis lethargica, von Economo's encephalitis, Magnetic Resonance Imaging, Oligoclonal bands*

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Encephalitis Lethargica (EL) was first described during the epidemic of influenza in 1917 by Constantin von Economo. The cardinal, clinical features in the acute phase consisted of sleep disturbance and ophthalmoparesis while parkinsonism, neuropsychiatric disorders, oculogyric crisis, or central respiratory irregularity were prominent in the post-encephalitic phase[1]. At that time, the pathogenesis was supposed to be related to influenza virus infection. After 1920, the incidence of EL patients had steadily declined and the world epidemic of influenza infection ended by 1930[1]. Thereafter sporadic cases of EL like patients have been reported. According to Medline search in the last decade (1995-2005), 40 patients have been reported in a small series and individual case reports. (1 case from Italy[2]; 3 cases from The United States of America[3-5]; 1 case from France[6]; 25 cases from England[7-11]; 6 cases from Austria[12]; 1 case from Singapore[13]; 1 case from Germany[14]; 1 case from Japan[15]; 1 case from Bulgaria[16]). No EL like patients have ever been reported in Thailand. Recent clinical, immunological and neuroimaging studies have documented that EL like illness is one of post-infectious neurological autoimmune diseases.

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Case Report

A 17 year-old man developed excessive daytime sleepiness and frequent yawning nine months before admission. He fell asleep while doing activities such as eating or standing. Seven days later, he was drowsy and inactive in daily activities. Nine days later, he developed ptosis and lateral squint on left eye. His past medical history was unremarkable. No history of previous infection or medication was reported. He did not have any familial history of Parkinsonism or neuropsychiatric disorders.

He was afebrile and general physical examination could not detect any abnormalities. On neurological examination, he was drowsy whereas, his orientation was intact. Bilateral ophthalmoparesis and ptosis were observed to be more prominent on the left. He had mild facial paresis of upper motor neuron type with pronator drifting on the left side.

Laboratory tests including complete blood count (CBC), blood urea nitrogen (BUN), serum creatinine (Cr), liver function tests and electrolytes were normal. Serum ANA, C3, CH50, AFP and B-HCG were within normal limits. Serum PCR for HIV-RNA was negative. Computed tomography (CT) scan of the brain with contrast enhancement appeared unremarkable. Magnetic Resonance Imaging (MRI) of the brain depicted bilateral symmetrical hypersignal intensity lesions on T2 weighted and FLAIR images at midbrain, hypothalamus, caudate nucleii, putamen, globus pallidus, mesial temporal lobe and pons (Fig. 1, 2). No abnormal enhancement of these lesions after gadolinium injection was observed. Cerebrospinal fluid (CSF) analysis demonstrated an open pressure of 130 mmH_2O. CSF was clear and acellular with glucose of 66 mg/dL (serum glucose 88 mg/dL), protein of 24 mg/dL and lactate of 1.3 mM/L (normal range 0.5-2.2 mM/L). CSF culture for bacterial and PCR studies for herpes simplex virus (HSV), varicella zoster virus (VZV), cytomegalovirus (CMV), Epstein-Barr virus (EBV), Pan-Enterovirus and Westnile virus were negative. CSF Dengue and Japanese encephalitis (JE) virus hemagglutination test were also negative.

During admission, he became drowsier. The suspected clinical diagnosis was viral encephalitis, therefore intravenous acyclovir 600 mg every 8 hours and dexamethasone 16 mg daily for 6 days were prescribed. The neurological status had not been improved, intravenous immunoglobulin 25gm was administrated daily for 4 days due to the possibility of post-infectious autoimmune process. MRI was repeated and showed more intensification of the same lesions without enhancement. Brain biopsy for definite diagnosis was planned but his relatives refused any surgical interventions. Therefore, he was discharged and followed at the outpatient department. One month later, his relatives reported that his basic activities such as eating, sitting, walking and speaking were gradually resumed.

