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Short communication

Burst suppression on EEG: Not always an ominous sign

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1. Introduction

Coma can be a result of a myriad of etiologies with some having distinct EEG signatures. A non-medically induced burst suppression pattern on EEG portends a universally dismal prognosis [1]. It has been described in mass lesions, anoxic brain injury, metabolic disturbances, infections and as a complication of medication overdose. That being said, there are cases described with relatively good outcomes particularly depending on the reversibility of the underlying etiology.

2. Case history

The patient was a retired 67 year old right hand dominant gentleman, with a history significant of poorly controlled diabetes mellitus, medically managed hypertension and hypercholesterolemia and a three year history of dementia with short term memory difficulties without improvement on Rivastigmine. He managed basic activities of daily living and had no history of seizures.

He was last seen well the evening prior and was found the next morning by his wife on the ground, confused and incontinent of urine with scrapes on his scalp. Three hours later, while eating breakfast, he suddenly became drowsy without choking on his food. He was brought to the local hospital with an oxygen saturation of 66% on room air and a Glasgow Coma Scale (GCS) of 8. His GCS rapidly decreased to 3 necessitating intubation and a transfer to the ICU at the University of Alberta Hospital. There was no resuscitation required and the period of hypoxemia was brief.

At the time of admission, he was intubated, ventilated but not sedated, normothermic with a regular but bradycardic heart rate of 53. Initial laboratory investigations were significant for a serum blood glucose of 23.4 mmol/L, beta hydroxybutyrate of 2.1 mmol/L, creatinine kinase of 205 U/L and a sodium of 149 mmol/L with an anion gap of 16 and an elevated serum osmolality of 346 mmol/L. Hyperglycemia was initially treated with an insulin infusion and blood glucose normalized by the time of discharge. Hypernatermia, thought to be secondary to hypovolemia associated with hyperglycaemia, increased to a maximum of 162 mmol/L by 10 h of admission but then gradually resolved by day 2 of admission. Serum lactate was 1.5 mmol/L with an elevation to a maximum of 3.1 by 4 h after admission and a normalization by 12 h. Serum troponin I was not elevated and an initial electrocardiogram

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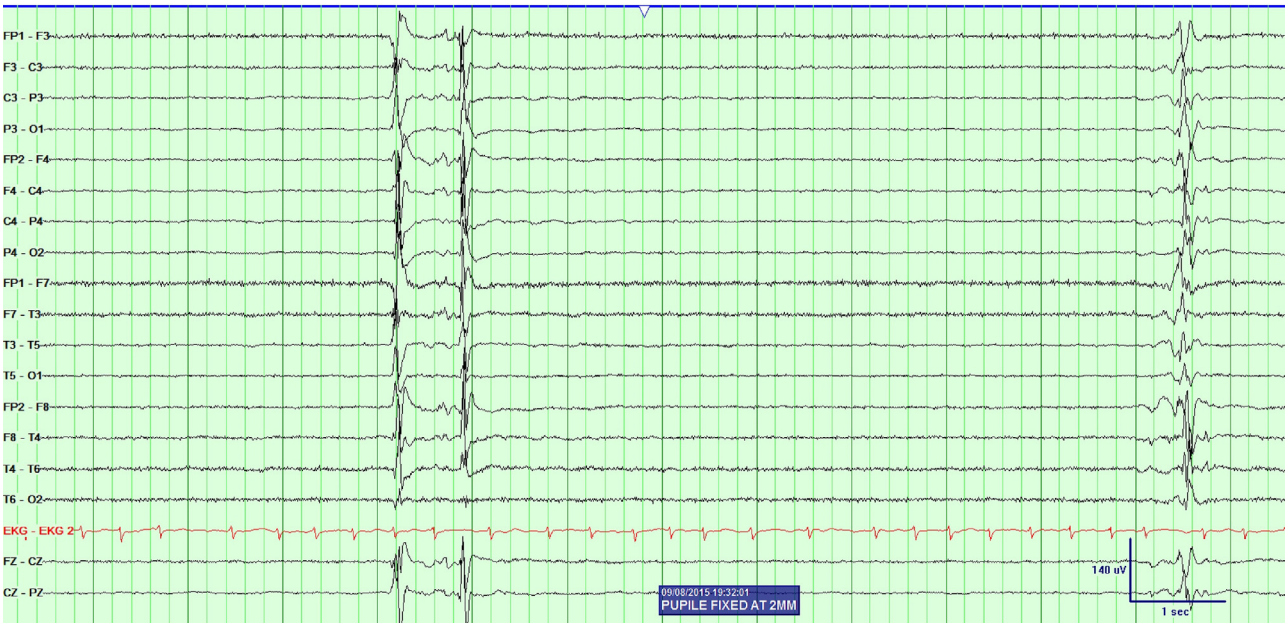

