
NEUROIMAGING OF THE CNS INVASION OF GNATHOSTOMA SPINIGERUM AND ANGIOSTRONGYLUS CANTONENSIS IN A NORTHERN THAI PATIENT

Patchrin PEKANAN¹, Sirintara PONGPECH¹,
Siriporn HIRUNPAT², Suthipong JONGJIRASIRI¹, Sophon KUMPOLPUNTH¹.

ABSTRACT

A case of mixed parasitic infestation of the CNS was demonstrated by CT scan, MRI scan and angiography. The patient was 51-yr-old female farmer presented with neurological deficits. Gnathostomiasis and Angiostrongyliasis was responsible for the symptoms. Multiple hematomas are seen in the spinal cord, central canal and in the brain. Dissection of the vertebral artery was also noted. Most of the changes represented the manifestation of Gnathostomiasis.

INTRODUCTION

Gnathostoma spinigerum is a tissue nematode with an exceptionally tremendous penetrating power. It can migrate through any anatomical structure of the human body, except bone. *G. spinigerum* third stage larvae have been recovered from various organs such as the urinary bladder, uterus, intestine, lungs, ears, eyes, spinal cord and brain (1). Common neurological syndromes, i.e. subarachnoid hemorrhage, meningitis, encephalitis, transverse myelitis, ascending myelitis or radiculomyeloencephalitis can be caused by invasion of the parasite into the central nervous system (1). The mortality rate of CNS gnathostomiasis was approximately 20 percent and one of the major causes of death was direct parasite invasion into vital

centers in the brain stem.

Various snails, slugs, prawns and crabs are intermediate hosts of the *Angiostrongylus cantonensis*. Humans are usually infected by eating infected intermediate hosts, that have not been properly cooked. In Thailand *Pila* snails are the main source of infections, the snails are often served in Thailand as a delicacy together with alcohol (2,3). In humans the parasite does not complete its life cycle and dies, for example, within the brain. Here it provokes a marked inflammatory response, the main clinical manifestation being eosinophilic meningitis (2,4)

We presented a case of mixed CNS infection of the two parasites, demonstrated by CT, MRI and angiographic imaging.

¹ Department of Radiology, Ramathibodi Hospital, Rama 6 Street, Bangkok 10400, Thailand.

² Department of Radiology, Prince of Songkla University Hospital Haadyai, Songkla, Thailand.

CASE REPORT

A fifty-one years old female from Pijit Province, came in with weakness of the lower extremities for one week. The weakness began firstly from the right side and then progressively towards the left side. She was drowsy. There was bilateral cranial nerve VI palsy and stiffneck. The sphincter tone was loose. The motor power of the lower extremities were grade 0, while the upper extremities showed grade 4-5. The CSF was sent to DR. Stitaya Sirisingh Laboratory, Department of Microbiology, Faculty of Sciences, Mahidol University. It was positive for Gnathostomiasis and Angiostrongyliasis with O.D. 1.71 and 0.59 respectively (positive cut-off point = OD. + 0.20). The serum was also positive for the two mentioned parasites with OD. 1.8 and 0.46 respectively.

CT scan of the brain showed subarachnoid hemorrhage (Fig. 1). MRI of the brain showed

multiple small intracerebral hematomas at pons, left posteroinferior part of the cerebellum and right cerebellar hemisphere. Large hemorrhagic infarct was noted at left parieto-temporo-occipital area (Fig. 2). The left vertebral artery was not visualized by MRA (Fig. 3).

MRI of the whole spine showed subdural hematoma at posterior aspect of C6 to T10. A hemorrhagic tract was seen along the right paramedian posterior aspect of C5 and C5-6, left paramedian area of C4 and C3. Associated cord edema was seen from CT to C6.

Angiography of the left vertebral artery showed dissection of this artery (Fig. 3).

Mild degree of hydrocephalus was present and CSF shunt was installed. The patient had hospital acquired infection. She was discharged and referred to the hospital in Pijit Province. She was quadriplegia and unable to communicate, at the discharge-time.

