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Postanoxic Parkinsonism: A Case Report

Postanoksik Parkinsonizm: Olgu Sunumu

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Summary

Postanoxic encephalopathy is a rare syndrome manifested by neurological signs in the late period. In this case we aim to discuss postanoxic parkinsonism occurring late following resuscitation. *(Turkish Journal of Neurology 2013; 19:23-4)*
Key Words: Postanoxic, parkinsonism, resuscitation

Özet

Postanoksik ensefalopati sendromu nadir görülür ve geç dönemde nörolojik bulgular ile ortaya çıkar. Biz bu olguda resüsitasyon sonrası geç dönemde görülen postanoksik parkinsonism tablosunu tartışmayı amaçlıyoruz. *(Türk Nöroloji Dergisi 2013; 19:23-4)*
Anahtar Kelimeler: Postanoksik, parkinsonism, resüsitasyon

Introduction

Late onset post-anoxic encephalopathy syndrome is a rare condition most commonly developing due to carbon monoxide (CO) intoxication. Following a brief improvement in clinical features, apathy, agitation, confusion and/or progressive neurological deficits continue (1-4). The first reported case is a patient who attempted suicide with a gas lamp in 1926 and developed parkinsonism 26 days later. The autopsy performed following the patient's death 2 months later showed bilateral symmetrical irregular widespread necrosis and demyelination in the globus pallidus (3). Common autopsy findings are widespread cerebral hemispheric demyelination and degeneration in the basal ganglia (4). On the other hand, subcortical arcuate fibres, corpus callosum, axons ans cerebral blood vessels are relatively preserved and generally there is no edema. Radioimaging findings show, similarly to pathology, widespread demyelination and bilateral symmetric basal ganglia involvement (5,6,7). The pathogenesis of late onset postanoxic encephalopathy is not clearly understood (3) and various hypotheses have been suggested including myelinosis due to the secondary effect of anoxic damage in small cerebral vessels and postanoxic demyelination caused by cerebral edema (8). Moreover, free radicals and the damage in the electron transport chain are thought to play a role in the pathogenesis of parkinsonism, as well (9). There are no proven specific pharmacologic treatment protocols. Some publications show that parkinsonism symptoms respond to levodopa treatment (10). In this report we aim to present the clinical picture and treatment of a patient presenting with posthypoxic parkinsonism.

Case

Sixty year old female patient was resuscitated and connected to a mechanical ventilator due to sudden respiratory distress and consequent loss of consciousness, 2 days after she had a total knee prosthesis surgery. She was monitored for 48 hours, treatment was planned with a diagnosis of pulmonary embolus, and she was transferred to a private room after her respiration improved and she was disconnected from the ventilator. Fully recovered on the eight day, she was discharged from the hospital. However, 10 days after the arrest, she started yelling at people around her, using inappropriate words, and gestures and showing irritability. Following complaints of resting tremor in both hands, which was more pronounced in the right hand, difficulty in walking, slowing down in movements, and difficulty in speech, she was hospitalized in our clinic. Her physical examination did not reveal a pathology. Her neurological examination showed that she was conscious, cooperative and her orientation was normal; cranial nerve examination was normal except for hypomimia, muscle strength was globally normal. Muscle tone examination showed clear rigidity in all extremities. There was marked bilateral resting tremor in the right hand. Deep tendon reflexes had increased. Bilateral Hoffman and Babinski reflexes were positive. Her speech was hypophonic, her gait was in anteflexion posture and with small steps. Mental state examination

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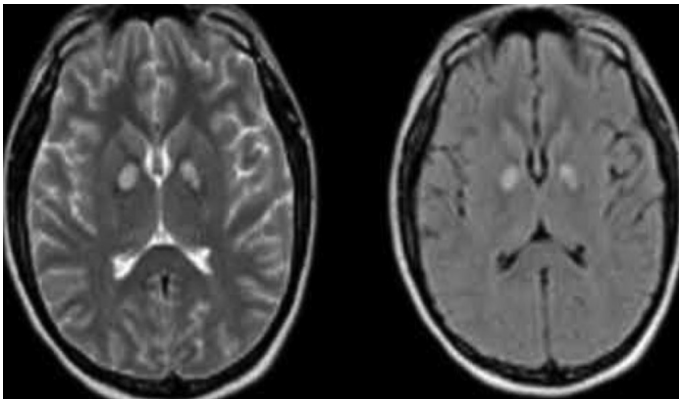

Figure 1. Radioimaging of the lesion due to hipoxia in the T2 and FLAIR sequences (hyperintense lesions in T2 and FLAIR sequences in both globus pallidus)

was normal. As these findings suggested secondary parkinsonism, further investigations were planned. Routine blood examinations were normal. Thyroid function tests and autoantibodies, vasculitis and infection markers were negative. Evoked potentials and EEG results were normal. Vertebral-Carotis Doppler USG and cranial angiogram results were normal. Cranial MRI showed hyperintense lesion areas in T2 and FLAIR sequences in both globus pallidus (Figure 1). These findings were thought to have developed due to hypoxia. Based on clinical examination findings and radioimaging results, the patient was diagnosed with postanoxic parkinsonism developing secondary to anoxia following cardiopulmonary arrest. The patient was initiated levodopa 250 mg/day treatment. Her gait started improving from the 7. day of treatment, and there was a decrease in her rigidity, and marked improvement in her tremor. As her complaints decreased, her treatment was managed and she was discharged.

