Kawasaki Med. J. Vol. 2: No. 1, 35-50, 1976

ACUTE NECROTIC MYELOPATHY ASSOCIATED WITH ACUTE DISSEMINATED ENCEPHALOMYELITIS: CASE REPORT WITH POSTMORTEM STUDY

Teruo SHIRABE, Sadaaki OKAMOTO and Masahiro YOSHIMURA

Division of Neuropathology, Department of Human Pathology, Kawasaki Medical School, Kurashiki, 701-01, Japan

Accepted for publication on Nov. 1, 1975

Abstract

A fatal case is described of a 32-year-old male with acute necrotic myelopathy unusually associated with acute disseminated encephalomyelitis. The patient developed rapidly progressive quadriplegia, urinary retention and subsequent unconsciousness in the later stage, preceded by a few days of fever, headache, back pain, vertigo, tinnitus and blurred vision. He died on the thirteenth day of his illness, complicated by bronchopneumonia. Postmortem examination revealed transverse necrosis with small hemorrhages extensively in the cervical, thoracic and, of milder degree, lumbar cord. Axons as well as myelins were almost completely destroyed with proliferation of small amounts of lipid-laden macrophages and scanty perivascular infiltrates of lymphocytes in severely involved areas of the cord. In addition, inflammatory demyelinations were scattered perivascularly throughout the cerebral hemispheres, cerebellum, brain stem and optic nerves. This case is assumed to be an example of acute disseminated encephalomyelitis with special involvement of the spinal cord from the clinical course and pathological features. The pathogenesis of acute necrotic myelopathy is discussed together with brief review of similar cases hitherto described.

INTRODUCTION

Although acute necrotic myelopathy, otherwise referred to as progressive necrotic myelopathy[1], acute necrotic myelitis, acute transverse myelitis or myelopathy[2], acute spinal cord necrosis, acute myelomalacia and so on, may occur without evidence of any obvious cause, it may also come about apparently due to insufficiency of vascular supply to the spinal cord. Moreover, even though this syndrome may naturally be encountered with definite intramedullary and extramedullary disorders such as cord tumor, direct cord trauma or radiation myelopathy[3], lumbar

anesthesia may cause unexpectedly acute necrosis of the spinal cord. In addition, this disorder may occur in conjunction with measles, mumps[4], herpes simplex[5] or varicella-zoster[6,7,8] and following smallpox or rabies vaccination. While, in a small number of patients, acute necrotic myelopathy represents the first bout of multiple sclerosis or a part phenomenon of neuromyelitis optica. This illness may also take place only rarely in association with acute disseminated encephalomyelitis. The purpose of this paper is to present an another example of this unusual association of acute necrotic myelopathy and acute disseminated encephalomyelitis, and to discuss about the pathogenesis of acute necrotic myelopathy together with brief review of similar cases as yet reported with this condition.

CASE REPORT

Clinical history

A 32-year-old clerk was admitted as an emergency to the Kawasaki Hospital on May 26, 1968, because of fever, headache, back pain, blurred vision, vertigo and tinnitus.

He had been in good health until May 20, 1968, when he had a general fatigue with a temperature of 37.5°C, but he continued to work. Next day, his temperature rose to 38.5°, and at the same time he noted headache and back pain. Vertigo and tinnitus appeared on May 24 along with vomiting. General fatigue became more severe and he felt weak in the four extremities. He complained of blurred vision and diplopia in the morning of the Kawasaki Hospital admission.

The patient had a history of acute glomerulonephritis at 11 year old, pulmonary tuberculosis at age 14 and acute hepatitis at 18 year old. He had undergone operations for sinusitis and hemorrhoids at 19 year old. He had not contracted any exanthematous disease nor received any vaccination just before taken ill. His family history was non-contributory.

On admission, his development and nutrition were rather fair. He had fever of 39°C, a pulse of 72 per minute and a blood pressure of 130/90 mmHg. The palpebral conjunctivae were injected. The tongue was whitely coated. The heart, lungs and abdomen were physically unremarkable.

Neurologically, he was alert. The pupils were isocoric and reacted promptly to light. Visual acuity was almost normal on beside testing, although he complained of blurred vision. The neck was stiff and there was a positive Kernig's sign. Gross power of the extremities was suffi-

cient. Deep tendon reflexes were normal and Babinski signs were not obtained.