Fig. 1. EEG done approximately 8 h after admission to the Intensive Care Unit at the University of Alberta. Relatively symmetric bursts with generalized epileptiform activity and prolonged periods of suppression. Bursts were not associated with myoclonus. Patient has been off of all sedating medications for 8 h.

showed sinus bradycardia (46 beats per minute) with a prolonged QT interval. There was no myoclonus on presentation and chest x-ray was clear.

Both computed tomography and subsequent magnetic resonance imaging of the head were unremarkable for acute injury other than longstanding frontally predominant atrophy.

Cerebrospinal fluid obtained one day after admission had a normal cell count with elevated glucose (8.5 mmol/L) and protein (0.85 g/L). A thorough infectious work up of serum, urine and CSF was negative. Serum was negative for acetaminophen, ethanol, ethylene glycol as well as methanol and isopropyl alcohol. Urine drug screen was normal. The patient was started on empiric antibiotics and antiviral medications at meningitic doses.

Neurology was consulted 8 h after admission given the decreased level of consciousness out of keeping with diabetic ketoacidosis. He was initially intubated and ventilated and on no sedating medications although he had received 2 mg of intravenous midazolam and a total of 100 mcg of fentanyl about 8 h prior for intubation. He had a GCS of 3, fixed symmetric pupils at 2 mm and intact bilateral corneal reflexes. There was no response to central and peripheral painful stimuli. Tone was decreased; muscle stretch reflexes and plantar responses could not be elicited. An EEG done at the time (Fig. 1) was significant for a burst suppression pattern with no reactivity or myoclonus; suggestive of a diffuse encephalopathy. There were no electrographic or clinical seizures. Repeat EEG 24 h later no longer demonstrated the burst-suppression pattern and continuous delta and theta activity was seen (Fig. 2a). His physical examination showed reactive pupils and movement of all four extremities. A further EEG done at 48 h after admission continued to show improvement with a continuous 5–6 Hz theta frequency rhythm with minimal reactivity to stimulation (Fig. 2b).

The patient suffered from delirium as well as new onset atrial fibrillation while in the ICU that were both medically managed and improved. Ten days after admission, he was transferred to the general medicine ward with a GCS of 14. He was discharged home 11 days after admission with follow up with both Neurology and Geriatrics services. In retrospect, the patient was not aware of any ingestion or overdoses that may have accounted for his presentation.

3. Discussion/Conclusion

This case underscores the fact that a burst suppression pattern does not always portend a poor outcome. This is especially true if the underlying etiology is unknown in which case, a repeat EEG within a reasonable timeframe is warranted to help with prognostication. Of the underlying causes of burst suppression, structural or anoxic cause have the worst outcome while a reversible metabolic or neuropharmacologic cause is relatively more benign [2]. Burst suppression is a dynamic state in continuous evolution between slow waves and an isoelectric point [3] and therefore serial EEGs are needed to indicate the progression. EEG has little specificity with regards to etiology [4] underscoring the importance of repeat EEGs especially if the underlying causes of burst-suppression is unclear.

Although cases of coma in patients with diabetic ketoacidosis have been described, this is the first report to our knowledge where a patient with hyperglycemia presented in coma with burst suppression on EEG with subsequent reversal. Previous work has shown that blood sugar above 22 mmol/L results in slowing of the EEG in the delta range with sporadic spikes [4] but a burst-suppression pattern has not been described.

This case is a good reminder of the conventional wisdom about the importance of reassessing patients both with clinical examination and with an EEG before making judgments about prognosis.