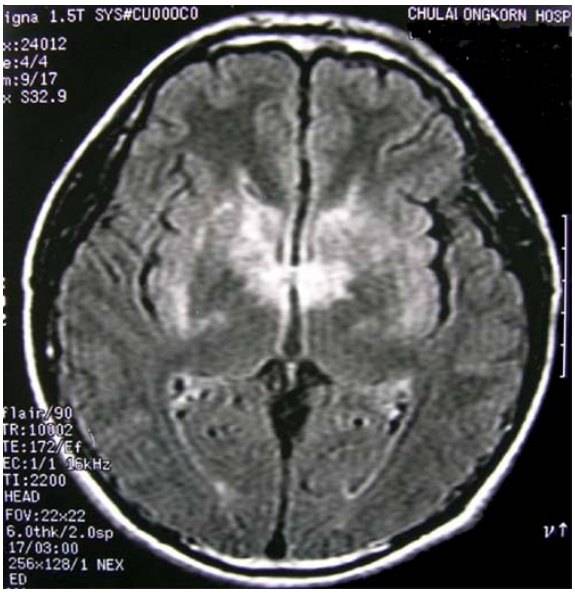

Fig. 1 FLAIR MRI shows bilateral symmetrical hypersignal intensity at basal ganglia, hypothalamus

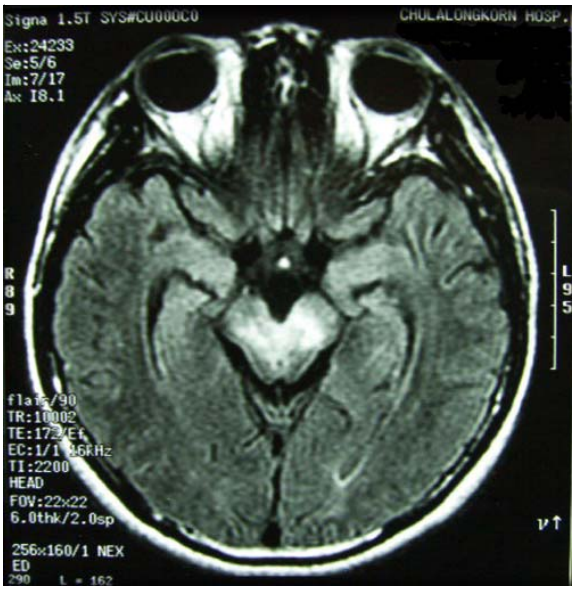

Fig. 2 FLAIR MRI shows bilateral symmetrical hypersignal intensity at midbrain and mesial temporal lobe

Three months after the onset, he could perform his basic activities independently. However, he had abnormal behaviour including increased oral activity such as chewing leaves and being talkative. He searched for smelly things like armpits and feces, collecting trash including bottle lids and plastic bags. Abnormal sexual behaviour such as frequent masturbation, flirting and embracing anyone, particularly young females had also been reported. Five months later, he started having head and upper extremities resting tremor. Before admission, the behavioural problems had improved while the abnormal movements kept persisting.

On neurological examination, he was fully conscious. Mild ptosis and minimal left medial rectus paresis were detected. Motor power appeared normal. No pyramidal tract signs were observed. He had mild generalized bradykinesia, masked face and severe resting tremor of the head and upper extremities. Grasping reflex was positive bilaterally. Bender-Gestalt test revealed motor incoordination, poor integration, closure difficulty and distortion of shape, which was compatible with organic brain syndrome. His abnormal behaviour was diagnosed as schizophrenicform features by a psychiatrist.

Laboratory tests included CBC, BUN, Cr, liver function tests and electrolytes were normal. CSF analysis demonstrated an open pressure of 130 mm. H_2O, clear and colourless CSF with lymphocyte 2 cells/mm[3], glucose of 67 mg/dL (serum glucose 73 mg/dL) and protein of 33 mg/dL were observed. Oligoclonal bands were detected in CSF while it was negative in the serum. Serum C3, CH50, thyroid function tests and ceruloplasmin were within normal limits. Serum ANA, anticardiolipin, VDRL and ASO antibody titer were negative. MRI of the brain was performed and demonstrated diminishing hypersignal intensity on T2 weighted and FLAIR images in the same previous areas. There was no abnormal enhancement after gadolinium injection.

EL like illness was diagnosed, based on clinical profiles, classical neuroimaging appearances and CSF studies. Levodopa/benserazide 100/25 mg and clonazepam 3 mg were prescribed daily for Parkinsonism. Sodium valproate 500 mg per day were prescribed for behavioural problems. During the admission, his behavioural problems subsided whereas the Parkinsonism was partially controlled.

Discussion

During the EL epidemic, between 1916 and 1927, which affected as many as 750,000 people, approximately one-third of the patients died in the acute phase, another one-third recovered completely while the remainders were left with chronic neurological deficits[17]. von Economo was the first who recognized and classified three distinct clinical forms of EL:1) somnolent-ophthalmoplegic form characterized by hypersomnolence and ophthalmoparesis 2) hyperkinetic form characterized by chorea and hemichorea 3) amyostatic -akinetic form characterized by Parkinsonism[18]. Toward the end of the worldwide epidemic, von Economo concluded that the somnolence-ophthalmoplegic form was the major clinical form of EL in the acute phase and the other clinical forms were predominated in the post-encephalitic phase[18].