The urine gave a + test for both protein and glucose, 3+ test for urobilinogen; the sediment contained 3 to 5 red cells and 10 to 15 white cells per high-power field. The feces showed a 3+ test for occult blood but no eggs of parasites. Examination of the peripheral blood revealed a red-cell count of 3.78 million, a hemoglobin of 10.0 g/dl, a hematocrit of 38 % and a white-cell count of 5,600 with normal differential count. Erythrocyte sedimentation rate was 7 mm per hour and 15 mm per two hours. Serologic test for syphilis was negative. The serum protein was 6.53 g/dl, with 51.6 % of albumin, 6.9 % of α_1-globulin, 12.9 % of α_2-globulin, 11.4 % of β-globulin and 17.3 % of γ-globulin. Blood glucose was 120 mg/dl, serum cholesterol 162 mg/dl, serum glutamic oxaloacetic transaminase 5 units and serum glutamic pyruvic transaminase 2 units. Serum creatine was 1.22 mg/dl, amylase 82 units, non-protein nitrogen 28 mg/dl, blood urea nitrogen 14 mg/dl and cholinesterase 213 I.U./dl. Serum sodium was 133 mEq/l, potassium 3.3 mEq/l, chloride 90 mEq/l, phosphorus 1.3 mEq/l and calcium 4.3 mEq/l. C-reactive protein was 4+, rheumatoid arthritis test was negative, and antistreptolysin O was less than 100 units. X-ray films of the chest and electrocardiogram were normal.

A lumbar puncture disclosed an opening pressure of 370 mm of water. The cerebrospinal fluid was considerably xanthochromic and turbid, containing 223 mononuclear cells and 600 mg/dl of protein with glucose 25 mg/dl. Nonne-Apelt reaction was 4+, Pandy reaction was 4+, and tryptophan test was negative. Any becterium was not yielded on smear nor culture of the fluid.

On May 27, the second hospital day, the patient's temperature rose further up to 39.8°C. Repeatedly he complained of blurred vision. A feeling of weakness in the four extremities became terrible and urinary retention was noted. A few kinds of antibiotics were started, accompanied with intravenous dripping of adrenocorticotropic hormone. The catheter was inserted for the urinary retention. Overt weakness and sensory impairment developed in the lower extremities on May 28, other than high fever, nuchal rigidity and Kernig's sign. Adrenocorticosteroids were prescribed instead of adrenocorticotropic hormone from this day on. In the morning of the next day, the patient became rather drowsy. Pupillary light reaction was sluggish at this point. Breathing was rapid and abdomen was distended. In the evening of the day, the level of consciousness lapsed into semi-coma. Weakness and sensory impairment

ascended to the upper extremities. Artificial respiration was required, as his breathing became more shallow and cyanosis appeared. Spinal tap showed fluid of opening pressure of 510 mm of water, containing 512 mononuclear cells, 400 mg/dl of protein and 50 mg/dl of glucose. Adrenocorticosteroids and antibiotics were continued, and central nervous system activators were added into the drop instillation.

Physical examination on May 30 revealed a temperature of 38.8°C and a pulse of 130 per minute. Spontaneous breathing was not noticed. Neurologically, he was comatose. Light reaction was absent. Bilateral optic discs were somewhat pale. The neck and the four extremities were completely flaccid with no spontaneous movements. Deep tendon reflexes were not elicited, and nuchal rigidity and Kernig's sign were absent. An opening pressure of cerebrospinal fluid was 370 mm of water. The fluid contained 535 mononuclear cells, 450 mg/dl of protein and 25 mg/dl of glucese. Pulmonary function test showed pulmonary acidosis, with values of pH 7.11, pCO_2 120.3 mmHg and base excess +3.0 mEq/l. Bronchopneumonia developed at the left lung on May 31. Pyrexia, coma and apnea persisted. On June 1, the value of serum potassium was elevated to 5.9 mEq/l and that of serum chloride was reduced to 79 mEq/l. The patient died in the evening of the same day, 13 days after the onset of his illness and 5 days after the first signs of transverse myelopathy. The clinical course was diagrammatically illustrated in Fig. 1.

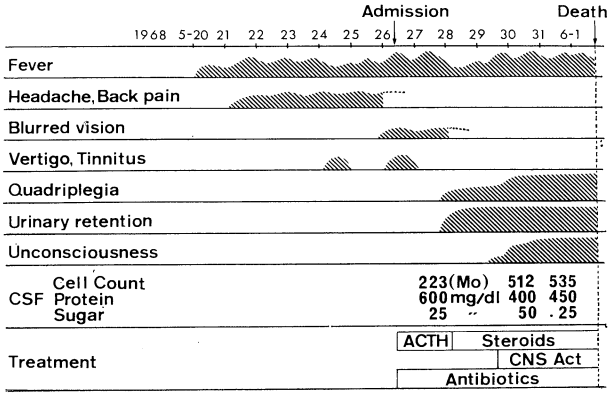

Fig. 1. Clinical course.

Neuropathological examination

The weight of the unfixed brain was 1,310 g. The cerebral hemi-

spheres were symmetrical and edematous with flattening of the gyri. The leptomeninges of the cerebral hemispheres were slightly congested and clouded with some minor hemorrhages in bilateral frontal lobe areas. There was no atherosclerosis in the blood vessels at the base of the brain. Coronal sections of the brain revealed slight congestion and edema in the cerebral parenchyma. The cerebellum, brain stem and optic nerves showed no abnormalities on the external appearance and cut slices.

