

Case Report

Acute Disseminated Encephalomyelitis Following Coronary Angiography in an Elderly Patient

Sengul Y^{1*}, Sengul HS², Gunduz N², Forta H³, Uysal E⁴ and Pazarci NK⁵

¹Department of Neurology, Erzurum Regional Training and Research Hospital, Turkey

²Department of Psychology, Erzurum Regional Training and Research Hospital, Turkey

³Neurologist, Turkey

⁴Department of Radiology, Sisli Hamidiye Etfal Regional Training and Research Hospital, Turkey

⁵Department of Neurology, Sisli Hamidiye Etfal Regional Training and Research Hospital, Turkey

*Corresponding author: Yildizhan Sengul, Department of Neurology, Erzurum Regional Training and Research Hospital, Erzurum, Turkey, Cad Yolu, Palandoken, Erzurum 25240, Turkey, Tel: 90442 232 5000; Fax: 90442 232 6435; Email: yysengul@gmail.com

Received: April 01, 2015; Accepted: May 09, 2015;

Published: May 31, 2015

Abbreviations

ADEM: Acute Disseminated Encephalomyelitis; CNS: Central Nervous System; MRI: Magnetic Resonance Imaging; CSF: Cerebrospinal Fluid; PET: Positron Emission Tomography

Introduction

ADEM is an uncommon, monophasic, demyelinating condition of CNS which is characterized by focal or multifocal white matter lesions. Possibly T cell mediated autoimmune response to myelin basic protein follows an infection or vaccination. Viral infections associated with ADEM are measles, mumps, rubella, varicella-zoster, Epstein Barr virus, cytomegalovirus, herpes simplex virus, hepatitis A, and coxsackievirus. Bacterial infections associated with ADEM are notably mycoplasma pneumoniae. The other bacterial infections are borrelia borgdorgeri, leptospira, and group-A beta-hemolytic streptococci. Anti-rabies vaccination was strongly found to be associated with ADEM. Other vaccinations associated with ADEM include pertussis, diphtheria, measles, mumps, rubella, and influenza [1-2]. It is most frequently seen in children and young adults [3]. ADEM cases in middle-aged or elderly adults are rarely reported [4]. Most adult patients present less frequency of headache, fever and meningismus, and a higher frequency of sensory deficits than children patients. Optic neuritis is also infrequent in adult ADEM [5]. ADEM usually follows 7–14 days after trigger event [1]. Initial clinical symptoms include encephalopathy ranging from lethargy to coma, and sudden onset focal or multifocal neurologic deficits such as visual field defects, aphasia, motor and sensory deficits, ataxia, movement disorders, focal or generalized seizures [6]. Psychiatric symptoms in ADEM are rare [7]. Common psychiatric symptoms include lethargy, irritability, and confusion [8]. There are case reports of patients presenting severe depression [9-10], acute psychosis [4,11] and anxiety [12]. CSF frequently shows some changes. Increased

Abstract

A 62-year-old female patient who had coronary angiography due to chest pain and tachycardia two weeks ago was brought to our psychiatric clinic because of suicidal ideation and behavioral changes. She was referred to our neurology clinic for her headache and somnolence. Her clinic and MRI features were consistent with acute disseminated encephalomyelitis. After methyl prednisolone therapy, her clinical and MRI findings were rapidly got better. Because of occurrence after coronary angiography, presentation of the disease, age of the patient and its prognosis, it was found worth to be mentioned.

Keywords: Acute disseminated encephalomyelitis; Coronary angiography; Suicidal ideation

pressure, lymphocytic pleocytosis and raised protein are mostly seen [1]. Oligoclonal bands in CSF are usually absent [2]. MRI is sensitive an essential diagnostic tool. T2 weighted, and FLAIR images shows multifocal, usually bilateral, but asymmetric and large hyper intense lesions, involving peripheral white and grey matter. Contrast enhanced T1 weighted images may show ring-enhancing lesions [13]. ADEM has usually favorable long term outcome [14]. Full recovery was reported in 50%-75% of patients and permanent neurological deficit in 10%-20% of patients [15]. Mild cognitive deficit was also reported as a sequela in children [16-17]. In adults persistent cognitive impairment was rarely reported [18].

