A Case Report on Wallenberg Syndrome

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Abstract

Introduction: Wallenberg Syndrome or lateral medullary syndrome is a rare prevailing disease detected among the young population. The particular stroke has shown to distort the speech, orientation, and vision of the bearer.

Case Report: A 31 years old lady migrant diagnosed with the acute infarct in the right dorsolateral medulla, complaint of dysphasia, dysarthria, ataxia of the gait, weakness of the right upper limb swaying to one side and numbness of face. The patient serum leukocyte and low density lipoprotein level depicted to be abnormally high. The physician chose to treat her symptomatically, however he was also concern on intra cranial pressure correction. Gradually, she regains her cognition and was discharged on the fifteenth day of admission.

Discussion: The treatment was based on hospital guideline and physician experience. The functional constrains as a result of the syndrome was rectified enough to satisfy her minimal life requirement.

Keywords: Young stroke; Female; Symptomatic care; Wallenberg Syndrome

Introduction

Wallenberg syndrome or lateral medullary syndrome was first described in 1895 by Gaspan Viessux [1]. Adolf Wallenberg, a German physician and neuroanatomist who gave an accurate description of the pathology of the syndrome in 1901 after an autopsy of the specimen [2]. The wallenberg syndrome is a rare syndrome involving the part of medulla oblongata with consequent cross loss of pain and temperature sensation in the ipsilateral orofacial region and counter-lateral body [3]. The clinical symptoms and signs are dysphagia, slurred speech, ataxia, facial pain, vertigo, nystagmus, diplopia, possibly palatal myoclonus. The patients experience absence of pain in the ipsilateral side of the face as well as absence of corneal reflex due to damage to the spinal trigeminal nucleus. The patient will be developing experience nystagmus and vertigo leading to dysphagia. The strokes in one of the two arteries in the brain stem lead to Wallenberg syndrome [4].

Case Report

A 31 years old female patient was admitted in the neurology department with chief complaints of sudden onset of dysphasia, dysarthria and ataxia of the gait. She had weakness of the right upper limb swaying to one side and numbness of face since one day. She was not able to stand, facial pain, vertigo and had difficulty to swallow. The patient had gestational diabetes mellitus which was managed with insulin therapy. The pulse rate, respiratory rate and blood pressure and her mental status were found to be normal. The patient had experienced hiccups during the hospital stay. She had experienced plantar flexor and nystagmus. Medical Research Council Scale was 4 infer for a slight resistance at her right lower limb. The physician requested for blood routine examination, MRI of brain, Fasting lipid profile and cardiology evaluation.

The blood routine examination results of the patient were normal expect mildly elevated leukocytes count. The MRI of brain gave crucial evidence in diagnosing the disease, an acute infarct in the right dorsolateral medulla. The V cranial nerve showed left face onion peel like appearance, VII cranial nerve appeared normal, IX and X cranial nerves were having elevated right uvula and XII showed defective right side. TOF MRA of neck and brain was performed using 3d TOF technique. The fasting lipid profile showed serum triglycerides were elevated. ECHO cardiograph revealed normal cardiac status.

The patient was managed with osmotic diuretic injection. Mannitol was preferred in this case in order to lower the intracranial pressure it 20% solution given as 300 ml bolus and then 100ml thrice for 7 days. During the therapy, serum sodium and serum osmolality was analysed. Enoxaparin 40mg/0.4ml, low molecular weight heparin was given as subcutaneously for
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seven days. Anticoagulant and antiplatelet agents such as aspirin 325mg OD followed by 150 mg OD and dospidrogel 300mg followed by 75mg was given. Cholesterol level was normalized by consuming atavostatin 40mg at night. Third generation cephalosporins, cefoperazone 1g combined with beta-lactamase inhibitor, sulbactam 500mg had been administered to the patient. Piracetam 3g QID initially followed by a dose of 800mg thrice daily was given to improve her cognition. Proton pump inhibitors, Pantoprazole was given 40mg once daily to prevent ulceration and gastric irritation associated with aspirin. The patient’s vertigo was treated with betahistine 16mg thrice daily and ondansetron 4mg was also provided. Supportive physiotherapy was also given. The symptoms began to subsided gradually and the patient was discharged after 15 days with proper advice and medications.

Discussion

Wallenberg syndrome otherwise known as young stroke and it is rare. H.Lzhang et al. [5] and R .Saha et al [6] has reported case of a 19 years old college student and a 50 years old male, respectively. Here, we have treated the patient who is 31 years old, mother of 2 children and was settled abroad. The evidence of the syndrome can be interpreted from the patients MRI of brain [7]. In our case MRI of the patient showed an acute infract in the right dorsolateral medulla. It gave the precise anatomic boundary of the intramedullary hematoma and was well correlated with the clinical findings. In H. Lzhang et al. [7] an arteriosclerotic-thrombotic occlusion of the homolateral intracranial vertebral artery or posterior inferior cerebellar artery, only occasionally and an occlusion of the basilar artery or of the distal extracranial vertebral artery was responsible. However, in our case the infract is seen in the right dorsolateral medullary region which may be caused due to the syphilitic vascular metastases or encephalitis in the region of dorsolateral medulla oblongata.

