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The fate of spargana inoculated into the cat brain and sequential changes of anti-sparganum IgG antibody levels in the cerebrospinal fluid†

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Abstract: To establish an animal model of intracranial sparganosis, the fate and behavior of the experimentally inoculated spargana were observed. A total of 102 scolices of spargana were injected into 22 cat brains, and the cats were sacrificed at 2 weeks, 1 month, 3 months and 6 months after the inoculation. Neurosparganosis was established in 77% of the cats. Of 43 recovered worms, 19 (44%) were located in the subdural or subarachnoid space, 16 (37%) in the brain parenchyme, and 2 (5%) in the lateral ventricle. One was detected at the diploic space of the skull and 5 were outside the cranial cavity. All but one were alive, and had grown tails. They were distributed in the brain parenchyme randomly. There was no place which they could not invade. No adult was found in the intestine. Cerebrospinal fluid (CSF) was collected before inoculation, 1 week, 2 weeks, 1 month, 3 months and 6 months after inoculation. The level of anti-sparganum IgG antibody in CSF measured by ELISA began to increase above the criteria of positivity 1 month after inoculation. Three months after inoculation, the values markedly increased.

The present findings reveal that intracranial inoculation of spargana into the brains of cats would be a good animal model of experimental neurosparganosis.

Key words: sparganum, cat, brain, experimental sparganosis, worm recovery, ELISA

INTRODUCTION

Sparganosis is a disease caused by the plerocercoid larvae of *Spirometra* spp., which are usually located in the subcutaneous tissues or muscles. In Asia, *Spirometra erinacei* is the inflicted species (Mueller, 1974; Lee *et al.*, 1984). In rare occasions the worms get into the cranial cavity causing headache, seizure, or neurological deficits. Since Takeuchi reported an autopsy case of cerebral sparganosis in 1918, more than 20 cases of human intracranial sparganosis had been described in the literature (Mineura and Mori, 1980; Kim *et al.*, 1981; Anders *et al.*, 1984; Hong *et al.*, 1985; Fan and Pezeshkpour, 1986; Chang *et al.*, 1987; Lee *et al.*, 1987; Youm *et al.*, 1987; Anegawa *et al.*, 1989). Recently, the cases of intracranial sparganosis are increasingly detected because of advanced diagnostic methods such as high reso-

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lution computerized tomography, magnetic resonance imaging and enzyme linked immunosorbent assay (ELISA) (Kim *et al.*, 1984; Chang *et al.*, 1987).

The human cases are infested by 1) ingestion of frogs or snakes infected by the plerocercoid larva, 2) ingestion of water contaminated with *Cyclops* which harbors the procercoid larva, 3) topical application of flesh of infected frogs or snakes to the wounds, inflamed skin, eye, vagina, etc., and 4) ingestion of the plerocercoid larva through vehicles (Cho *et al.*, 1975). In Korea, 4% of frogs and 60% of snakes are infested by the plerocercoid larvae of *Spirometra* (Kim and Shin, 1975; Cho *et al.*, 1982; Kim, 1983). Moreover, the survival training in the ranger corps and the misbelief in the medicinal effect of snake make the sparganosis prevalent in Korea (Cho *et al.*, 1975). Among the 25 cases of intracranial sparganosis in the literature, 15 including seven surgically proven cases, were reported by Korean authors.

The route of migration of the worm from the intestine to the cranial cavity is still unknown. Also any experimental animal model has not been developed yet. The development of the model will give basic informations for understanding the nature of the disease itself and for the better diagnosis. The present study was performed to observe the fate of the spargana inoculated into the cranial cavity and to evaluate the feasibility of an experimental model of intracranial sparganosis. Spargana were inoculated into the brain parenchyme of cats and the recovery rate, distribution of spargana and the sequential change of cerebrospinal fluid (CSF) levels of anti-sparganum IgG antibody were observed.

MATERIALS AND METHODS

1. Cats and spargana: Twenty-five adult mongrel cats were used (Table 1). They were all egg negative for *S. erinacei* on stool examination. Spargana were taken from the subcutaneous tissue of snakes, *Rhabdophis trigrina*

Table 1. Number of cats used in the experiment

Group	No. of cats used for	
	Autopsy	ELISA
Control	—	25
1 week	—	21
2 weeks	5	20
1 month	8	4
3 months	8	7
6 months	1	1
Total	22	78

tigrina, collected at Sancheong-Gun, Kyungsangnam-do from July to September, 1987.