Case Presentation

A 62 year old female was brought to our neurology clinic by her family. She had coronary angiography two weeks before her headache and suicidal thoughts had started. On her neurological examination, she had somnolence but she was cooperative. Her reaction time was prolonged. She occasionally had orientation disorder. She had no meningeal signs and cranial nerve pathologies. Motor and sensory system examinations were normal. Deep tendon reflexes were normal and plantar reflex was bilaterally flexor. There was no history of immunization, infectious or vaccination in recent days. She had no known chronic illness and history of drug use. Hemogram, urine, and routine blood examination (liver and kidney functions, glucose levels etc.) were normal. Lumbar puncture showed mildly increased pressure, raised protein. Oligoclonal bands in CSF were type 3 positive. Magnetic resonances imaging showed axial T2WI and FLAIR sequence demonstrated multifocal hyper intensities in peri ventricular and subcortical region on both cerebral hemispheres. There are also small hyper intensities on both basal ganglia. Axial contrast enhanced T1 WI showed that hyper intensities demonstrated punctate and ring enhancement. Figure 1 shows

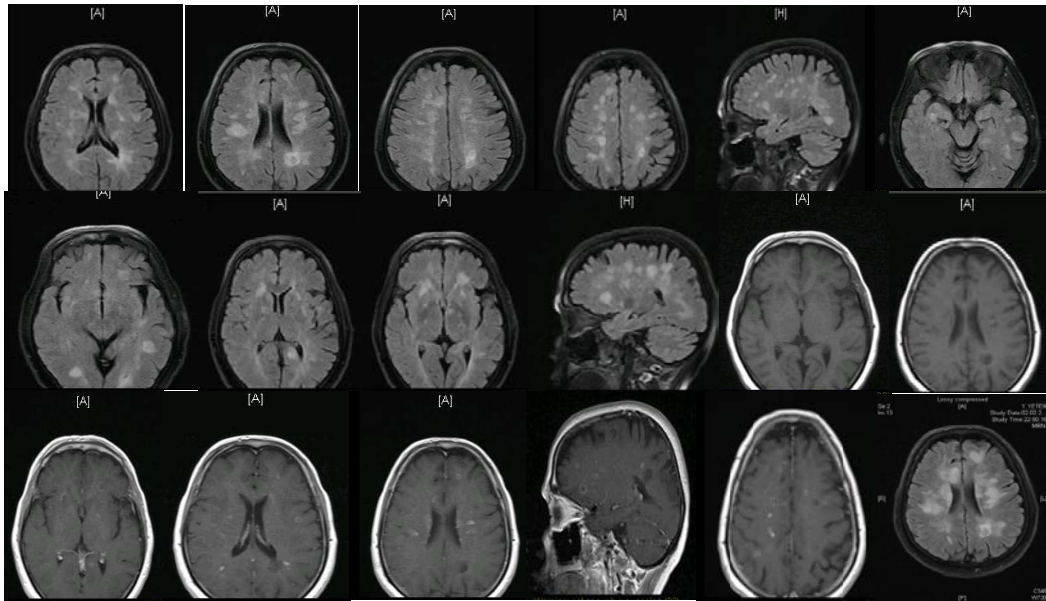


Figure 1: T2WI and FLAIR sequence shows multiple rounded lesions involving basal ganglia. Axial contrast enhanced T1 WI shows that hyper intensities demonstrate punctate and ring enhancement.

her MRI features when she hospitalized. Electroencephalography showed mild generalized slowing. Infectious pathologies (such as viral encephalitis etc.), other demyelinating diseases (eg. systemic vasculitis) and metastatic lesions were considered as differential diagnoses. Infectious endocarditis was also considered because of coronary angiography story. CSF culture and tuberculosis culture were negative. Vasculitis panel for systemic vasculitis was negative. PET was normal. Transthoracic echocardiography was normal. Intravenous methylprednisolone treatment resulted dramatic improvement. Twenty days later when we were discharging her from hospital, she was recovered. Her neurological examination was normal but when we applied mini mental test (MMT) her response was prolonged. MMT score was 22(22/30). Her husband and children implied us that she was an analphabetic and also she reverted back to the person who she was before the disease. After six months follow-up we performed a neurologic examination, full neuropsychological assessment. She had difficulties in adapting to tests. Paying attention and focusing were also impaired. Short term memory was better than long term memory but they were both under norms. Visuospatial functions were partially preserved but as a result of not focusing to the test it could not be fully evaluated. In addition because of her intellectual deficiency, her test performance was quite low. MRI showed nearly total recovery.

Discussion

Coronary angiography is defined as the radiographic visualization of the coronary vessels after injection of radiopaque contrast media. ADEM following by coronary angiography has not been reported yet. Radiopaque contrast media might have trigger the immune mediated reaction and that could cause ADEM. Type 3 oligoclonal bands are mostly seen in most immune mediated reactions. In this case, period of time between coronary angiography and the onset of neurological symptoms, MRI features and CSF findings were consistent with the diagnosis of ADEM. Clinical and MRI findings can vary by a wide

range in ADEM. Neurological symptoms include varying degrees of mental state changes ranging from drowsiness to coma [9]. The neuropsychiatric symptoms described include aggression, agitation, auditory hallucinations, catatonic waxy flexibility, delusions, disorganized behavior, disorganized thinking, disorientation, inappropriate laughter, hostility, irritability, mania, mood lability, mutism, paranoia, and personality change [12]. There have been reported several severe depression cases but [9-10], our case presented suicidal ideation and suicidal thoughts at the beginning of the disease process. This was also never mentioned before in any ADEM cases. Another difference of our case was her cognitive situation. ADEM is known as its favorable outcome, most of cases result full recovery. Attention, concentration and long term memory deficits were determined in our neuropsychological examination. Up to the day there is only one case report about persistent cognitive dysfunction in an adult patient [18].