The leptomeninges of the spinal cord were markedly congested, being scattered with many petechial hemorrhages. The parenchyma of the spinal cord was swollen and soft throughout the whole length from the upper cervical cord down to the lower thoracic cord. Horizontal sections of each segment disclosed transvere necrosis in the cervical and thoracic cord. Especially the lower cervical and upper thoracic cord was severely involved, where gray and white matters could not be apparently discriminated. The lumber cord was grayish with semi-transparent tinge in color, although without any obvious necrotic areas.

On microscopic examination, the cervical and thoracic cord was extensively necrotic, only sparing some parts of the gray matter and posterior funiculus of the cervical cord (Fig. 2 and 3). The tissue in this region was rough and spongy. No distinction could be discerned

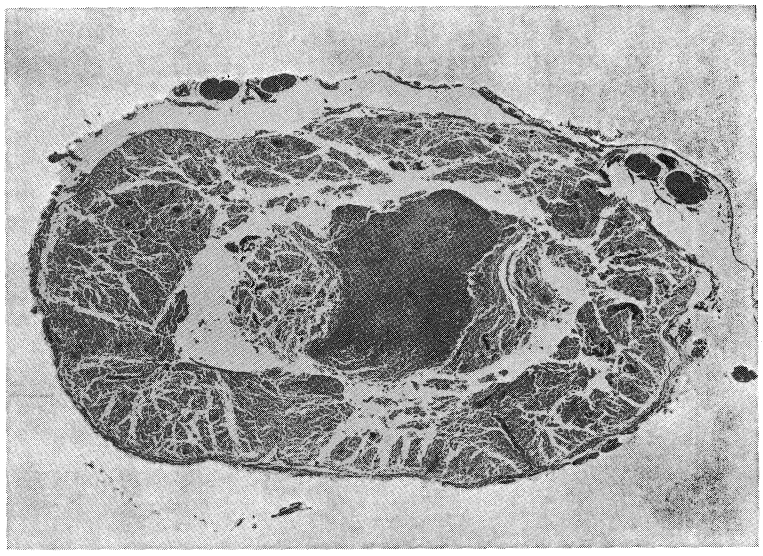

Fig. 2. Lower cervical cord demonstrating transverse necrosis, only sparing parts of the gray matter and posterior funiculus. Luxol fast blue and periodic acid-Schiff, ×8.3.

between gray and white matter. Most of the nerve cells had disappeared in the gray matter. Small amounts of microglial cells and lipid-laden macrophages were scattered in the involved areas (Fig. 4). Oligodendrocytes were not seen and astrocytic increase was not obvious. Occasionally small recent hemorrhages were present. There was a perivascular infiltration of the lymphocytes. Leptomeninges over the intensely affected areas were also infiltrated by a small number of lymphocytes, macrophages and, of rare occurrence, neutrophils. The lumbar cord was less severely involved. The gray matter and parts of the lateral and posterior funiculi were relatively well preserved, but the remaining areas of the lumbar cord had still similar histological features to the lesions in the cervical and thoracic segments. The ground substance was considerably loosened. Anterior horn cells were shrunken and reduced in number. Capillaries and small blood vessels of the spinal cord were mostly dilated and their endothelial cells were often hypertrophied. Thrombosis and gross atherosclerosis were not evident in any of the blood vessels related to the cord.

Perivascular demyelinations with lymphocytic infiltration and accumulation of macrophages were scattered in the optic nerves. Moreover, necrotic zones with hemorrhages were distributed along with the peri-

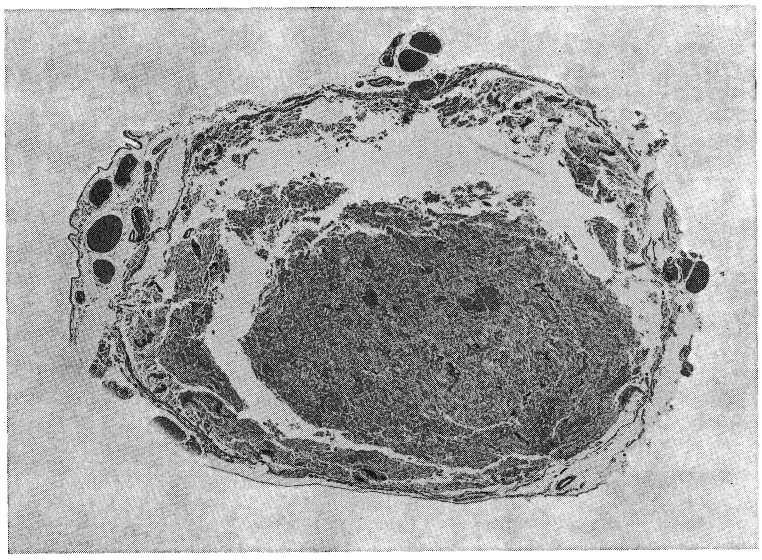

Fig. 3. Upper thoracic cord with complete transverse necrosis. Distinction between gray and white matter is not discernible. Luxol fast blue and periodic acid-Schiff, ×9.2.

phery of the optic nerves adjacent to the leptomeninges, where moderate amounts of lipid-laden macrophages and reactive protoplasmic astrocytes were observed (Fig. 5). Leptomeninges surrounding the optic nerves were fibrously thickened and infiltrated by lymphocytes also together with small hemorrhages.