2. Preparation of spargana: Spargana were placed in warm saline. Worms with active movement were chosen, and their 0.5 cm long scolex proper was cut. The scoleces were rinsed several times in sterile saline.

3. Inoculation of spargana into the brain parenchyme: Cats were anesthesized by intramuscular injection of 25∼30 mg/kg of ketamine (Ketalar, Yuhan Corp., Korea) and 0.025 mg/kg of atropine (Atropine, Daewon Pharm. Co., Korea). The head was fixed on a stereotaxic frame. With a 23 gauge needle, cisterna magna was punctured and 2 ml of CSF was taken. After midline scalp incision, a small hole of 3 mm in diameter was made on the skull at 2 cm posterior to the coronal suture and 8 mm right to the midline. With an 18 gauge needle, three to five scoleces of spargana were inoculated with saline of under 2 ml into the brain parenchyme about 5 mm deep to the cortical surface. The scalp wound was sutured.

4. Sampling of CSF: CSF was collected by cisternal puncture one week, two weeks, one month, three months, and six months after the inoculation until the cats were sacrificed. At one month after inoculation, some of the cats were not subjected to the CSF sampling because of poor general conditions.

5. Observation of worm recovery: Cats were sacrificed with intravenous injection of potassium chloride. The cats were examined for spargana. Their whole brains were sliced with

5 mm thickness from the frontal lobe, and all parts of the subcutaneous tissue, muscle fasciae, upper half of spinal cord and intraspinal cavity, lungs and thoracic cavity, gastrointestinal tracts and peritoneal cavity were examined for the worm.

6. Sampling of serum: Just after the sacrifice of the cats, 5 ml of blood was withdrawn through cardiac puncture. The sera were collected after clotting and frozen at −70°C until used.

7. Micro-ELISA: The conventional micro-ELISA for the anti-sparganum IgG antibody was performed (McLaren *et al.*, 1978). One gram of spargana obtained from snakes were homogenized at 4°C with a tissue homogenizer. It was centrifuged with 10,000 g 24 hours after homogenization and the supernatant was frozen at below −20°C. Protein content of the saline extract was 0.312 mg/ml by Lowry method. The antigen was thawed and used after 1 : 800 dilution with carbonate buffer(pH 9.6). Antigen coating and further reactions were carried out in the wells of micro-ELISA plates (Titertek, U.S.A.). The CSF or sera were used under 1 : 800 dilution after chequerboard titration. Peroxidase conjugated anti-cat IgG (H and L chain specific) goat serum (Cappel Lab., U.S.A.) was used at 1:4,000 dilution as conjugate, while o-phenylenediamine (2 mg in 10 ml phosphate citrate buffer, PCB) was used with hydrogen peroxide (H_2O_2, 4 μl in 10 ml PCB) as substrate. After stopping the reaction with 8 N sulfuric acid, the absorbance was read at 492 nm with an ELISA reader(Titertek, U.S.A.).

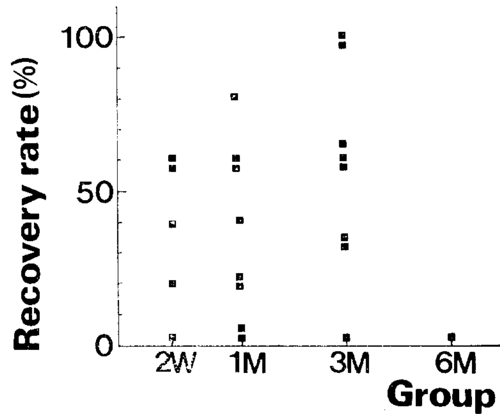

Fig. 1. Recovery rate of worms from each cat.

RESULTS

1. Recovery and distribution of spargana

Spargana were detected in 17 of the 22 examined cats. The overall recovery rate of the inoculated worms was 42% (43/102). The recovery rates of individual cat were variable from 0 to 100% (Fig. 1). The frequent sites of recovery were the subdural or subarachnoid space (19 worms) and the brain parenchyme (16 worms) (Tables 2 & 3, Figs. 2 & 3).

