



# Author's experience of lateral medullary infarction – thermal perception and muscle allodynia

Shuji Kamano\*

*Kami-Itabashi Hospital, 4-36-9 Tokiwadai, Itabashi-ku, Tokyo, Japan*

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## Abstract

The patient, the author (S.K.), is a 67-year-old male. He has the typical dissociated pain, altered temperature sensation and ataxia often encountered by patients with lateral medullary infarction. This started at the time of his admission to hospital.

Several weeks after discharge, he experienced the withdrawal reaction to high temperature, first mentioned by Rousseaux (Stroke 30 (1999) 2223), and movement allodynia as described by Bowsher (J Neurol Neurosurg Psychiatry 61 (1996) 62). The article describes his personal experience of the symptoms of central post-stroke pain and allodynia. A difference between muscle allodynia and conventional allodynia caused by dermal stimulation is proposed.

Follow-up evaluation of sensory symptoms that appear several weeks after discharge is essential to ensure measures to alleviate them are provided.

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## 1. Introduction

The author (S.K.) has suffered from lateral medullary infarction; symptomatically it is similar to typical Wallenberg syndrome. Concerning this disease, Wallenberg (Wallenberg, 1895, 1901) is usually credited with being the first person to describe it, however, Swiss physician Gaspard Vieusseux presented his own experience of dissociated sensory loss to the Medical and Chirurgical Society of London in 1810. (Romano and Merritt, 1941; Norrving and Cronqvist, 1991).

One of Japan's pioneering neurosurgeons, Mizuho Nakata suffered this disease from the age of 60, and he reported his personal experience in a Japanese medical journal (Nakata, 1953). He offered thanks for having the chance to experience complicated neurological symptoms first hand and because of this he realized that information gained by physicians who are affected by this disease is indispensable.

## 2. Case report

The patient is a 67-year-old retired male neurosurgeon. He experienced heavy sweating and occipitalgia after a doze on February 2, 2001. He did not consult a neurologist immediately because his neck was supple, his Kernig's sign was negative and his finger–nose test was normal. The next morning, he could not walk without assistance. Right sided ataxia was obvious, but consciousness was clear without any sign of vertigo, nausea or headache. He suspected that he had had some form of cerebellar stroke and consulted a neurologist.

Computerised tomography was not helpful but the magnetic resonance diffusion image revealed a high signal area in the right half of the medulla oblongata. T2 weighted image showed a slightly increased signal, whereas T1 weighted image demonstrated a high signal band along the right vertebral artery suggesting dissecting aneurysm of the right vertebral artery (Fig. 1).

On admission, he presented with hoarseness and sensory disturbance in the right half of his face including nostril and outer ear meatus. The sensory disturbance included dull touch sensation as well as analgesia and thermoanesthesia. Sensory disturbance below the neck was not definite at that

\* 10-3, Oyamacho. Shibuya-ku, Tokyo, 151-0065 Japan. Tel.: +81-3-3467-8203; fax: +81-3-3467-8203.

E-mail address: [skamano@mb.kcom.ne.jp](mailto:skamano@mb.kcom.ne.jp) (S. Kamano).