Demyelinating foci of various sizes were randomly disseminated in many places of the brain stem such as midbrain tegmentum, cerebral peduncles, pontine basis, around the inferior olives and pyramids (Fig. 6). Not only myelins were severely destroyed, but axons were also intensely damaged with occasional axonal swelling in these lesions. Boundaries of the lesions were not always so sharply demarcated. Recent small hemorrhages were frequently coexistent, but lipid-laden macrophages were scarcely observed. Small demyelinating lesions were mostly perivascular together with perivascular accumulations of small amounts of macrophages, lymphocytes and plasma cells (Fig. 7). In addition, necrotic lesions with small hemorrhages and lymphocytic infiltration were located along with the marginal zones adjacent to the leptomeninges, especially of the cerebral peduncles and pontine basis. Lipid-laden macrophages were rather prominent in these places and slightly hyper-

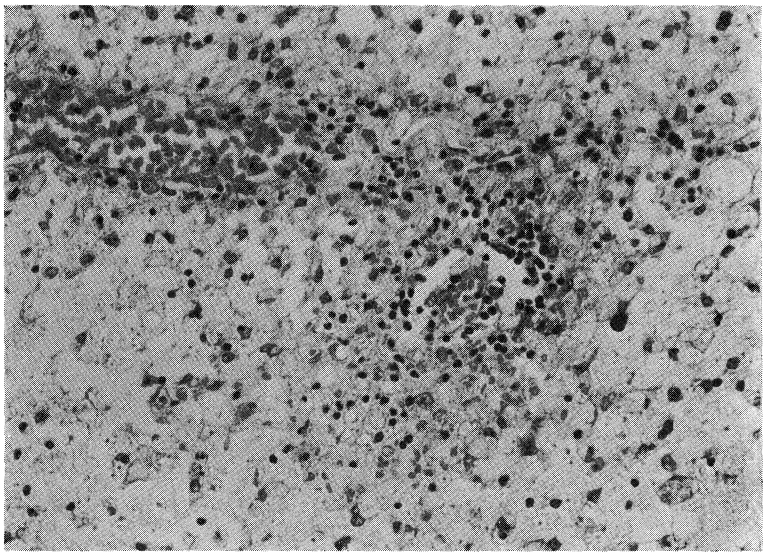

Fig. 4. Microscopic aspects of the necrotic upper thoracic cord. Proliferation of microglial cells and lipid-laden macrophages are observed. There is a perivascular infiltration of small amounts of lymphocytes. Hematoxylin and eosin, ×208.

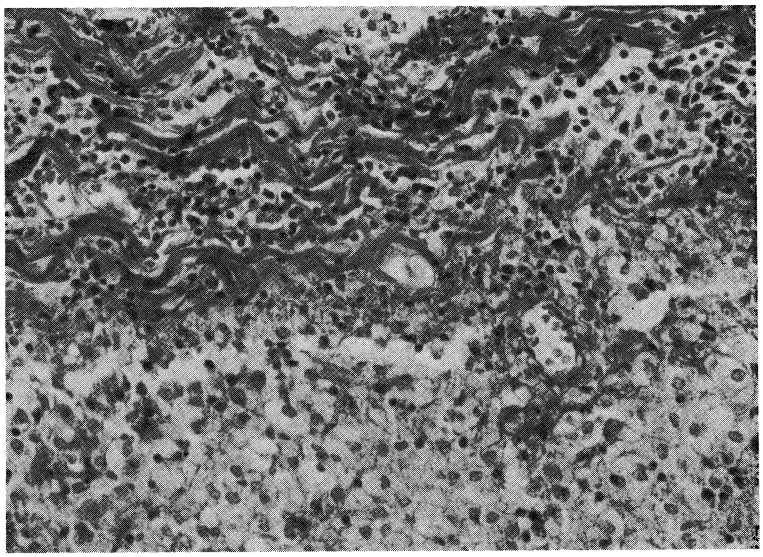

Fig. 5. Optic nerve showing meningeal infiltration by lymphocytes and necrosis of the parenchyma adjacent to the leptomeninges. Hematoxylin and eosin, ×208.