The worms were in the subdural or subarachnoid space diffusely, such as olfactory cistern, suprasellar cistern, subtemporal area, suboccipital area, interhemispheric fissure, cerebral convexity area, etc. One worm was dead, but others were grown up to the length of 1.5~4 cm. They were moving in characteristic waves in warm saline.

Parenchymal involvements by the worms were

Table 2. Recovery of the worms by time and location

Group	No. of cats	Total No. of inoculated worms	No. of positive cats	No. of recovered worms from*					
				V	P	SD/SA	Skull	EC	Total recovery rate (%)
2 weeks	5	25	4	0	2	7	0	0	9(36)
1 month	8	40	6	1	8	3	0	2	14(35)
3 months	8	32	7	1	6	9	1	3	20(63)
6 months	1	5	0	0	0	0	0	0	0 (0)
Total (%)	22 (100)	102 (100)	17 (77)	2 (5)	16 (37)	19 (44)	1 (2)	5 (12)	43(42) (100)

*V=ventricle, P=parenchyme, SD/SA=subdural or subarachnoid, EC=extracranial

Table 3. Distribution of spargana in cats after inoculation

Location	Group			Total
	2 weeks	1 month	3 months	
Subdural/Subarachnoid	7	3	9	19
Periolfactory	2	0	1	3
Suprasellar	1	0	0	1
Subtemporal	1	1	2	4
Suboccipital	0	2	1	3
Interhemispheric	2	0	0	2
Convexity	1	0	5	6
Parenchymal	2	8	6	16
Hemispheric white matter	1	1	2	4
Internal capsule	0	3	0	3
Basal ganglia*	0	2	0	2
Thalamus*	1	1	2	4
Hippocampal formation*	0	1	0	1
Cerebellum	0	0	1	1
Brain stem	0	0	1	1
Ventricular	0	1	1	2
Diploic	0	0	1	1
Extracranial	0	2	3	5
Scalp	0	2	0	2
Back & chest wall	0	0	3	3

*The worms were usually located at the junctions of these structures and the adjacent white matters.

observed at the thalamus, internal capsule, cerebral hemisphere, caudate nucleus, hippocampal formation, cerebellum and middle cerebellar peduncle. There was no predilection site. However, the worms were more frequently found around the thalamus rather than posterior hemispheric area where the worms were inoculated. Two worms were recovered from lateral ventricle. And one worm was found at the diploic space of skull 5 mm apart from the hole made for the inoculation of worms. Five worms were detected at the extracranial tissue such as back muscles, chest wall, and the subcutaneous tissue of scalp.

There found no relationship between the location of worm and the duration of infestation. Spargana migrated significantly in the brain after the experimental inoculation.

2. Sequential change of specific IgG antibody in CSF

The absorbances at 492 nm (IgG antibody level) of CSF taken before inoculation, 1 week, 2 weeks, 1 month, 3 months and 6 months after inoculation were 0.131±0.033, 0.141±0.028, 0.155±0.036, 0.278±0.073, 0.655±0.345, 0.289, respectively (Table 4 & Fig. 4). From 2 weeks after inoculation the absorbance increased significantly ($p<0.05$). The absorbances of serum ELISA were plotted in the Fig. 4. Even though the size of sample was too small, they

Table 4. Results of ELISA of cerebrospinal fluid

Group	Absorbance (mean±S.D.)*
Control	0.131±0.033
1 week	0.141±0.028
2 weeks**	0.155±0.036
1 month**	0.278±0.073
3 months***	0.655±0.345
6 months	0.289

*O.D.=optical density
S.D.=standard deviation
**$p<0.05$ on paired t-test
***$p<0.01$ on paired t-test
absorbance of blank; 0.113