Dysphagia was also experienced by the patient according to R. Saha et al. [8].Paralysis of the palate and vocal cord (the ninth and tenth cranial nerves) is related to the dysphagia, hoarseness and diminished gag reflex [9,10]. In this case the patient had experienced nystagmus, vertigo, nausea, vomiting which was observed to have similarities with the case report published by McGhie et al. [9]. Here the patient had experienced ipsilateral ataxia and falling towards the side of lesion, which is also described in the case report by McGhie et al [11]. Ipsilateral ataxia is caused by infarction of the inferior cerebellar peduncle and vertigo from infarction of the vestibular nuclei. Feeling of falling towards the side of lesion is due to the disease of the spinocerebellar tract.

Patient had experienced numbness of the face and weakness. The facial weakness is possibly due to fibres of the facial nerve that loop caudally into the medulla before exiting at the pontomedullary junction [12]. Hiccups are repeated involuntary spasmodic contractions of the diaphragm accompanied by sudden closure of the glottis producing hic sound. In the study done by M H park et al. [13], 51 patient with lateral medullary syndrome infraction were investigated by magnetic resonance imaging within three days of onset of infraction and all 51 patient developed hiccups. In the present case also the patient had experienced hiccups. Here in this case the patient was managed with symptomatic treatment and physiotherapy. Saha et al [14] described the same treatment pattern which was followed for our patient with the syndrome. The patient was given osmotic diuretic to reduce the intracranial pressure.

The widely prescribed drug is mannitol, it’s use in patients with cerebral edema after infraction does not alter midline tissue shifts or worsen neurologic status [5]. 20 % in 300ml constitute 60g of mannitol and subsequently 100ml was given thrice per day. The dose was calculated on the bases of weight i.e. 1g/kg bolus administered initially followed by 0.3g/kg which comply to the institutional guideline [15-21]. The physician was cautious about the serum sodium level, serum osmolality and the renal function of the patients taking into the consideration the complication associated with mannitol. The patient was hospitalized after 4.5 hours as a result the neurologists opt to start with antiplatelet and anticoagulant. Aspirin, which is always the preferred choice in this case as it reduces the risk of early recurrent ischemic stroke. The International Stroke Trial allocated 300mg dose of the drug to the subjects within 48 hours of symptom onset experienced significant reductions in the recurrence of ischemic stroke [19]. Similarly study conducted in China, 160mg aspirin was given to the enrolled subjects, 14 percent relative risk reduction in mortality was found [6]. Considering the above two studies, the efficacy of aspirin is well sound and as a result improvement was observed [22].

Early initiation and short-term use of combination antiplatelet agents for acute ischemic stroke is beneficial. According to the CHANCE trial, patients enrolled with 24 hours of onset ischemic stroke with clopidogrel 300 mg loading dose, then 75 mg daily for 90 days, plus aspirin 75 mg daily for the first 21 days, the rate of haemorrhagic stroke was low in the treatment groups [23]. The choice of aspirin combined with clopidogrel is a usual practice in the hospital and such a pattern lead to optimization of medical needs confined to the patient. Among patients receiving clopidogrel following acute infarction, concommitant therapy with proton pump inhibitors other than pantoprazole was associated with a loss of the beneficial effects of clopidogrel and an increased risk of reinfarction [14].

The available evidence suggests that early anticoagulation with heparin or low molecular weight heparin is associated with a higher mortality and worse outcomes compared with aspirin treatment initiated within 48 hours of ischemic stroke onset [2]. However, enoxaparin 40mg twice subcutaneously is administered on the first day of treatment, taking into account of the onset of symptoms occurred early than 24 hours of hospitalization and the patient response to the therapy was
pleasant. Antibiotic administered as injection, empirically a broad spectrum is always preferred. Also, leukocyte level elevation can suspect prior invasion of micro-organism, which left untreated, would worsen the patient’s health status. Cognition enhancer was given for neuronal protection. Post hoc analyses suggest that piracetam may confer benefit when given within 7 hours of onset, particularly in patients with stroke of moderate and severe degree [14]. High dose i.e. 12 g per day regimen was given initially than dose was reduced to a comfortable size. The patient was mental alertness improved gradually pertaining to the novel treatment strategy.

Statins were started as the triglyceride level was elevated. In patients with hyperlipidemia, treatment with HMG CoA reductase inhibitors decreases the risk of stroke, when comparing with other agents (fibrates, resins) [7,4]. Therefore, it seems plausible that the protective effects of statins are not mediated by cholesterol lowering [9], but by anti-atherothrombotic properties. Physiotherapy further helped the patient to get better [21]. Betahistine, a vasodilator available in Europe, Central and South America, and Canada, is reported to act by improving microvascular circulation in the stria vasularis of the cochlea [12] or by inhibiting vestibular nuclei activity [21]. Antihistamine meclizine 25mg every six hourly could have been added along.

Conclusion

Being a rare syndrome, the physician managed to treat the patient in an optimal style. The patient was satisfied with her health outcome.

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