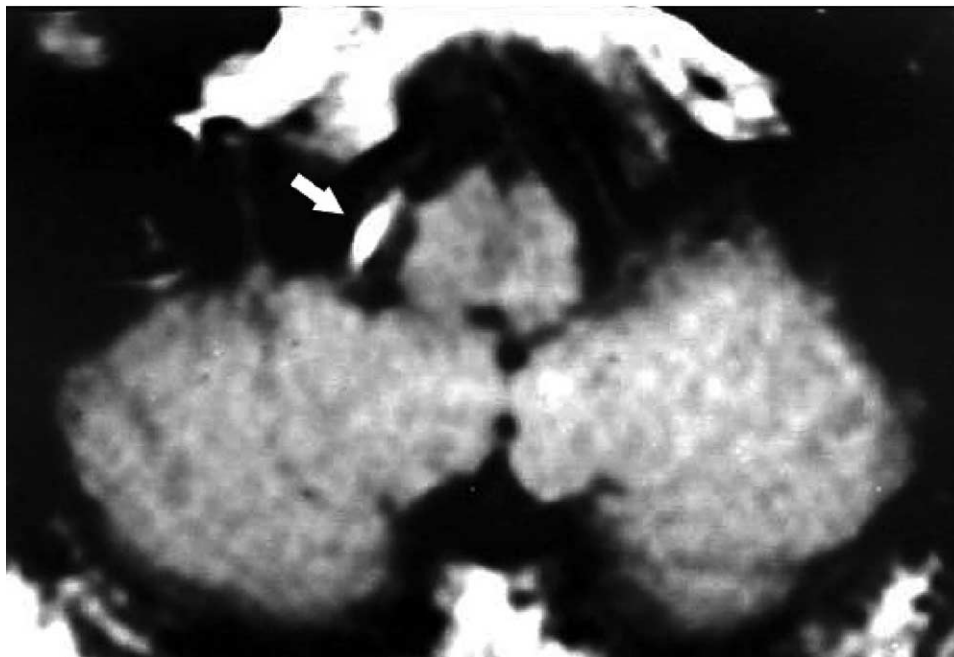


Fig. 1. T1 weighted image at the lower medulla. High signal band along the right vertebral artery suggests a dissecting aneurysm.

time. Dysmetria and dysdiadochokinesis were prominent on the right side. No motor weakness was obvious. The attending physician noted a right sided Horner's syndrome and rotating nystagmus in the patient's left lateral gaze. The right vocal cord was completely paralysed. Urination was difficult so an indwelling catheter was placed. Although food intake was possible, water intake was almost completely impossible during the first 2 days. Analgesia and thermoanesthesia in the left half of the body became apparent within 2 days.

The patient's systolic blood pressure increased to 190 mm Hg for 3 days and the occasional administration of 10 mg of Nifedipine was necessary to control this. Thereafter, Nivadipine at 4 mg/day was prescribed. A selective anti-thrombin drug, (2R4R)-4-methyl-1- $\{N2-[(RS-3-methyl-1,2,3,4-tetrahydro-8-quinoliny)]-L-arginy]$ -2-piperidinecarboxylic acid, Algotroban (Mitsubishi Chemical Co., Japan) (Kawai et al., 1996; Kobayashi and Tazaki, 1997; Fukuuchi et al., 2001) was administered at 60 mg/day for the first 2 days and 20 mg/day for the next 3 days by infusion. Ticlopidine 200 mg/day was prescribed thereafter. Ten days after admission, a rehabilitation program was started and measures of activity improved markedly within a week. He was discharged after 18 days hospitalization.

One month after the onset, he started to feel the return of thermal sense in his left lower extremity while taking a bath. The recovery of pain and thermal sense below the T10 level was confirmed by careful self examination. However, sensation above T10 level has remained analgesic and thermoanesthetic.

About 7 weeks after onset he returned to his outpatient clinic for part-time work. When he held a cup of hot tea with

his left hand on that day, he felt an electric pain sensation after a 4 or 5 s interval and could not continue holding it. This phenomena is consistent with the withdrawal reaction which was documented by Rousseaux et al. (Rousseaux et al., 1999). This condition still persists for objects with a temperature above 50°C.

Around 3 months after the start of disease, when he had tried to pull up the roof of a convertible, he felt a sharp, stabbing pain in his whole left upper limb. This corresponds with movement allodynia as described by Bowsher (Bowsher, 1996). The following day he tested his muscles in various ways including doing push-ups, tensing muscle groups and simply putting pressure on muscles in the analgesic area and found that they all provoked allodynic pain. No pain was felt with passive joint movements.

The most annoying problem that started to occur after 4 months was a continuous unpleasant freezing pain, which occasionally changes to a burning pain, in the lateral angle of the right eye and the left forearm, hand and foot, particularly in the medial sole. Warming alleviates these pains but cold environments exacerbate them. These symptoms are consistent with central post-stroke pain (CPSP).