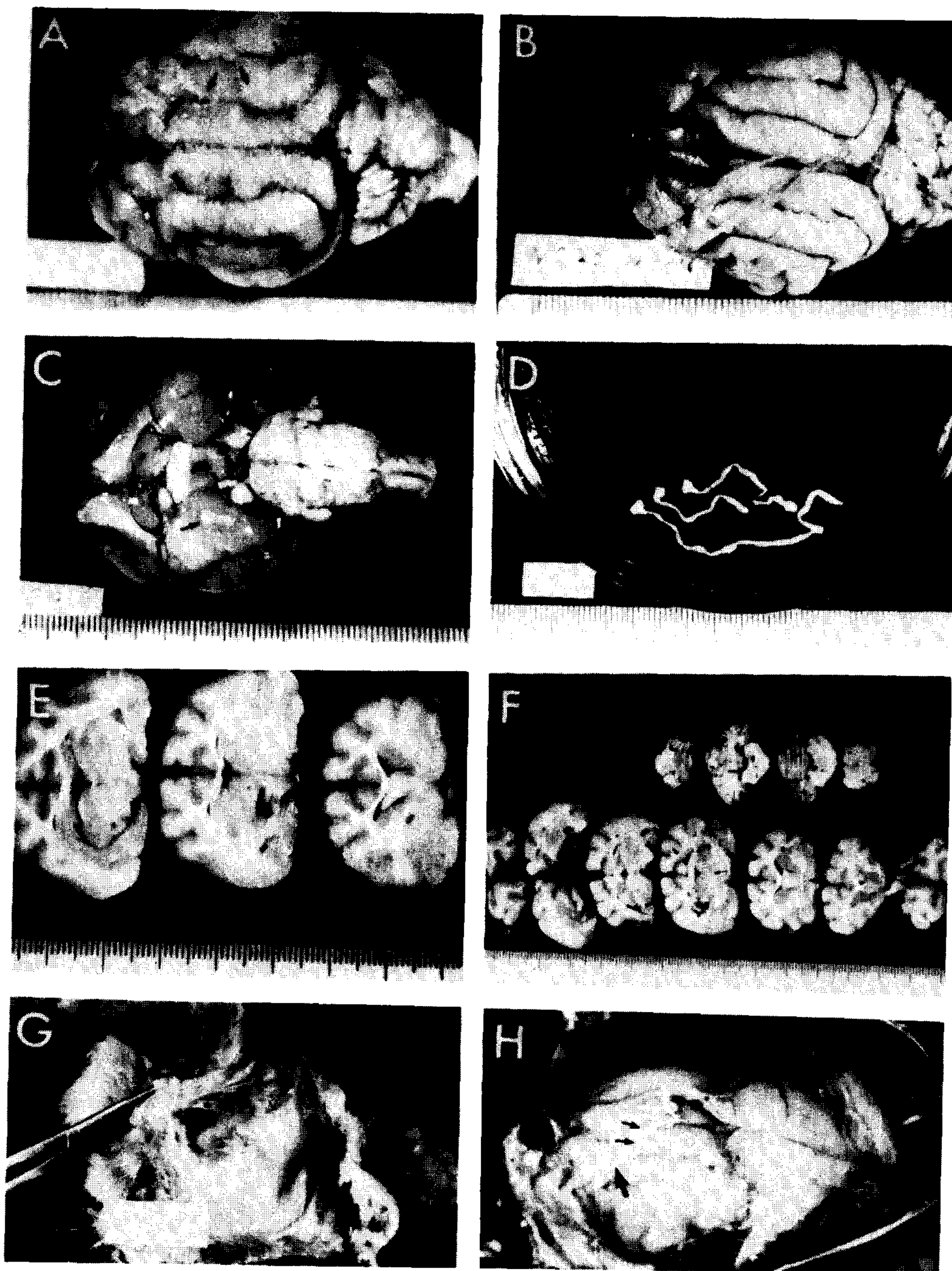

Fig. 2. Gross findings of the brain and recovered worms. A; (3 months after inoculation) a worm on the suprasylvian gyrus (arrows), B; (2 weeks after inoculation) a worm in the interhemispheric fissure (arrow), C; (3 months after inoculation) a worm beneath the temporal lobe (arrow), D; (3 months after inoculation) growing worms with tail from the subdural space, E; (2 weeks after inoculation) a worm in the thalamus and worm tracts anterior and posterior to the worm, F; (1 month after inoculation) worms in the internal capsules of both sides and tracts including worms in the brain stem and the cerebellum, G; (3 months after inoculation) a worm recovered from the scalp (arrow), H; (3 months after inoculation) a worm recovered from the diploic space of the skull (small arrows) 5 mm apart from the hole made for the inoculation of the worms (large arrow).

