

A Case Report on Wallenberg Syndrome

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I. INTRODUCTION

Wallenberg syndrome or lateral medullary syndrome was first described in 1895 by Gaspar 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 contralateral 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]. The most common risk factors for Wallenberg syndrome include hypertension (high blood pressure), smoking, and diabetes. Other, less common, risk factors include connective tissue disorders, such as Marfan syndrome, Ehlers-Danlos syndrome, and fibromuscular dysplasia. In younger people, the most common cause of Wallenberg syndrome is vertebral artery dissection. Like most ischemic stroke syndromes, initial diagnosis is usually suspected from the patient's clinical characteristics upon physical examination. Physical examination reveals sensory and motor deficits affecting the face and cranial nerves on the ipsilateral (same) side of the infarct. Ataxia, nystagmus, Horner syndrome, and damage to the spinal trigeminal nucleus causes absence of pain and temperature sensation on the ipsilateral side of the face, as well as a diminished corneal reflex. Patients with a suspected diagnosis should undergo MRI evaluation, including diffusion-weighted imaging, which is the best tool to confirm the infarct in the inferior cerebellar area or lateral medulla. The management of Wallenberg syndrome

is similar to the management of any acute stroke. Rapid evaluation is an essential component in improving the patient's clinical outcome and prognosis. The goal of treatment is to reduce the size of infarction and prevent medical complications. Additional long term treatments include speech and swallowing therapies, along with early physical and occupational therapies.

II. CASE REPORT

A 44 years old male patient known case of T2DM came with complaints of gait unsteadiness and double vision. He is a known case of recent ischemic stroke. The pulse rate, respiratory rate and blood pressure were found to be elevated. On examination patient had gait ataxia and lateropulsion to left, power 5/5 all four limbs; right hemianesthesia. The physician requested for blood routine examination, MRI of brain, Fasting lipid profile and cardiology evaluation. MRI Brain with MRA done showed acute left lateral medullary infarct. He was started on dual antiplatelet and other supportive medication. His blood pressure was high at the time of admission and started on antihypertensive medication. Patient had elevated lipid profile and was started on statin. Cardiology consultation was done to rule out cardioembolic events and orders were carried out. ECHO done showed EF 69%. Patient had elevated liver enzymes for which gastrology consultation was done and orders were carried out. Patient was advised to do OGD however he was not willing for the same and planned to do later. USG abdomen done showed hepatic parenchymal changes right renal simple cyst grade 1 prostaticomegaly. His blood sugar values were controlled with Insulin and OHA. Physiotherapy was done during hospital stay and advised to continue physiotherapy at home. 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. Antibiotic administered as injection, empirically a broad spectrum is always preferred. Anticoagulant and antiplatelet agents such as aspirin 325mg OD followed by 150 mg OD and clopidogrel 300mg followed by 75mg was given. Cholesterol level was normalized by consuming atorvastatin 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 subside gradually and the patient was discharged after 15 days with proper advice and medications. 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 [4]. High dose i.e. 12 g per day regimen was given initially than dose was reduced to a comfortable size. The patient's 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) [3,4]. 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 [9]. Betahistine, a vasodilator available in Europe, Central and South America, and Canada, is reported to act by improving microvascular circulation in the stria vascularis of the cochlea [12] or by inhibiting vestibular nuclei activity [9]. Antihistamine meclizine 25mg every six hourly could have been added along.

III. DISCUSSION

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.

The treatment was based on hospital guideline and physician experience. The functional constrains as a result of the syndrome was rectified enough to satisfy his minimal life requirement. In our case the infarct 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 infarction were investigated by magnetic resonance imaging within three days of onset of infarction 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, its use in patients with cerebral edema after infarction 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 Trail 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 [13]. 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, concomitant 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 vascularis 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.

Keywords: Young stroke; male; Symptomatic care

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