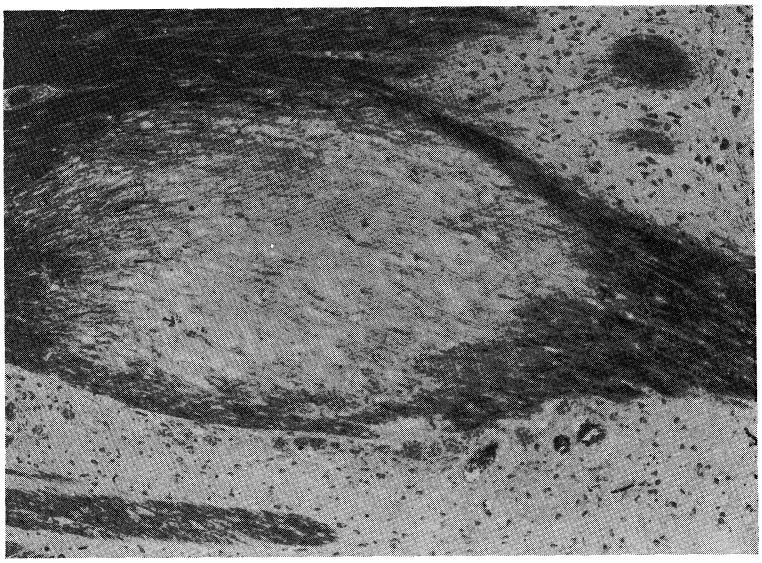

Fig. 6. Demyelinated focus in the pons. The border of the lesions is not so sharply defined. Luxol fast blue and cresyl violet, ×42.

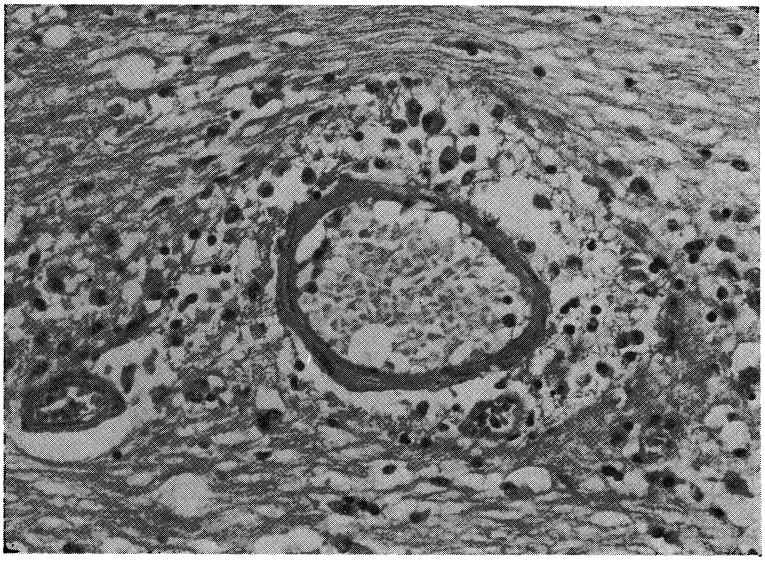

Fig. 7. Small perivascular lesions with perivascular accumulations of small amounts of macrophages, lymphocytes and plasma cells in the pons. Hematoxylin and eosin, ×208.

trophied protoplasmic astrocytes were proliferated to some degree. Most of the nerve cells in the lesions had fallen into necrosis and somewhat reduced in number, although there was no tendency such like that nerve cells of each cranial nerve nucleus, substantia nigra, pontine nuclei and inferior olives were specifically involved, nor there were not intranuclear inclusion bodies, glial nodules and neuronophagia.

The cerebellum showed perivascular infiltration of lymphocytes of mild degree and perivascularly localized demyelinations mainly in the white matter. Small amounts of lymphocytes and macrophages were recognized also in the leptomeninges of the cerebellum.

Lymphocytes were infiltrated perivascularly again in the basal ganglia and thalamus with occasional small perivascular demyelinations and small hemorrhages. The parenchyma had become generally loose, accompanied with small hemorrhages, lymphocytic infiltrates and appearance of macrophages, at the areas just beneath the ependymal cells of the lateral and third ventricles. A few protoplasmic astrocytes were proliferated in the regions. Nerve cells of the thalamic nuclei were frequently shrunken. Lymphocytes were accumulated mainly perivascularly at the choroid plexus.

Microglial cells were scattered diffusely in the cortex and white matter of the cerebral hemispheres, including the frontal, parietal, temporal and occipital lobes, apart from perivascular infiltration of lymphocytes with occasional perivascular demyelinated lesions. Cortical nerve cells appeared partially shrunken. In particular, pyramidal cells of the Sommer's sectors showed marked ischemic cell changes. Numerous areas of the first layer of the cerebral cortex underneath the pia mater had been softened with lymphocytic infiltration, and proliferations of lipid-laden macrophages and protoplasmic astrocytes of mild degree. The leptomeninges of the cerebral hemispheres contained frequent foci of perivascular infiltration of lymphocytes, plasma cells, monocytes and scanty neutrophils as well as proliferating macrophage. Still more, blood vessels of the leptomeninges were fairly congested and localized hemorrhages were occasionally observed.

The specimens from representative areas of the central nervous system were examined electron microscopically, but any virus particles were not demonstrated.