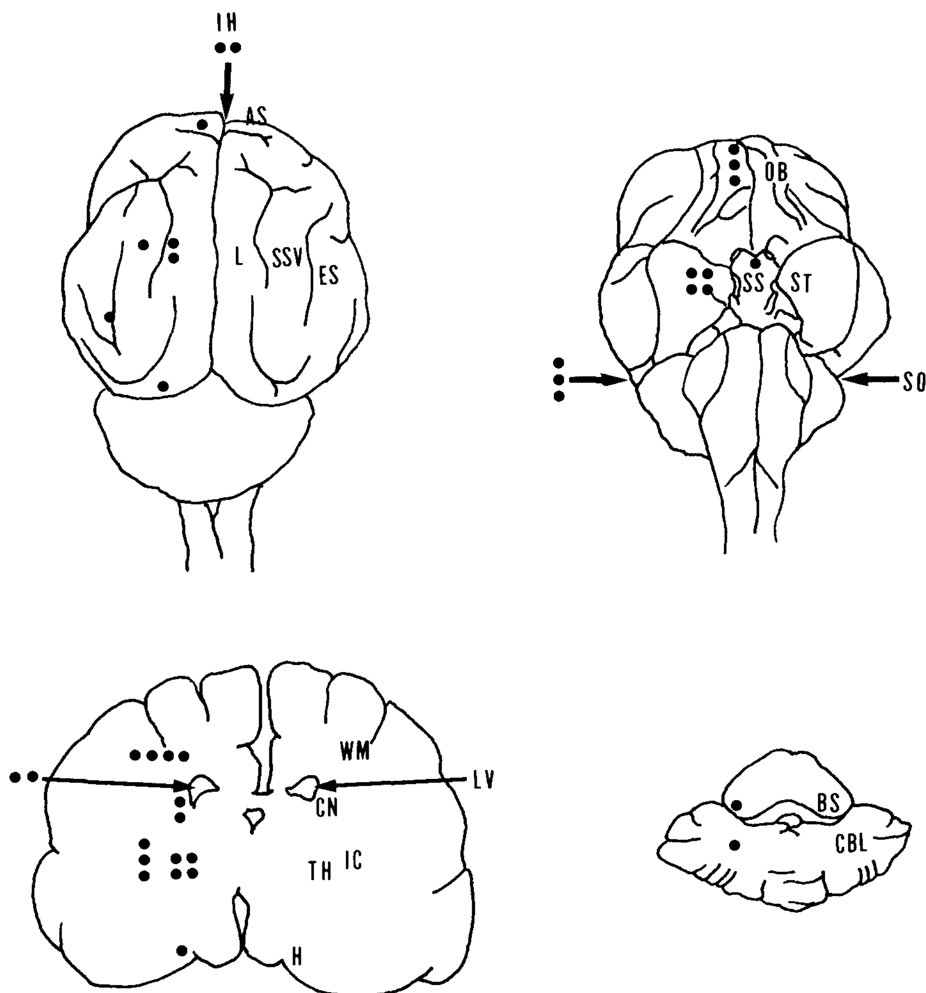

Fig. 3. Schematic drawing of the distribution of total recovered worms. Upper; worms in the subdural/ subarachnoid (IH=interhemispheric fissure, AS=anterior sigmoid gyrus, L=lateral gyrus, SSV= suprasylvian gyrus, ES=ectosylvian gyrus, OB=olfactory bulb, SS=suprasellar cistern, ST= subtemporal, SO=suboccipital), Lower; intraparenchymal worms (WM=hemispheric white matter, LV=lateral ventricle, CN=caudate nucleus, IC=internal capsule, TH=thalamus, H=hippocampal formation, BS=brain stem, CBL=cerebellum)

also showed increasing tendency after two weeks.

DISCUSSION

Cats were selected for establishment of experimental neurosparganosis because the anatomy of the cat brain is familiar and also because the brain size of cats permits a bolus injection of about 1 ml saline solution with spargana. Also repeated CSF samplings are possible. However, this cat model had some limitations. First, the cat is a natural definitive host of *Spirometra* to harbour an adult worm in the intestine. Therefore, sparganosis of cats is unnatural and very rare. Cats are infected by the sparganum only when the cat ingested procercoid larvae (Corkum, 1973) or when the plerocercoids were inoculated intraperitoneally(Beaver *et al.*, 1984). In such circumstances, spargana were in the subcutaneous tissue or skeletal muscle just same as in humans. In the present study, the possibility of migration into the intestine and growth to adults were conjectured. However, no adults were detected from the intestine of the cats.

