A 73-year-old man, with a one month past history of bilateral cerebellar infarct, presented with a continuous 4 Hz “no-no” type head tremor at rest, with increasing amplitude while maintaining posture or during voluntary movements. The tremor disappeared during sleep. The family history of tremor or dystonia was negative. On the neurological examination, his speech was dysarthric and he was unable to walk or sit because of a severe gait ataxia. The patient was quite ataxic on bilateral finger to nose and heel to knee testing. Upward gaze of both eyes was impaired. There were no coexistent symptoms of dystonia. Brain computed tomography showed a bilateral infarct in the superior cerebellar artery territory. He was treated with 1000 mg levodopa daily associated to clonazepam at the dosage of 1 mg three times a day with a slight improvement.

Tremor, as a manifestation of stroke, is an uncommon neurologic manifestation. Isolated head tremor without upper and lower limb tremor, like in our case, is even more infrequent [1]. The head tremor presented here resembles Holmes tremor (HT). HT, also known asrubralor midbrain tremor, is an irregular rest tremor with a slow frequency below 4.5 Hz. It is characterized by its enhancement while maintaining posture or during voluntary intentional movements. The precise mechanism remains unknown and requires elucidation. However, any lesion that occurs within the cerebello-thalamo-cortical circuit or the dentato-rubro-olivarypathway “Guillain-Mollaret triangle” could result in HT [2]. Magnetic Resonance Imaging may show, within four or six months, hyper intensity of the olive in the medulla oblongata due to hypertrophic olivary degeneration, which is considered as trans-synaptic degeneration subsequent to lesions in the “Guillain-Mollaret triangle” [3]. No explanation is available why the tremor appeared only in the head. Probably, cerebellar representations of head and cervical muscles were exclusively eliminated by the ischemic lesion. The third cranial nerve dysfunction is probably due to third nerve nucleus infarction. A delay of few weeks to 8 years between the lesion and tremor onset was reported [4]. Pharmacological treatment of HT is usually unsuccessful, although isolated responses to levodopa have been reported [5]. Stereotactic surgery such as pallidotomy and deep brain stimulation or repeated injections of botulinum toxin has been reported to cause improvement in some cases [6].

REFERENCES