DISCUSSION

The patient described here developed rapidly progressive quadriplegia, urinary retention and, afterwards, coma following high fever, back pain, vertigo, vomiting and blurred vision. The whole course of his illness was thirteen days. Postmortem examination exhibited numerous foci of inflammatory demyelinations scattered perivascularly in the cerebral hemispheres, cerebellum, brain stem and optic nerves, while the spinal cord showed transverse necrosis extensively spreaded. Clinically, neuromyelitis optica was suggested at the time when quadriplegia occurred immediately after blurred vision. Yet continued high fever, leucocytosis in the cerebrospinal fluid and coma in the later stage had seemed more favorable for acute encephalitis. Pathological findings in the cerebral hemispheres, cerebellum, brain stem and optic nerves were most resembling to those of acute disseminated encephalomyelitis[9]. Blurred vision clinically observed might be explained by the fact that the lesions of acute disseminated encephalomyelitis had spreaded over the optic nerves as well. On the other hand, the spinal cord revealed extensive necrosis in the cervical and thoracic level. Axons as well as myelins in the gray and white matter of these segments had been almost indiscriminately destroyed together with shrinkage and loss of nerve cells. Still, intramedullary and extramedullary blood vessels of the spinal cord had been intact except for the endothelial hypertrophy of varying

degrees. These findings of the spinal cord seemed to coincide with those of acute necrotic myelopathy[10].

Both acute disseminated encephalomyelitis and acute necrotic myelopathy are respectively not supposed to be a disease entity of one etiology. Acute disseminated encephalomyelites[9] is considered to be a group of diseases in which symptoms and signs of acute encephalitis are encountered and pathologically innumerable foci of demyelinations are observed around the small vessels of the brain and spinal cord. Acute necrotic myelopathy[10] is, on the contrary, understood to be a general term of conditions showing clinical symptoms and signs of acute transverse myelopathy and pathological findings of complete necrosis with loss of all nervous elements of the spinal cord. The difference between the pathological findings of the brain identical with acute disseminated encephalomyelitis and those of the spinal cord equivalent to acute necrotic myelopathy in our patient may be attributed simply to separate reaction to the same etiology, when the clinical course is taken into account. Although the findings of the brain and those of the spinal cord were superficially quite different each other, the brain stem showed rather extensive demyelinated lesions as well as small perivascular demyelinations. These brain stem lesions were thought to be transitional features between the findings of the brain and those of the spinal cord. The presence of these transitional areas in the brain stem suggests, further, from the pathological point of view, that the changes of the brain and spinal cord may indicate merely the different reactive pattern to the same etiology.

From these pathological findings of the brain and spinal cord, ours might be apprehended to be a case of acute disseminated encephalomyelitis with special involvement of the spinal cord, or conversely acute necrotic myelopathy spreading perivascularly over the whole brain. Clinically, however, elements suggestive of encephalitis such as coma in the later stage were far more dominant only for acute necrotic myelopathy. From the pathological viewpoint also, as the lesions had been expanded all over the brain including the leptomeninges, the patient presented might be more pertinently considered to be a case of acute disseminated encephalomyelitis with special involvement to the spinal cord.

Acute necrotic myelopathy may occur not only as a partial sign of acute disseminated encephalomyelitis, but also as a first attack of multiple sclerosis or a part phenomenon of neuromyelitis optica. The spinal

cord lesions of acute multiple sclerosis or neuromyelitis optica are made up of extensive and confluent demyelinated lesions of the gray and white matter. Axons as well as myelins are destructed in the lesions often forming cavities. Nevertheless, in acute multiple sclerosis, demyelinated lesions of considerable extension are usually found also in the cerebral hemispheres, cerebellum and brain stem, especially periventricularly, other than demyelinations of the optic nerves and spinal cord. Sometimes differential diagnosis between neuromyelitis optica and necrotic myelitis comes into question[11,12]. It is difficult also to distinguish the spinal cord lesions of our case from those of acute multiple sclerosis or neuromyelitis optica only from the spinal cord, but it is pretty easy to discriminate this case from acute multiple sclerosis or neuromyelitis optica, if the lesions of the cerebral hemispheres, cerebellum, brain stem and optic nerves are compared.

Acute disseminated encephalomyelitis may be divided into three categories, namely, parainfectious, postvaccinal and idiopathic subgroups. Acute necrotic myelopathy is reported to occur accompanied with mumps or herpes infections, or after vaccinations. As our patient did not have any infections nor any vaccinations just before taken ill, he might belong to the idiopathic subgroup of the disease. In our case, however, viral infection might also be suspected from clinical features such as high fever and pleocytosis in the cerebrospinal fluid, and pathological findings such as perivascular and meningeal infiltrates of lymphocytes, other than allergic reaction which is said to be a pathogensis of acute disseminated encephalomyelitis. Acute necrotic myelopathy is known to occur on the occasion of infections of Coxsackie, influenza, Epstein-Barr, herpes simplex, varicella-zoster and polio viruses. And yet, the findings such as glial nodules, neuronophagia and inclusion bodies often seen in cases with viral infections were not encountered, nor there was no tendency to be involved especially in the nerve cells. Still more, electron microscopic examination failed to detect any virus particles. From these observations, therefore, viral infection is less likely in our case. But, as sufficient viral examinations have not been carried out, it is not conclusive as to the possibility of viral infection.