An additional problem that started after 4 months is an unpleasant acute sensation when touching cold objects suddenly in the analgesic areas. Unexpected touch or rubbing movements with an object of moderate temperature also causes an unpleasant feeling. These allodynic sensations are felt in the left upper extremity and above the T10 level of the body. Other remaining symptoms are hypesthesia, analgesia and thermoanesthesia in the right half of the face, right sided Horner's syndrome, mild right sided ataxia and occasional choking over fluid intake.

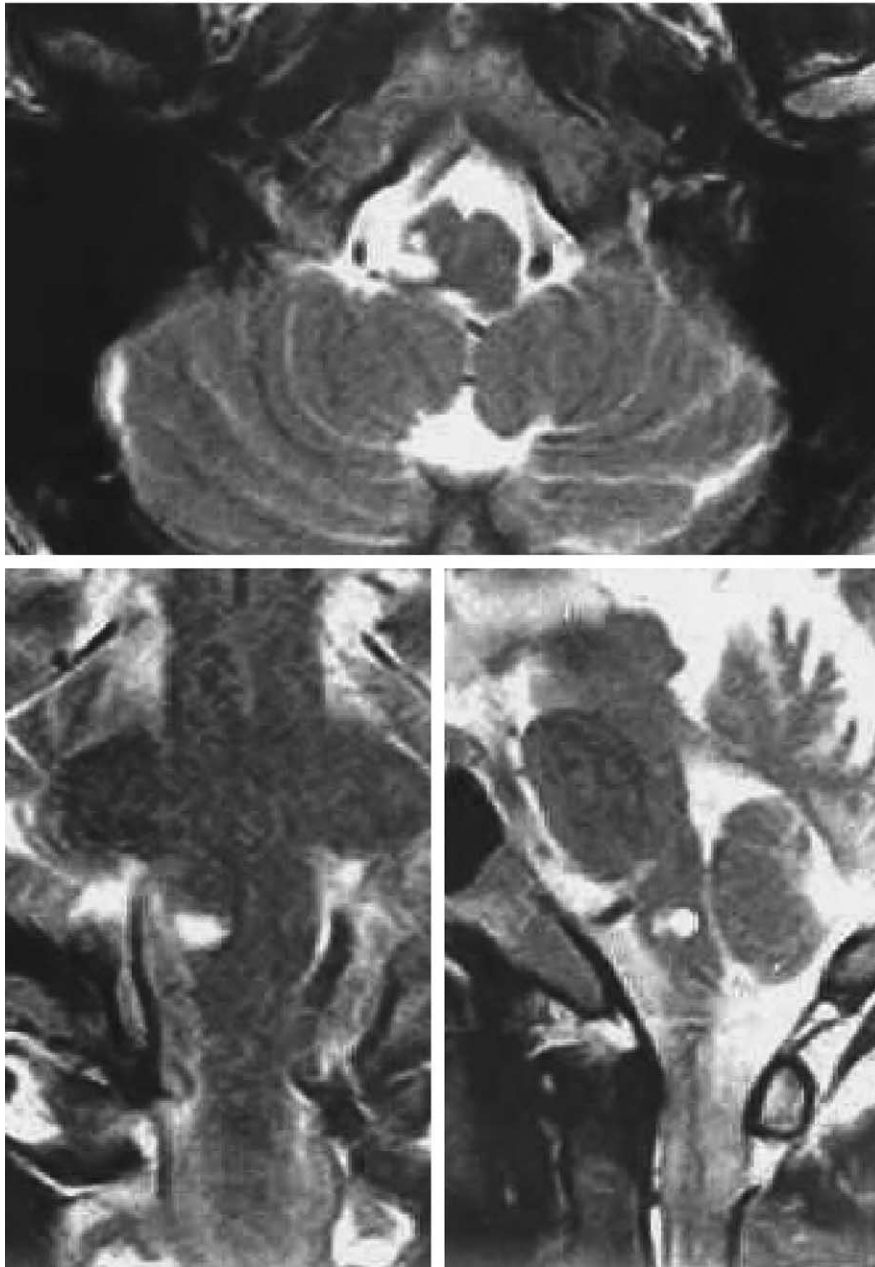


Fig. 2. Axial, coronal and sagittal views of T2 weighted image of the lesion 4 months later.

The axial, coronal and sagittal views of T2 weighted image of the lesion about 4 months after the onset are shown in Fig. 2.