In EL, modes of onset were variable ranging from a dramatically acute onset to a mild episode that may pass unnoticed until post-encephalitic sequelae developed[1]. Some cases began with non-specific febrile illness and one-third of the patients had lethargy[1]. Patients may fall asleep unpredictably but were easily aroused[1,18]. Ocular abnormalities occurred in the large majority of patients in the acute phase[1]. Ptosis is one of the first and most frequent symptoms[1,18]. The bilateral occurrence of ocular palsies is usually incomplete and asymmetrical[18]. Hemiplegia is an unusual presentation[1]. Postencephalitic Parkinsonism can emerge at any time and may be delayed as long as 20 years or more[17]. However, the latency is less than 5 years in 50% and less than 10 years in 85% of patients[17]. The important clinical features in post-encephalitic phase are neuropsychiatric disorders, Parkinsonism, other involuntary movements such as chorea, motor tics and dystonia[11], oculogyric and respiratory crises[1,18]. In adults, personality changes occur in the form of debilitating obsessive-compulsive disorders, hypomanic and other affective disorders and less commonly presented with schizophreniform features[1,11].

In 1987, Howard and Lees documented 4 cases and proposed the diagnostic criteria which included a history of acute or subacute encephalitic illness plus three out of seven of the following features:1) signs of basal ganglia involvement, 2) oculogyric crises, 3) ophthalmoplegia, 4) obsessive-compulsive behaviour, 5) akinetic mutism, 6) central respiratory irregularities, 7) somnolence and/or sleep inversion[19].

Neuroimaging is now the well-recognized diagnostic test of EL like illness especially MRI. CT scan of the brain may not be able to detect any abnormalities. MRI T1 weighted images usually has normal appearance while T2 weighted and FLAIR images depict

high signal intensity mainly in midbrain, substantia nigra, basal ganglia, thalamus[11,13,15]. High signal intensity of temporal lobe[11], frontal lobe[10], deep white matter[15,16] and hypothalamus[15] are occasionally observed. Recently, in the series of 20 cases, Dale et al described a typical MRI changes in 40% of EL like patients at midbrain tegmentum, substantia nigra and basal ganglia[11].

CSF analysis in EL like patients may reveal a non-specific reaction such as mild lymphocytosis and elevated protein concentration (range 0.13-1.2 g/dL)[11]. CSF oligoclonal bands were detected in 69% of cases reported by Dale et al[11]. CSF oligoclonal bands, which reflect the immunological reaction in the central nervous system, may thus serve as a supporting diagnostic test as well as a clue to the pathogenesis of EL like illness.

Since MRI technology and CSF studies for oligoclinal bands are now widely available, they should be included in the diagnostic criteria for EL like illness together with the clinical profiles proposed by Howard and Lees. However, other sporadic viral infections and autoimmune diseases should be rule out.

Forty EL like patients were reported during the last decade[2-16]. The clinical presentations are summarized in Table 1. Abnormal movements were the most common clinical features followed by sleep disorder, neuropsychiatric disorder and ophthalmoplegia respectively. Parkinsonism was the most common abnormal movement. Hypersomnia was the most common sleep disorder. Depression, catatonia and apathy were often found in neuropsychiatric disorders. Oculogyric crisis and respiratory irregularities were occasionally observed. MRI finding and CSF oligoclonal band detection are summarized in Table 2. Abnormal hyper intensity lesions on T2 weighted image at basal ganglia and midbrain were commonly detected. CSF oligoclonal bands could be detected in 76.5% of patients.

The presented patient had subacute somnolent-ophthalmoplegic features followed by Parkinsonian features with neuropsychiatric disorders and the clinical course was compatible with EL like illness. Symmetrical bilateral hypersignal intensity on T2 weighted and FLAIR images revealed by MRI at midbrain, mesial temporal lobe and basal ganglia were nicely correlated with the clinical features of somnolence-ophthalmoplegia, abnormal behaviours and Parkinsonism, respectively. CSF oligoclonal bands detection further supported the clinical diagnosis in the presented case. Other etiologies of rapid course and young Parkinsonism such as Wilson's disease, systemic autoimmune disease had also been ruled out by appropriate investigations.