Acute necrotic myelopathy is considered to be a rare disease. Acute necrotic myelopathy associated with acute disseminated encephalomyelitis appears to be by far rarer, Only a few cases resembling to ours are documented. The patient described by Hoffman and Norman[13] had spinal symptoms of the second and third thoracic segments and died on the

26th day of neurological illness. Postmortem examination revealed acute necrosis and perivenous encephalomyelitis in the spinal cord, perivascular demyelinations in the cerebral hemispheres and optic nerves, and further, edematous necrosis in the cerebellum. They have mentioned that the edematous swelling of the tissue and compression of vessels following edema are responsible for the pathogenesis of the spinal cord softening. The case of Miller and Ramsden[14], which seems most closely resembling to our own, is a 20-year-old female with malignant nephrosclerosis. She developed acute transverse symptoms of the spinal cord accompanied with unconsciousness in the later stage and died after the course of 10 days. At autopsy, the findings of perivenous encephalomyelitis were found in the brain, and those of acute necrotic myelopathy, in the spinal cord. They proposed that hyperactive reaction to some kinds of bacterial toxins and following edema owing to secondary circulatory disturbance might be important for the etiopathogenesis of necrotic myelopathy. The second case of acute or subacute myelitis recorded by Wagner and his co-workers[15] are somewhat resembling to ours also. Theirs seemed to be a case of perivenous demyelinating encephalomyelitis with a tendency of spinal cord necrosis.

Other than in cases of acute necrotic myelopathy associated with acute disseminated encephalomyelitis, acute multiple sclerosis or neuromyelitis optica, spinal cord necrosis is known to bring about in cases with subacute necrotic myelitis reported by Foix and Alajouanine[16] in 1926. In this disorder, according to them, spinal cord symptoms and signs progress subacutely in one or two years, and pathologically spinal cord necrosis is present in accompany with considerable thickening of the walls of the intramedullary and extramedullary vessels of the spinal cord. Spinal cord necrosis in this condition is thought to be apparently due to circulatory disturbance following to vascular anomalities. Our case is undoubtedly different from this Foix-Alajouanine disease. After that, Greenfield and Turner[17] mentioned, in 1939, one case of acute and two of subacute necrotic myelitis. Of these, pathologic features of two cases of subacute necrotic myelitis are quite the same as those of Foix-Alajouanine disease, in which spinal cord lesions could be explained solely by circulatory disturbance. They have said that the lesions in a case of acute necrotic myelitis also appear to be altogether secondary ones to circulatory disturbance, because the condition consisted essentially of primary obliterative sclerosis of the small intramedullary and meningeal vessels in the lower segments of the spinal cord, and was

associated with great thickening of the walls of the larger meningeal veins and sometimes also of the larger arteries. Miyazaki et al.[18] advocated vascular occlusion as a cause of spinal cord necrosis in their article of acute necrotic myelopathy.

On the contrary to these views, Jaffe and Freeman[19], in 1943, suggested that an existence of some kinds of toxi-infectious causative factors and their tissue reactions might be important as the pathogenesis of the spinal cord necrosis, presenting four cases of spinal necrosis and softening of obscure origin with review of similar cases from the literature. They further predicted that this specific agent to occur necrosis will be found in the near future, although it has not yet identified at the present time. And further, Hassin and Broder[20] have discussed a 46-year-old male of acute myelitis, who developed paraplegia in association with a complete anesthesia up to the level of the eighth thoracic segment. They contended that inflammatory softening and necrosis had occurred in the course of some toxi-infectious disease process.

Later, in 1955, Kahle and Schaltenbrand[21] described eight cases with diffuse spinal cord lesions as myelitis necroticans diffusa. Their cases seem somewhat different from ours. The clinical course was longer and characteristically the spinal cord lesions had spreaded spindle-shapedly to upward and downward directions, centering aroud deep the posterior column. In the same year, Hoffman[22] had presented three cases of acute necrotic myelopathy with postmortem findings and two additional cases in which the patients survived a disease clinically indistinguishable from this condition, adding summarization of the literature. He suggested that circulatory disturbrance might not be participated in the production of the spinal cord necrosis, because of lack of significant alteration in the intra- or extramedullary blood vessels in not a few cases. Hoffman and Norman[13] have explained the mechanism of necrosis production as follows in another paper. At first, the toxic or infectious agent, particularly bacterial infection, may cause spinal cord edema, which would in turn raise the internal pressure of the spinal cord, as the spinal cord is surrounded closely by relatively inelastic pia mater. Then, capillaries or small veins would be compressed, resulting in succeeding obstruction of circulation. And finally, the liquefaction necrosis would be produced.