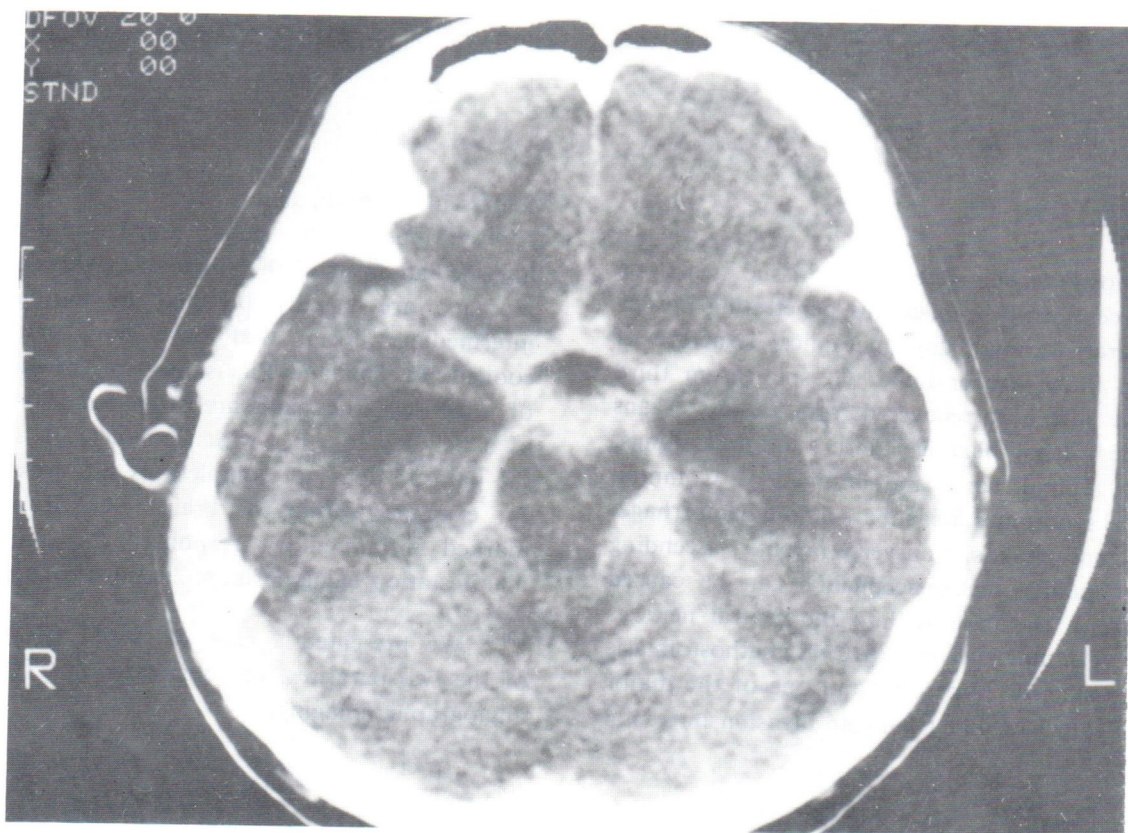


Fig. 1 Non i.v. enhanced CT scan of the brain at the cut level of the suprasellar cistern showed subarachnoid hemorrhage.

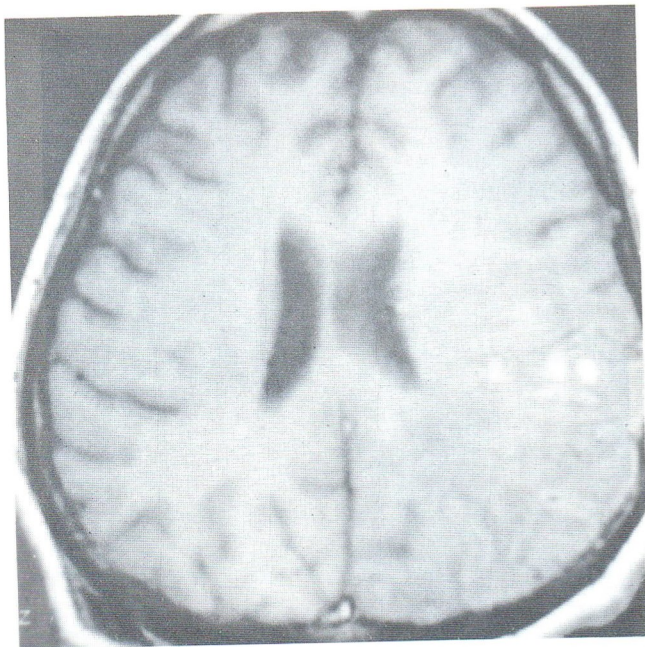
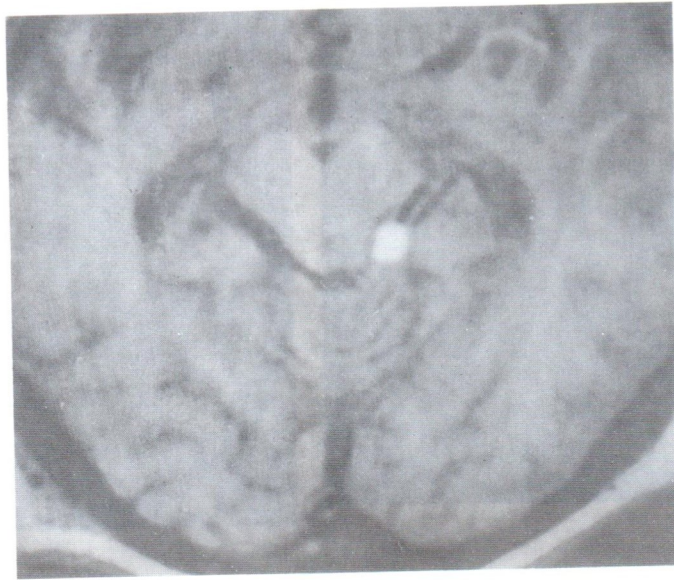