### 3. Discussion

The dissociated loss of pain and temperature sensation in the ipsilateral face and contralateral limbs and trunk, indicates my disease is consistent with classic Wallenberg's syndrome. These 'typical' Wallenberg's syndrome symptoms are not always experienced by all lateral medullary infarction patients. According to recent clinical magnetic

resonance imaging correlation studies (Sacco et al., 1993; Vuilleumier et al., 1995; Kim et al., 1997; Bowsher et al., 1998; Brochier et al., 1999; Cerrato et al., 2000), areas of infarction vary greatly therefore, neurological symptoms may be equally variable.

The fact that my symptoms of ataxia and dissociated thermoalgnesia sensation have decreased over time, might be due to the recovery of the penumbra zone due to reduction of edema and associated inflammatory change. Recovery of thermoalgnesia sensation in the left lower quadrant of the body might also be explained this way. The lesion in this case is somewhat medially and dorsally located, despite the involvement of the posterior spinocerebellar tract, the

spinothalamic tract and the descending nucleus of the trigeminal nerve may account for the residual symptoms.

Hypesthesia in the right half of the face may imply involvement of subnucleus caudalis (Fitzek et al., 2001). Unstable blood pressure during admission suggests the involvement of the nucleus fasciculus solitarius (Phillips et al., 2000).

The incidence of CPSP in all stroke patients was found to be up to 8% according to Andersen et al. (Andersen et al., 1995), whereas Bowsher estimated the rate to be at least 11% in aged patients (Bowsher, 2001). MacGowan et al. reported chronic CPSP syndrome in 16 out of 63 patients (25%) with lateral medullary infarct syndromes within 6 months of onset (MacGowan et al., 1997). All 16 CPSP patients in this series had constant pain with intermittent allodynic exacerbation by innocuous stimuli; more commonly after cold than tactile stimuli. Bowsher recorded allodynia in 72% of patients with CPSP after cerebro-vascular accidents (Bowsher, 1996). Bowsher also discussed movement allodynia, which seems not to have been described previously. Fitzek et al. described eight out of 12 patients as having developed CPSP between 12 days and 24 months after lateral medullary infarction (Fitzek et al., 2001).

I propose that movement allodynia be described more suitably as muscle-allodynia or allomyodynia. This is different again from conventional allodynia caused by dermal stimulation because even passive pressure on muscles provokes an allodynic sensation.

The other phenomenon of withdrawal reaction I experienced is just as described by Rousseaux (Rousseaux et al., 1999). This is the electric pain and the unpleasant reaction I felt when holding objects whose temperatures are  $\geq 50^{\circ}\text{C}$ .

In patients with severe spinothalamic injury, noxious thermal stimulation can still be perceived via extra spinothalamic pathways. The author (S.K.) supposes this phenomenon is one type of thermal allodynia.

There have not been enough of these symptom descriptions published, because the appearance of these symptoms is often delayed for several months after onset. As a result, as Bowsher commented, many patients will be back in the community and beyond the reach of a neurologist or other hospital specialist by the time pain develops (Bowsher, 1993).

Fitzek et al. suggested that facial pain after lateral medullary infarction is due to a lesion of the lower spinal trigeminal tract, leading to differentiation of the spinal trigeminal nucleus neurons (Fitzek et al., 2001). In PET studies, Peyron et al. showed thalamic overactivity which might explain abnormal transduction and amplification of sensory inputs after spinothalamic differentiation (Peyron et al., 1998). I feel some impulses are conveyed through tactile sensation pathways since a cold atmosphere severely provokes CPSP. Peyron et al. also reported abnormal circulate responses after induced allodynia.

Early use of a first generation anti-depressant drug such as

amitriptyline is recommended to alleviate pain (Leijon and Boivie, 1989; Bowsher, 1995). In my case, amitriptyline was not helpful, nor indeed tolerable, because of its side effects. Mexiletine, however, appeared to have some effect (Awerbuch and Sandyk, 1990; Bowsher, 1995) and it is my experience that the topical non-steroidal anti-inflammatory agent Felbinac temporarily alleviates the effects of freezing pain.

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