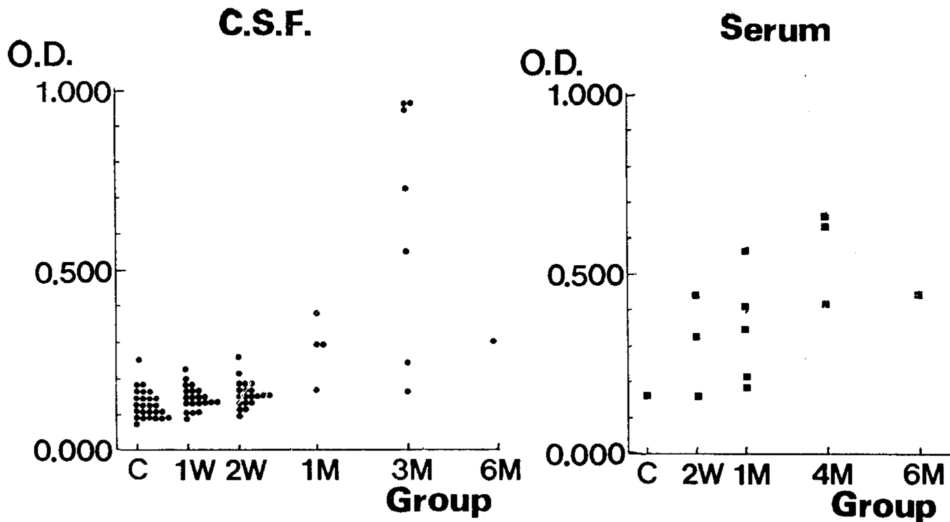

Fig. 4. Results of ELISA on the cerebrospinal fluid (CSF, left) and the serum (right)

Second, intracerebral inoculation is not a natural route of sparganosis. In sparganosis of mammals, the worms usually enter the host via the mouth and penetrate into the peritoneal cavity through the intestinal wall (Chi *et al.*, 1980; Choi, 1984). Most of the larvae dwell in the subcutaneous tissue or skeletal muscle. The frequency of cerebral invasion is very rare, but such an event can still occur probably due to their activity of tissue penetration. To overcome the rarity of spontaneous intracranial sparganosis, we had to inoculate the larvae intracranially. We tried to minimize the volume of one injection bolus. Otherwise, the cat should suffer from sudden increase of the intracranial pressure. Two cats died just after the injection probably because of the abruptly increased intracranial pressure and another died because of epidural hematoma. Also this procedure should include deep anesthesia and drilling the skull.

Third, complete dissection of the whole carcass of all cats was very difficult. We searched all parts of the brain, upper half of the spinal cord and intraspinal cavity, subcutaneous tissue, fascia of muscles, brain, lungs and thoracic cavity, gastrointestinal tracts and peritoneal cavity. This procedure required lots of time. The total low recovery rate, 42%, and the wide fluctuation of the rates by the individual cats from 0% to 100% in the present study were partly due to the difficulty in detection of spargana. Theoretically the recovery rates must be 100%. Then where had the 58% of the inoculated worms gone? The recovery rates in experimental subcutaneous sparganosis of mice with oral inoculation were from 72% to 100% (Hong *et al.*, 1989). The recovery rates of the present study should be interpreted as the minimum recovery.

All of the worms except one were alive and showed evident growth. Nearly half (44%) of recovered worms were in the subdural or subarachnoid spaces. This location might be a result of migration through the injection tract from the brain parenchyme in early phase especially in the two week group as seven out of nine recovered worms were detected in these spaces. To the contrary, majority of the worms, eight of 14, were in the parenchyme of the brain in the one month group, but more worms were in the subdural or subarachnoid space again than in the brain parenchyme in the three month group. This finding might suggest the possibility that the worms could migrate between the parenchyme and the subdural or subarachnoid space. In the distribution of worms within the intracranial spaces or in the brain parenchyme, any

tendency of predilection sites was not observed. The larvae showed random distribution in all intracranial parts including the ventricle. They could reach at the cerebellum and brain stem. Furthermore, the migration tracts were observed in almost all parts of the brain parenchyme. No worm was found in the spinal cord although the subdural and subarachnoid spaces are connected with those of the brain. We could not examine lower half of the spinal cord because of the extreme impregnability. This part might be one of the plausible hiding spaces for the unrecovered worms. Also the base of the skull was the next hiding location because the worms looked migrating through the olfactory bulb to enter the skull base. We could not search the worms in that portion.