Histopathological findings in EL reveal perivascular inflammation and neuronal necrosis affecting mainly at midbrain and basal ganglia in acute phase while in chronic phase substantia nigra is uniformly depigmented[1]. EL first appeared at the time of the

Table 1. Clinical features in 40 cases of EL like patients

Clinical	Number	Percentage
Sleep disorder	29/40	72.5
- Hypersomnia	20/40	50.0
- Sleep inversion	5/40	12.5
- Insomnia	4/40	10.0
Ophthalmoplegia	18/40	45.0
Oculogyric crisis	2/40	5.0
Abnormal movement	39/40	97.5
- Parkinsonism	29/40	72.5
- Dystonia	8/40	20.0
- Stereotypies	3/40	7.5
- Myoclonus	2/40	5.0
- Chorea/hemiballismus	2/40	5.0
- Motor tics	2/40	5.0
- Blepharospasm	2/40	5.0
- Facial grimacing	1/40	2.5
Neuropsychiatric disorder	21/40	52.5
- Depression	6/40	15.0
- Catatonia	5/40	12.5
- Apathy	4/40	10.0
- Obsessive compulsive disorder	3/40	7.5
- Anxiety	2/40	5.0
- Psychosis	1/40	2.5
Respiratory irregularities	2/40	5.0

Table 2. Investigation in 40 cases of EL like patients

Investigation	Number	Percentage
MRI		
Normal	9/27	3.3
Hypersignal intensity on T2WI	18/27	66.6
Basal ganglia	11/18	61.1
Midbrain	9/18	50.0
Thalamus	3/18	16.7
Periventricular white matter	2/18	11.1
Temporal lobe	1/18	5.6
Frontal lobe	1/18	5.6
Hypothalamus	1/18	5.6
CSF Oligoclonal band		
Positive	13/17	76.5
Negative	4/17	23.5

influenza pandemic, thus it was believed to be related to influenza viral infection for many decades[1,11,17.20-22]. However, recent investigations of archival EL brain materials have failed to demonstrate influenza RNA[23] which supports the evidence that EL is not invasive influenza encephalitis. Furthermore, the detection of intrathecal oligoclonal bands synthesis which reflects immunological response in the central nervous system[8,9,11,19,22] and the beneficial effects of immunomodulation such as immunosuppressive medications and intravenous immunoglobulin therapy during the acute phase of EL like illness[2,6,8,10,11,15,16,22] supported the immunological mechanisms. Dale et al also observed the relationship between the previous infection such as upper respiratory tract infection, tonsillitis and the rising of ASO antibody titer in more than half of EL like patients[11], thus supported the linkage between EL like illness and infection in the form of post-infectious neurological complications especially post-streptococcal syndrome[11,22]. Moreover, Dale et al could detect autoantibodies reaction against human basal ganglia antigen to different protein bands (40,45,60,98 kDa) in EL like patients[11,22]. These findings strongly supported an autoantibody-mediated mechanism in EL like illness.

Some classical post-infectious autoimmune neurological syndromes such as Sydenham's chorea, pediatric autoimmune neuropsychiatric disorders associated with streptococcal infection (PANDAS) and Bickerstaff's brainstem encephalitis share the clinical syndrome, imaging findings and immunological profiles with EL like illness. Sydenham's chorea which is the most well known post-streptococcal syndrome characterized by chorea and behavioural disorders has basal ganglia autoantigens of molecular weights 40,45,60 kDa except in 98 kDa[11,22,24-26] which was found only in EL like patients. In PANDAS, characterized by neuropsychiatric symptoms and motor tics, autoantibodies reactive against human basal ganglia antigen of molecular weight 60kDa has also been detected[25-27]. Besides, both Sydenham's chorea and PANDAS also show abnormal signal intensity in MRI on T2 weighted images at basal ganglia[28,29]. In Bickerstaff's brainstem encephalitis, characterized by hypersomnolence, ophthalmoplegia and ataxia has abnormal MRI in the form of hypersignal intensity on T2 weighted and FLAIR images at brainstem especially midbrain and bilateral thalamic lesions[30]. It also has autoantibodies, nevertheless in the form of anti-GQ1b antibodies[30]. From these documented syndromes and the recently proposed pathophysiology as mentioned above, EL like illness should be classified as post-infectious neurological syndrome with close linkage to Sydenham's chorea, PANDAS and Bickerstaff's brainstem encephalitis.