Recently, Veron and his associates[10] have investigated three cases of acute necrotic myelopathy and similar 28 cases from the literature. They have postulated that vascular abnormalities seem secondary and there is no obvious vascular etiology. Further, they have stressed that

this disorder seems to belong to demyelinating group of disease, in which vascular structure does not take part in the production of transverse necrosis.

As indicated above, there are various factors as the pathogenesis of spinal cord necrosis. In our case, because of lack of changes of the spinal cord vessels, the necrosis is difficult to be explained merely by mechanical obstruction of the blood supply to the spinal cord. Because there were findings of coexisting acute disseminated encephalomyelitis in the brain, allergic mechanism may be appropriately considered and secondary vascular disturbance due to edema of the cord would participate in the production of the spinal cord softening of our case as its pathogenesis.

REFERENCES

1. Follisn, A. G. H. and Netsky, M. G.: Progressive necrotic myelopathy. Handbook of Clinical Neurology, ed. by Vinken, P. J. and Bruyn, G. W. Vol. 9: 452-468, 1970, North Holland Pbl. Co., Amsterdam.
2. Altrocchi, P. H.: Acute transverse myelopathy. Arch. Neurol., 9: 111-119, 1963.
3. Marty, R. and Minckler, D. S.: Radiation myelitis simulating tumor. Arch. Neurol., 29: 352-354, 1973.
4. Benady, S., Zvi, A. B. and Szabo, G.: Transverse myelitis following mumps. Acta Pediat. Scand., 62: 205-206, 1973.
5. Klastersky, J., Cappel, R., Snoeck, J. M., Flament, J. and Thiry, L.: Ascending myelitis in association with herpes-simplex virus. New Engl. J. Med., 287: 182-184, 1972.
6. Hogan, E. L., Krigman, M. R. and Hill, C.; Herpes zoster myelitis. Evidence for viral invasion of spinal cord. Arch. Neurol., 29: 309-313, 1973.
7. Nigam, P., Tandon, V. K. and Kumar, R.: Varicella (Chickenpox) myelitis. J. Indian Med. Assoc.. 59: 520-521, 1972.
8. Nyberg-Hansen, R.: Relapsing herpes zoster myelitis. Acta Neurol. Scand. (Suppl.), 51: 397-400, 1972.
9. Alvord, E. C.: Acute disseminated encephalomyelitis and allergic neuroencephalopathies. Handbook of Clinical Neurology, ed. by Vinken, P. J. and Bruyn, G. W. Vol. 9: 500-571, 1970, North Holland Pbl. Co., Amsterdam.
10. Veron, J. P., Escourolle, R., Buge, A. and Castaigne, P.: Acute necrotic myelopathy. Europ. Neurol., 11: 83-96, 1974.
11. Ortiz de Zarate, J. C., Tamaroff, L., Sica, R. E. P. and Rodriguez, J. A.: Neuromyelities optica versus subacute necrotie myelitis. Part II. Anatomical study of two cases. J. Neurol. Neurosurg. Psychiat., 31: 641-645, 1968.
12. Tsukagoshi, H., Sugita, H., Miki, E., Harada, K. and Tsubaki, T.: An autopsy case of diffuse necrotic myelitis, with special reference to the difference between Foix-Alajouanine's disease and optic neuromyelitis. Clin. Neurol., 3: 367-375, 1963.
13. Hoffman, H. L. and Norman, R. M.: Acute necrotic myelopathy associated with perivenous encephalomyelitis. J. Neurol. Neurosurg. Psychiat., 27: 116-124, 1964.
14. Miller, A. A. and Ramsden, F.: Acute necrotic myelitis and perivenous encephalomyelitls associated with hypertension and renal infection. J. Clin. Path., 20: 821-825, 1967.

15. Wagner, B., Vuia, O. und Hager, H.: Beobachtungen zum Problem der nekrotisierenden Myelitiden. Zbl. allg. Path., 117: 375-388. 1973.
16. Foix, E. et Alajouanine, T.: La myélite nécrotique subaigue. Rev. Neurol., 33: 1-42, 1926.
17. Greenfield, J. G. and Turner, J. W. A.: Acute and subacute necrotic myelitis. Brain, 62: 227-252, 1939.
18. Miyazaki, M., Gemba, H. Fujita, M. and Yonezawa, T.: A case of necrotic myelopathy. Clin. Neurol., 13: 325-330, 1973.
19. Jaffe, L. D. and Freeman, W.: Spinal necrosis and softening of obscure origin. Necrotic myelitis versus myelomalacia; review of literature and clinicopathologic case studies. Arch. Neurol. Psychiat., 49: 683-707, 1943.
20. Hassin, G. B. and Broder, S. B.: Acute myelitis. A clinical-pathologic study. J. Neuropath. Exp. Neurol., 5: 106-115, 1946.
21. Kahle, W. und Schaltenbrand, G.: Zur Klinik und Pathologie der Myelitis necroticans diffusa. Deutsch. Ztschr. Nervenh., 173: 234-266, 1955.
22. Hoffman, H. L.: Acute necrotic myelopathy. Brain, 78: 377-393, 1955.