Discussion

Late onset postanoxic deterioration (LAD) should be discussed within the scope of postresuscitation syndromes (PRE). Some patients exit from coma secondary to PRE, and start improving, but GAD syndrome characterized by apathy, confusion, impairment in walking, spasticity, incontinence, movement disorders and disarthria may develop in the ensuing period.

The latent period between the anoxic event and the onset of neurologic symptoms reflects the time needed for remyelination, oxidation reactions, central synaptic reorganization, trans-synaptic neuronal degeneration, collateral formation and hypersensitivity of recepteor due to denervation.

These patients usually improve with supportive treatment, but residual deficits are a rule more than an exception. The dominant clinical picture is parkinsonizm. The main pathology is varying degrees of demyelination in the semioval centrum (area of subcortical tracts) as well as bilateral pallidal necrosis and patchy cortical necrosis (especially hypocampal). Although oligodendroglial apoptotic damage is thought to play a role, there is no adequate evidence (11,12,13,14).

While our case, similar to those in literature, improved clearly following resuscitation, neurological symptoms developed about 10 days later, and parkinsonism was the main clinical picture after approximately day 20.

There are reports in literature of visualizing symmetrical localized hyperintense lesions in bilateral basal ganglia in neuroimaging (6). Similar to the reports in literature, we found these MRI changes in our case, as well.

The most important point in treatment is early resuscitation to prevent hypoxia and correct metabolic deficits. There is no proven neuroprotective agent to prevent the development of posthypoxic encephalopathy. Animal studies have shown that hypothermia is partially beneficial, but human studies are not yet adequate (15). Levodopa may be used for Parkinson's symptoms (10). As in the cases in literature, we treated our case with levodopa and our results were successful.

Cardiopulmonary arrest is an significant emergency clinical condition clinicians commonly encounter. Late onset postanoxic encephalopathy should be considered in patients who have responded to treatment and improved initially, but later deteriorated and developed neurologic symptoms. We would like to emphasize that early initiation of symptomatic treatment and rehabilitation is of utmost importance.

References

1. Hori A, Hirose G, Kataoka S, Tsukada K, Furui K, Tonami H. Delayed postanoxic encephalopathy after strangulation. Serial neuroradiological and neurochemical studies. Arc Neurol 1991;48:871-874.
2. Dooling EC, Richardson EP Jr. Delayed encephalopathy after strangling. Arc Neurol 1976;33:196-199.
3. Ginsberg MD. Delayed neurological deterioration following hypoxia. Adv Neurol 1979;26:21-44.
4. Plum F, Posner JB, Hain RF. Delayed neurological deterioration after anoxia. Arch Intern Med 1962;110:18-25.
5. Bhatt MH, Obeso JA, Marsden CD. Time course of postanoxic akinetic-rigid and dystonic syndromes. Neurology 1993;43:314-317.
6. Takahashi W, Ohnuki Y, Takizawa S, Yoshii F, Takagi S, Kamei T, Shinohara Y. Neuroimaging on delayed postanoxic encephalopathy with lesions localized in basal ganglia. Clin Imaging 1998;22:188-191.
7. Li JY, Lai PH, Chen CY, Wang JS, Lo YK. Postanoxic parkinsonism: clinical, radiologic, and pathologic correlation. Neurology 2000;55:591-593.
8. Choi IS. Delayed neurologic sequelae in carbon monoxide intoxication. Arch Neurol 1983;40:433-435.
9. Gillespie ND, Hallhead G, Mutch B, James PB, McMurdo ME. Severe parkinsonism secondary to carbon monoxide poisoning. JR Soc Med 1999;92:529-530.
10. Ringel SP, Klawans HL Jr. Carbon monoxide-induced Parkinsonism. J Neurol Sci 1972;16:245-251.
11. Barnes MP, Newman PK. Delayed encephalopathy following cardiac arrest. Postgrad Med J 1985;61:253-254.
12. Heckmann JG, Erbguth F, Neundörfer B. Delayed postanoxic demyelination registry. Neurology 1998;51:1235-1236.
13. Choi IS. Parkinsonism after carbon monoxide poisoning. Eur Neurol 2002;48:30-33.
14. Chalela JA, Wolf RL, Maldjian JA, Kasner SE. MRI identification of early white matter injury in anoxic-ischemic encephalopathy. Neurology 2001;56:481-485.
15. Bona E, Hagberg H, Løberg EM, Bågenholm R, Thoresen M. Protective effects of moderate hypothermia after neonatal hypoxia-ischemia: short- and long-term outcome. Pediatr Res 1998;43:738-745.