EL like illness has been recognized as a re-emerging post-infectious encephalitis. The clinical profiles suggest an EL like illness including abnormal movements, sleep disorder, neuropsychiatric disorder and ophthalmoplegia. The most common abnormal movement was Parkinsonism. Abnormal hypersignal intensity on T2 weighted MRI at basal ganglia, midbrain, thalamus and detection of oligoclonal band in CSF may further support the diagnosis of EL like illness. In the near future, basal ganglia autoantibodies may be the most specific diagnostic test. EL like illness treatments should focus on immunomodulators such as steroid or intravenous immunoglobulin especially in the acute phase.

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โรคที่เหมือนเอนเซฟฟาไลทิส ลีธาจิกา: รายงานผู้ป่วยและทบทวนวารสาร

ณัฐพันธุ์ ศรีดำ, กัมมันต์ พันธุมจินดา

จากการศึกษาเมื่อไม่นานมานี้พบว่า เอนเซฟฟาไลทิส ลีธาจิกา (อีแอล) อาจจะไม่เกี่ยวข้องกับเชื้อ อินฟลูเอ็นซา ไวรัส แต่น่าจะเป็นโรคของภูมิคุ้มกันตนเองหลังจากการติดเชื้อ เกณฑ์ในการวินิจฉัยโรคที่เหมือนอีแอล ประกอบด้วย การนอนมากกว่าปกติและการกลอกตาผิดปกติในระยะฉับพลัน ตามด้วยอาการ พาร์กินสัน, ออคคูโลจัยริก ไครสิส, ความผิดปกติทางจิตประสาท และการหายใจส่วนกลางผิดปกติ ปัจจุบันการถ่ายภาพคลื่นแม่เหล็กไฟฟ้า (เอ็มอาร์ไอ) ซึ่งพบความเข้มมากขึ้นในภาพที่ 2 และแฟลร์บริเวณมิดเบรน เทกเมนตัม และเบสัลแกงเกลียช่วยในการวินิจฉัยเป็นอย่างมาก อย่างไรก็ตามไม่เคยมีรายงานโรคที่เหมือนอีแอลในประเทศไทย ผู้ชายอายุ 17 ปีมาด้วยนอนมากกว่าปกติ 1 สัปดาห์ก่อนมานอนโรงพยาบาล ตรวจร่างกายพบว่าซึม และการกลอกตาผิดปกติ ภาพคลื่นแม่เหล็กไฟฟ้าแสดงความเข้มมากขึ้นในภาพที่ 2 และแฟลร์ที่ มิดเบรน, เบสัลแกงเกลีย และสมองส่วนเทมพอรัล ทั้งสองข้าง ผลการศึกษาน้ำไขสันหลังพบว่าปกติ การตรวจน้ำไขสันหลังโดยวิธี พีซีอาร์ หาเชื้อ เฮอร์ปีส์ซิมเพล็กไวรัส, วาริเซลลาซอสเตอร์ไวรัส, ไซโตเมกะโลไวรัส, เอพส์ไตน์-บาร์ไวรัส, แพน-เอนเทอโรไวรัสและเวสต์ไนล์ไวรัสไม่พบ การตรวจน้ำไขสันหลังหาเชื้อไวรัสเดงกีและเจแปนนิส เอนเซฟฟาไลทิส โดยวิธีฮีมเมกกลูติเนชันไม่พบ ผู้ป่วยได้รับการรักษาโดยการฉีดยา เดกซาเมธาโซนและอิมมูโนโกลบูลิน เข้าหลอดเลือดดำ การนอนและการกลอกตาดีขึ้น 2 เดือนต่อมาผู้ป่วยมีพฤติกรรมแบบจิตเภทและอาการพาร์กินสันเอ็มอาร์ไอพบรอยโรคบริเวณ มิดเบรนและเบสัลแกงเกลียดีขึ้น การศึกษาน้ำไขสันหลังปกติขณะที่ตรวจพบโอลิโกโคลนัลแบน การตรวจทางระบบภูมิคุ้มกันตนเองและระบบน้ำเหลืองหาการติดเชื้อสเตร็พโตคอคคัลก่อนหน้านี้และซิฟิลิสไม่พบ การตรวจผลการทำงานของต่อมธัยรอยด์และระดับเซรูโลพลาสมินในน้ำเหลืองปกติ ได้จ่ายยา ลีโว-โดปา, โคลนาซีแปมและโซเดียมวาลโปรเอท กลุ่มอาการต่าง ๆ ค่อย ๆ ดีขึ้น