Fig. 2A T1WI-axial view MRI of the brain shows multiple small subacute hemorrhagic areas at left quadrigeminal cistern, and left parietal lobe.

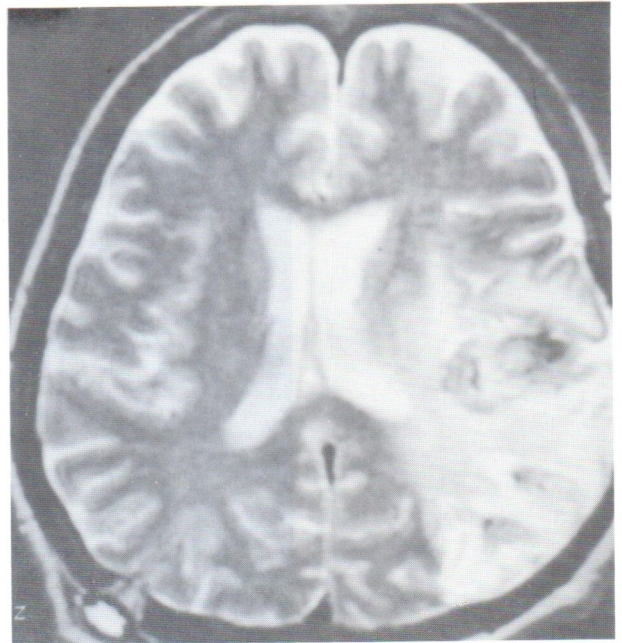


Fig. 2B T2WI-axial view MRI of the brain showed ischemic areas at postero-inferior aspect of both cerebellum and left parietal lobe, left basal ganglia with hemorrhagic component.



Fig. 3 MRA of the intracranial vessels showed non-visualized left vertebral artery.

Fig. 4 Dissection of left vertebral artery was seen at left vertebral angiographic injection.

DISCUSSION

The first evidence of nervous system invasion by *Gnathostoma spinigerum* was demonstrated by Chitanondh and Rosen in 1967 (1,5). They found a gnathostome larva embedded in the thoracocervical segment of the spinal cord of a 37-year-old Thai housewife who died of fatal eosinophilic encephalomyelitis. Adult male and female gnathostomes live in the stomach wall of definitive hosts such as the cat, dog, tiger and leopard. A *G. spinigerum* egg is extruded into the stomach, excreted and hatches in fresh water into the first stage larva. Three larval stages and two intermediate hosts are needed in order to complete the whole life cycle. Man is apparently an accidental host acquiring the parasite by consuming raw or inadequately cooked food which harbours *G. spinigerum* third stage larvae (1).

To gain access into the central nervous system, they have to go through bone openings or foramina for nerve roots, nerves, and/or blood vessels or perhaps go directly into the arterial supply system of the brain and the spinal cord. Multiple hemorrhagic tracts are the most important pathognomonic findings. These tracts may be widely distributed in the whole axis of the central nervous system or heavily concentrated in a certain segment of the nervous system such as the spinal cord in case of extensive damage, hematoma have been found in the cerebrum, cerebellum, nerve roots, and cauda equina (1,6). Massive intracerebral hematoma can be the primary cause of death (7). Secondary subarachnoid hemorrhage can either be mild, or severe with intraventricular clots (7). Microscopic examination of recent parasitic tracts reveals only hemorrhage, with none or very few cellular responses. In older lesions, microcavitation, tissue necrosis, swollen axis cylinders with phagocytosis and perivascular infiltration are seen. Cellular infiltrates may be predominantly eosinophils or other mononuclear cells, such as plasma cells, lymphocytes, and macrophages. The brain, if involved, is edematous and congested, with cellular infiltration extended to the covering meninges (1).

Paraplegia is more common than quadriplegia or triplegia. Monoplegia is also noted. The variation of weakness and sensory deficits are shown. Urinary retention is always the rule in case of radiculomyelitis of radiculomyeloencephalitis. Multiple cranial nerve palsies are noted in the encephalic form. All the cranial nerves from the second to the twelfth have

been involved. Cranial nerve palsies commonly begin after paralysis of the extremities.