Conclusion

We presented an unusual ADEM case. Although majority of patients are children and young adults, it can be seen adults and older ages. ADEM has been associated with numerous immunological triggers mostly infections or vaccinations, maybe we determine frequently these factors in children but half of adults with ADEM show no clear associations with these trigger factors [19]. Our patients had a history of coronary angiography. We thought radiopaque contrast media might be a trigger factor in this case. ADEM can manifest a wide spectrum of psychiatric symptoms. Our case presented suicidal ideation. For follow up of ADEM patients we must pay more attention about cognitive functions.

Acknowledgements

We thank to Dr. Murat Terzi, Dr.Gulay Ozgen Kenangil and Dr.Dilek Necioglu Orken for their valuable contributions.

References

1. Garg RK. Acute disseminated encephalomyelitis. *Postgrad Med J*. 2003; 79: 11-17.
2. Murthy JM. Acute disseminated encephalomyelitis. *Neurol India*. 2002; 50: 238-243.
3. Stonehouse M, Gupte G, Wassmer E, Whitehouse WP. Acute disseminated encephalomyelitis: recognition in the hands of general paediatricians. *Arch Dis Child*. 2003; 88: 122-124.
4. Wang PN, Fuh JL, Liu HC, Wang SJ. Acute disseminated encephalomyelitis in middle-aged or elderly patients. *Eur Neurol*. 1996; 36: 219-223.
5. Schwarz S, Mohr A, Knauth M, Wildemann B, Storch-Hagenlocher B. Acute disseminated encephalomyelitis: a follow-up study of 40 adult patients. *Neurology*. 2001; 56: 1313-1318.
6. Kumar P, Kumar P, Sabharwal RK. Acute disseminated encephalomyelitis: case report and brief review. *J Family Med Prim Care*. 2014; 3: 443-445.
7. Mahgoub N, Adegbola O, Alexopoulos GS. Acute demyelinating encephalomyelitis presenting with psychiatric symptoms. *Int J Geriatr Psychiatry*. 2013; 28: 1318.
8. Patel SP, Friedman RS. Neuropsychiatric features of acute disseminated encephalomyelitis: a review. *J Neuropsychiatry Clin Neurosci*. 1997; 9: 534-540.
9. Matsuda M, Miki J, Tabata K, Ikeda S. Severe depression as an initial symptom in an elderly patient with acute disseminated encephalomyelitis. *Intern Med*. 2001; 40: 1149-1153.
10. Krishnakumar P, Jayakrishnan MP, Devarajan E. Acute disseminated encephalomyelitis presenting as depressive episode. *Indian J Psychiatry*. 2011; 53: 367-369.
11. Nasr JT, Andriola MR, Coyle PK. ADEM: literature review and case report of acute psychosis presentation. *Pediatr Neurol*. 2000; 22: 8-18.
12. Habek M, Brinar M, Brinar VV, Poser CM. Psychiatric manifestations of multiple sclerosis and acute disseminated encephalomyelitis. *Clin Neurol Neurosurg*. 2006; 108: 290-294.
13. Steiner I, Budka H, Chaudhuri A, Koskiniemi M, Sainio K, Salonen O, et al. Viral meningoencephalitis: a review of diagnostic methods and guidelines for management. *Eur J Neurol*. 2010; 17: 999-999e57.
14. Anlar B, Basaran C, Kose G, Guven A, Haspolat S, Yakut A, et al. Acute disseminated encephalomyelitis in children: outcome and prognosis. *Neuropediatrics*. 2003; 34: 194-199.
15. Alexander M, Murthy JM. Acute disseminated encephalomyelitis: Treatment guidelines. *Ann Indian Acad Neurol*. 2011; 14: S60-64.
16. Suppiej A, Cainelli E, Casara G, Cappellari A, Nosadini M, Sartori S. Long-term neurocognitive outcome and quality of life in pediatric acute disseminated encephalomyelitis. *Pediatr Neurol*. 2014; 50: 363-367.
17. Hahn CD, Miles BS, MacGregor DL, Blaser SI, Banwell BL, Hetherington CR. Neurocognitive outcome after acute disseminated encephalomyelitis. *Pediatr Neurol*. 2003; 29: 117-123.
18. Adamec I, Klepac N, Kolenc D, Ozretić D, Habek M. Isolated and persistent cognitive dysfunction in a patient with acute disseminated encephalomyelitis. *Cogn Behav Neurol*. 2013; 26: 30-35.
19. Noorbakhsh F, Johnson RT, Emery D, Power C. Acute disseminated encephalomyelitis: clinical and pathogenesis features. *Neurol Clin*. 2008; 26: 759-780, ix.