Acta Medica Okayama

Volume 39, *Issue* 1 1985 *Article* 9

FEBRUARY 1985

Cerebral edema associated with acute hepatic failure.

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Abstract

The clinicopathological findings of cerebral edema were investigated in patients with acute hepatic failure autopsied at Okayama University Hospital between 1970 and 1980 retrospectively. Nine (64%) of 14 hepatic failure cases were found to have cerebral edema during a post-mortem examination of the brain. Clinical features of the patients with cerebral edema were not significantly different from those of the patients without cerebral edema. However, general convulsions were observed more frequently in patients later found to have cerebral edema. Moreover, the length of time from deep coma to death was much shorter in the brain edema cases with cerebral herniation than without herniation.

KEYWORDS: acute hepatic failure, fulminant hepatitis, cerebral edema, neurological abnormalities

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*PMID: 3984783 [PubMed - indexed for MEDLINE]

Acta Med. Okayama **39,** (1), 73-75 (1985)

— BRIEF NOTE —

CEREBRAL EDEMA ASSOCIATED WITH ACUTE HEPATIC FAILURE

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Received July 23, 1984

Abstract. The clinicopathological findings of cerebral edema were investigated in patients with acute hepatic failure autopsied at Okayama University Hospital between 1970 and 1980 retrospectively. Nine (64 %) of 14 hepatic failure cases were found to have cerebral edema during a post-mortem examination of the brain. Clinical features of the patients with cerebral edema were not significantly different from those of the patients without cerebral edema. However, general convulsions were observed more frequently in patients later found to have cerebral edema. Moreover, the length of time from deep coma to death was much shorter in the brain edema cases with cerebral herniation than without herniation.

Key words : acute hepatic failure, fulminant hepatitis, cerebral edema, neurological abnormalities

Cerebral edema associated with acute hepatic failure is frequently lethal. The frequency of death has been reported to be 50 % (16 out of 32) by Ware *et al.* (1), 38 % (36/92) by Gazzard *et al.* (2) and 81 % (13/16) by Silk *et al.* (3). The low survival rate of patients with acute hepatic failure could be improved by controlling cerebral edema. The present paper was undertaken to clarify how frequently cerebral edema is associated with acute hepatic failure and how brain edema can be diagnosed and treated.

Post-mortem examination of the brain was carried out in 14 among 36 hepatic failure patients autopsied at Okayama University Hospital between 1970 and 1980. Cerebral edema was diagnosed in nine cases by macroscopic findings such as swollen cerebral hemispheres, narrowed gyri and flattened convolutions. In two of the 9 cases, cerebral edema was complicated with cerebral herniation. One was an uncal herniation and the other was a parahippocampal and tonsillar herniation. Clinical findings of the cases of cerebral edema, such as age, sex and biochemical examinations, were not significantly different from those of the cases without cerebral edema (Table 1). The frequency of high fever, tachycardia, distress of respiration and laterality of profound reflexes was similar in both types

TABLE 1. CLINICAL AND PATHOLOGICAL FINDINGS OF ACUTE HEPATIC FAILURE PATIENTS WITH CEREBRAL EDEMA

	Cerebral edema	
	Present	Absent
Number of cases	9	5
Age	46 ± 17	49 ± 17
Male : Female	4 : 5	2 : 3
Total bilirubin (mg/dl)	35 ± 10 (7)	33 ± 32
GPT (KU)	1990 ± 2270 (7)	920 ± 700
Blood ammonia (μg/dl)	273 ± 128 (5)	197 ± 84
Blood sugar (mg/dl)	244 ± 163 (8)	244 ± 148
Arterial PCO_2 (mmHg)	22 ± 5 (2)	40 ± 10
Liver weight (g)	842 ± 196	860 ± 196
Brain weight (g)	1373 ± 135 (8)	1275 ± 191
Lung congestion (%)	44	60
Adrenal atrophy (%)	56	40

( ) : Number of cases analyzed. Mean ± SD.

of cases, but general convulsion was more frequent in cases of cerebral edema. Most of the patients had received corticosteroid, glucagon-insulin therapy, blood transfusion, hemoperfusion and/or hemodialysis following admission, but hypertonic solutions such as mannitol for cerebral edema were not used. The types of therapy were unrelated to the development of cerebral edema. Corticosteroid therapy did not effectively control cerebral edema as previously claimed (4, 5). Long-term usage of a respirator has been reported to lead to cerebral edema (6), but in the present study such the usage did not induce cerebral edema.

There were 8.8 ± 7.5 days (mean ± SD) between the onset of conscious disturbance and death in the cases of cerebral edema and 14.0 ± 12.0 days in non-edema cases; the difference was not significant. The time between the beginning of deep coma and death was shorter in the 2 cases of cerebral herniation (1 and 3 days) than in remained 7 cases of cerebral edema but no herniation (5.0 ± 2.4 days). We need to pay attention to cerebral edema in patients with acute hepatic failure.

The interrelationship between hepatic encephalopathy and cerebral edema is close, although the exact mechanisms remain to be disclosed. Williams *et al.* (4, 5) have mentioned that monitoring intracranial pressure was very useful in the diagnosis of cerebral edema in acute liver failure. Treatment of cerebral edema should be performed at the early stage when edema is suspected by measuring intracranial pressure. We plan to study the pathogenesis of cerebral edema both clinically and experimentally in the near future in order to develop proper and effective treatment.

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