Five patients suffering from angiostrongyliasis were reported by Schmutzhard (2). All five presented with the signs and symptoms of meningitis, and one patient presented with bilateral abducens nerve palsy and papilledema of the left eye. No death occurred in this group.

Many helminths have been reported to be able to invade the CNS and cause a wide variety of neurological signs and symptoms. These are, besides *Gnathostoma spinigerum* and *Angiostrongylus cantonensis*, *Strongyloides stercoralis* (8), *Trichinella spiralis* (9), *Toxocara canis* (10), *Lagochilascaris minor* (11), *Baylisascaris procyonis* (12), *Anisakis* spp (13), *Paragonimus westermanni* (14), *P. mexicanus*, *Schistosoma haematobium*, *S. japonicum*, *S. mansoni* (15), *Echinococcus granulosus* (16), *E. multilocularis*, *Cysticercus cellulosae* (17), *Spirometra mansonoides* (18).

Our presented case was the mixed infection of *Gnathostoma spinigerum* and *Angiostrongylus cantonensis*. The clinical picture was dominated by the firstly mentioned nematode, which produced hemorrhagic incidence. The 6th nerve palsy could be seen in both parasitic infestation.

It is seldom to see the images as shown by us in this condition.

REFERENCES

1. Boongird P, Vejjajiva A. Gnathostomiasis. *Travel Medicine International* 1994;12:214-219.
2. Schmutzhard E, Boongird P, Vejjajiva A. Eosinophilic meningitis and radiculomyelitis in Thailand, caused by CNS invasion of *Gnathostoma spinigerum* and *Angiostrongylus cantonensis*. *Journal of Neurology, Neurosurgery, and Psychiatry* 1988;51:80-87.
3. Punyagupta S, Bunnag T, Juttijudata P, Rosen L. Eosinophilic meningitis in Thailand. Epidemiologic studies of 484 typical cases and the etiologic role of *Angiostrongylus cantonensis*. *Am J Trop Med Hyg* 1970;19:950-58.
4. Punyagupta S, Juttijudata P, Bunnag T. Eosinophilic meningitis in Thailand. Clinical studies of 484 typical cases probable caused by *Angiostrongylus cantonensis*. *Am J Trop Med Hyg* 1975;24:924-31.
5. Chitanondh H, Rosen L. Fatal eosinophilic encephalomyelitis caused by nematode

- Gnathostome spinigerum. *Am J Trop Med Hyg* 1967;16:638-45.
6. Bunnag T, Comer DS, Punyagupta S. Eosinophilic myeloencephalitis caused by *Gnathostoma spinigerum*. *Neuropathology of nine cases. J Neurol Sci* 1970;10:419-34.
 7. Punyagupta S, Jittijudata P, Bunnag T, Comer Ds. Two fatal cases of eosinophilic myeloencephalitis: a newly recognized disease caused by *Gnathostoma spinigerum*. *Trans Roy Soc Trop Med Hyg* 1968;62:801-9;
 8. Scowden EB, Schaffner W, Stone WJ. Overwhelming strongyloidiasis: an unappreciated opportunistic infection. *Medicine* 1978;57:527-44.
 9. Most H, Abeles MM. Trichiniasis involving the nervous system. *Arch Neurol Psychiatr* 1937; 37:589-616.
 10. Woodruff AW. Toxocariasis. *Br. Med J* 1970; 2:589-616.
 11. Rosenberg S, Lopes MBS, Masuda Z, Campos R, Viera Bressan MCR. Fatal encephalopathy due to *Lagochilascaris minor* infection. *Am J Trop Med Hyg* 1986;35:575-8.
 12. Fox AS, Boyer KM. Fatal eosinophilic meningoencephalitis and visceral larva migrans caused by the racoon ascarid. *Baylisuscaris procyonis*. *N Engl J Med* 1985;312:1619-23.
 13. Khalil LF. Larval nematodes in the herring (*Clupea harengus*) from British coastal waters and adjacent territories. *J Marine Biol Assoc UK* 1969;49:641-59.
 14. Oh SJ Cerebral paragonimiasis. *Trans Am Neurol Assoc* 1967;92:275-7.
 15. Scrimgeour EM, Gajdusek DC. Involvement of the central nervous system in *Schistosoma mansoni* and *S. haematobium* infection. *Brain* 1985;108:1023-38.
 16. McCorkell SJ, Lewall DB. Computed tomography of intracerebral echinococcal cysts in children. *J Comp Assist Tomogr* 1985;9:514-8.
 17. McGormic GF, Zee CS, Heiden J. Cysticercosis cerebri, review of 127 cases. *Arch Neurol* 1982;39:534-9.
 18. Fan KJ, Pezeshkpour GH. Cerebral sparganosis. *Neurology* 1986;36:1249-51.