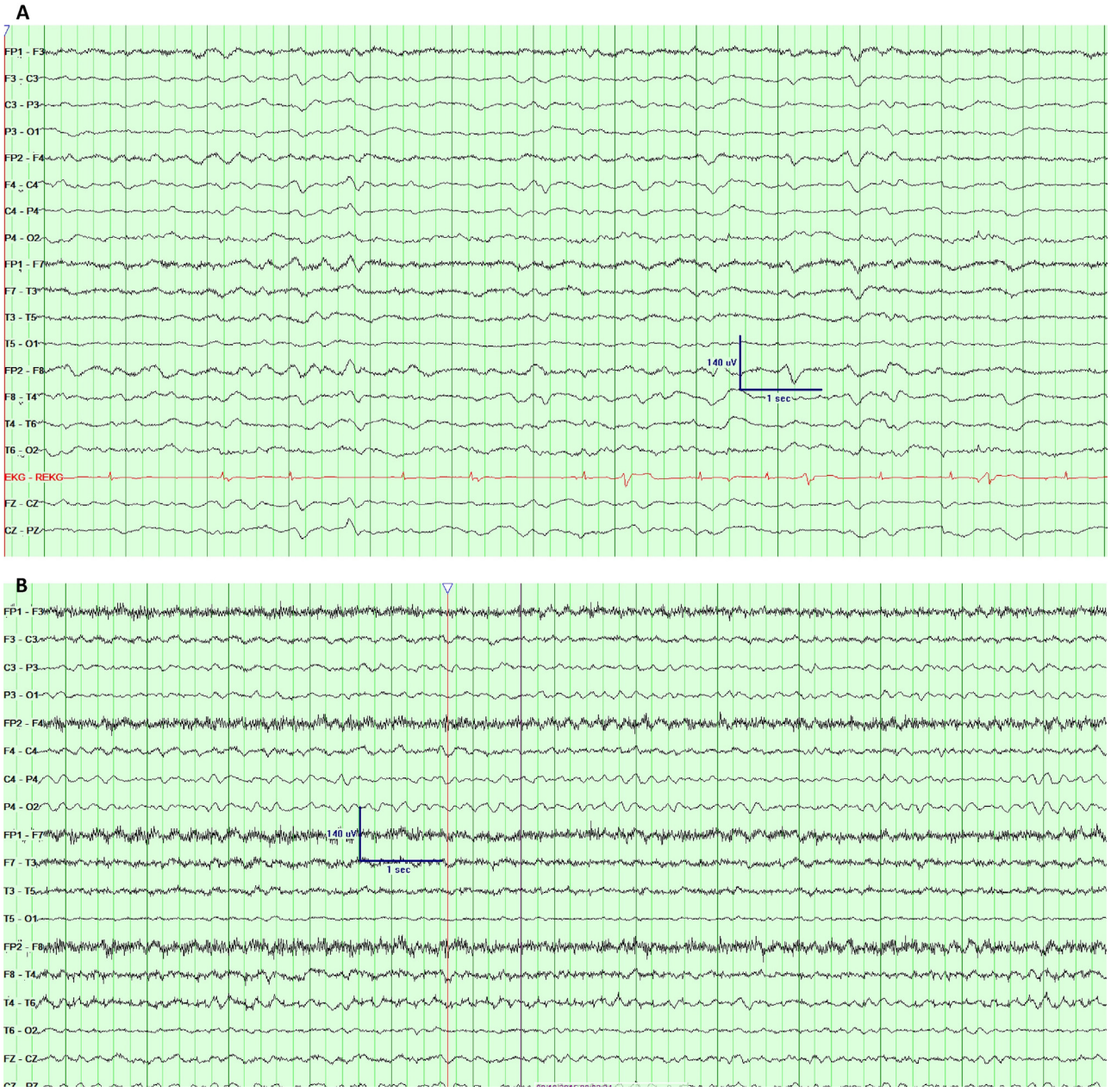

Fig. 2. EEG Day 2 (A) and Day 3 (B). Day 2 (A) Continuous delta and theta frequencies. Note the irregular heart rate. Day 3 (B) theta frequencies with reactivity to stimulation and spontaneous movements.

Declaration of conflict of interest

We hereby attest that none of the authors have any conflicts of interest. This publication has not been published prior to this and that is not under consideration for publication elsewhere.

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