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Corrigendum

Corrigendum to: "Detailed clinical course of fatal acute encephalopathy in children" [Brain Dev. 41(8) (2019) 691–698]

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The author regrets that the above article lacks information on the legend in Fig. 1. The Fig. 1 legend is updated below.

The authors and Publisher would like to apologise for any inconvenience caused.

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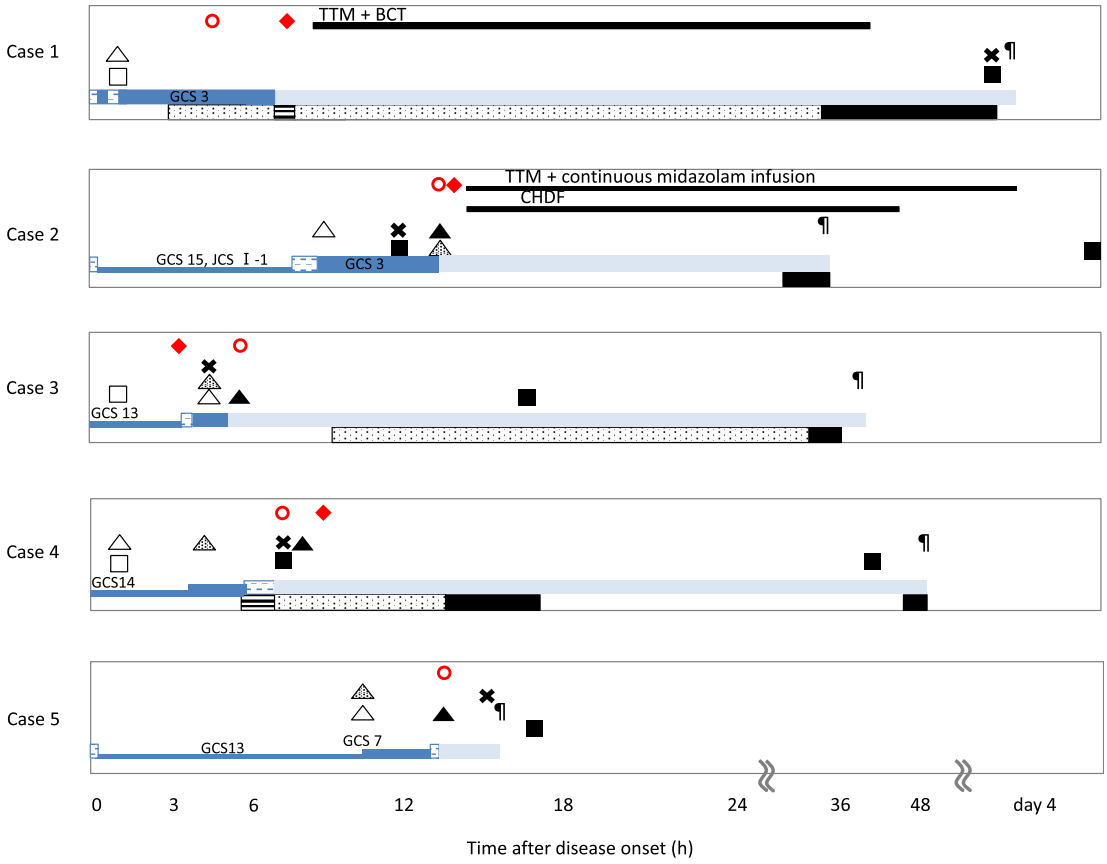

Fig. 1. □ normal head CT, ■ abnormal head CT or MRI, ▲ shock, △ systemic inflammatory response syndrome, ⟁ disseminated intravascular coagulation, × definite diagnosis of acute encephalopathy, ¶ brain death, ◆ initial high dose steroid, ○ intubation. Neurological state: ▬ impaired consciousness, ▭ clinical seizure, ▭ under sedation. EEG findings; ▨ slow wave, ▤ electrical seizure, ▬ low voltage < 10 μV. GCS, Glasgow coma scale; JCS, Japan coma scale. Clinical time courses for all patients. Case 1: She was hospitalized with clinical seizure. Initial CT was normal. She was intubated at 4 h 43 min because of unconsciousness and clinical seizure. High dose steroids (HDS) were administered at 7 h 40 min. Continuous electroencephalogram (cEEG) from 3 h after onset identified slow wave and electrical seizures. Barbiturate coma therapy (BCT; bolus dosage 39.2 mg/kg, maintenance dosage 5 mg/kg/h) with targeted temperature management (TTM) was initiated 9 h after symptom onset because of unconsciousness and refractory electrical seizures. On day 4, diffuse brain edema was identified, and she was diagnosed with Reye-like syndrome and consequently died. Case 2: He was hospitalized with clinical seizure and impaired consciousness, and intubated at 13 h 40 min because this deteriorated to unconsciousness with recurrent clinical seizure. Brain edema was identified on initial CT, and he was diagnosed with Reye-like syndrome at 12 h. Intensive therapies including HDS, TTM, and continuous hemodiafiltration (CHDF) were initiated. However, low voltage (<10 μV) was identified on EEG, and he died at 36 h after onset. Case 3: She was hospitalized with impaired consciousness. Initial CT was normal. HDS was administered at 3 h 35 min because influenza encephalopathy was suspected. She was diagnosed with hemorrhagic shock and encephalopathy syndrome (HSES) because of multiple organ failure (MOF) and disseminated intravascular coagulation (DIC) at 4 h 29 min. Intensive therapies including intubation and inotropic agents were initiated but her condition deteriorated rapidly and she died at 40 h after onset. Case 4: She was hospitalized with impaired consciousness. Initial CT was normal. Consciousness level deteriorated with recurrent seizures. Second CT revealed symmetrically distributed brain lesions of the thalamus, and she was diagnosed with acute necrotizing encephalopathy (ANE) at 7 h 30 min. Intensive therapy including intubation and HDS was conducted but she developed shock and died at 48 h. Case 5: He was hospitalized with clinical seizure and impaired consciousness. Consciousness level deteriorated, and clinical seizure recurred at approximately 12 h. MOF and DIC were identified. He developed shock, and was diagnosed with HSES at 16 h. Despite intensive therapies, he died at 16 h 26 m. CT at autopsy identified brain edema.

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