A total of five larvae were found outside the brain. Two of them were in the scalp, which migrated through the injecting hole of the skull. Three worms were recovered from the subcutaneous tissue of the back or chest wall. This result showed the fact that the sparganum inoculated into the brain parenchyme could escape from intracranial location though the route of their migration into the subcutaneous tissue from the brain parenchyme was not clear. One worm was in the diploic space of skull near the hole made for injection. Sparganum can penetrate every tissue except the cortical bone in mammals.

ELISA is a sensitive and specific serodiagnostic method for subcutaneous or cerebral sparganosis (Kim *et al.*, 1984; Chang *et al.*, 1987). The present study revealed false negative serology of CSF during the first two weeks and increased level of anti-sparganum IgG antibody from one month after infection just same as those found in the subcutaneous sparganosis of mice (Hong *et al.*, 1989). The specific IgG antibody in cerebral cysticercosis was found mainly produced in the local granulation tissue of the lesion (Cho *et al.*, 1988; Han *et al.*, 1988). This may be also plausible because the level of anti-sparganum IgG antibody in the CSF was higher than that in the serum in the three month group the lesions of which were formed of granulation tissue (Wang, by personal experience). However, the cat of six month group showed higher level of IgG antibody in the serum than that in the CSF. The serum level was higher than the positive criteria level of absorbance 0.22 while the level in the CSF was near the borderline. This suggested that the sparganum might be in the body of the cat but not in the brain although the worm was not found by the inspection of the brain and body. The present serological data proved that chronologic pattern of production of anti-sparganum IgG antibody in intracranial sparganosis was the same as that in subcutaneous sparganosis or in other tissue helminthiases (Choi *et al.*, 1986; Hong *et al.*, 1989).

This model showed 77% establishment rate of experimental neurosparganosis. Total 42% of the inoculated spargana were recovered and 37% of the recovered worms were in the brain parenchyme. The present model may be a promising scheme for further studies on neurosparganosis, such as histopathological, radiological, etc.

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=국문초록=

고양이 뇌에 주입된 스파르가눔의 운명과 숙주 뇌척수액 IgG 항체가의 경시적 변화

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고양이 두개강 내에 주입된 스파르가눔의 형태와 운명을 파악하고 숙주의 항체형성 양상을 관찰하고자 102마리의 스파르가눔을 22마리의 고양이의 뇌실질 내에 주입하고 2주, 1개월, 3개월, 6개월 후에 도살하여 충체의 회수율 및 분포를 육안으로 확인하였고 스파르가눔 감염전, 감염후 1주, 2주, 1개월, 3개월, 6개월의 뇌척수액을 얻어 효소면역법(ELISA)을 이용하여 IgG 항체가를 측정하여 다음의 결과를 얻었다.

1. 부검을 시행한 고양이 22마리 중 17마리에서 충체가 회수되었고, 뇌에 주입한 충체 총 102마리 중 43마리(42%)를 회수하였다. 충체의 두개강 내 분포는 경막하 또는 지주막하 공간에서 19마리(44%)가 검출되어 가장 많았고, 다음으로 뇌실질 내 16마리(37%), 측뇌실 내 2마리(5%)의 순이었다. 두개골의 판간층(diploic space)에 위치한 것이 1마리 (2%)이었으며 두개골 외에서도 5마리(12%)가 관찰되었다. 이들 충체는 한 마리를 제외하고 모두 생존하고 있었으며 상당히 성장하여 있었다. 뇌에서 충체의 분포는 특별한 편중이 없이 전 부위에서 고르게 관찰되었고 척수에서는 검출되지 않았다.

2. ELISA를 이용하여 측정한 뇌척수액 내 IgG 항체가는 감염후 1개월부터 양성기준치 이상으로 증가하기 시작하여 감염후 3개월에는 현저하게 증가되었다.

고양이를 이용한 실험적 두개강 내 스파르가눔증은 주입한 충체의 약 40% 정도가 두개강 내에서 회수되므로 몇가지의 제약에도 불구하고 이 질병의 연구에 유용한 모델로 판단된다.

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