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HAŠIMOTO ENECEFALOPATIJA SA POZITIVNIM OLIGOKLONALNIM TRAKAMA U LIKVORU

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SAŽETAK: Uvod: Hašimoto encefalopatija (HE) je retka bolest koja se često pogrešno dijagnostikuje. Osim visokog nivoa antitela na tiroid-specifičnu peroksidazu (anti-TPO) i antitireoglobulin (anti-TG), neurofiziološki i psihološki testovi su korisni za dijagnozu.Veoma je retko prisustvo oligoklonskih traka u likvoru (CSF) ovih pacijenata. Predstavljamo pacijenta sa HE i oligoklonalnim trakama u likvoru sa dobrim kliničkim odgovorom na terapiju kortikosteroidima. **Prikaz slučaja:** Pacijent, 39 godina, iznenada je razvio fokalni neurološki deficit. Imao je povišena anti-TPO i anti TG antitela, oslabljenu koncentraciju po izveštaju psihologa i oligoklonalne trake u likvoru. Usporavanje električne aktivnosti mozga je normalizovano sa potpunim kliničkim oporavkom pacijenata, posle terapije kortikosteroidima. Pacijent je u kliničkoj remisiji 5 godina posle postavljanja dijagnoze. **Zaključak:** Oligoklonske trake u likvoru mogu biti od pomoći u dijagnozi HE s obzirom da je to još uvek slabo shvaćena bolest. Takođe, brza dijagnoza HE i lečenje kortikosteroidima su važni za potpuni oporavak ovih pacijenata
Ključne reči: Hašimoto encefalopatija, cerebrospinalna tečnost, oligoklonalne trake

UVOD

Hašimoto encefalopatija (HE) je cerebralni poremećaj kod pacijenata sa autoimunim oboljenjem štitne žlezde [1]. Prvi slučaj HE sa visokom serumskom koncentracijom antitela na tiroid-specifičnu peroksidazu (anti-TPO) i antitireoglobulin (anti-TG), tiroiditisom, kognitivnim opadanjem i tremorom je opisao Brain 1966 [2].

Danas je poznato da je prevalencija ovog poremećaja 2/100 000, pri čemu dominiraju žene (odnos polova 4:1). HE obično počinje u 5-toj ili 6-toj deceniji života [3]. Moguće kliničke manifestacije HE su kognitivno oštećenje, psihijatrijske manifestacije (konfuzija, smetnje u pamćenju, promena svesti od pospanosti u komu, gubitak pamćenja, psihotični simptomi), napadi, fokalni neurološki deficiti, poremećaji pokreta, glavobolja [4]. HE se smatra prazninama malih krvnih sudova mozga. Ovo je praćeno perivaskularnim limfocitnim zapaljenjem u moždanom tkivu, prisustvom nepoznatih patogenih autoantitela i imunih kompleksa kod nekih pacijenata sa HE, specifičnim vezivanjem anti-TPO antitela za astrocite i dobrim odgovorom na lečenje steroidima kao obeležjem dijagnoze [5]. Prisustvo oligoklonskih traka u likvoru prijavljeno je u samo nekoliko slučajeva HE [6]. Predstavljamo slučaj pacijenta sa HE i oligoklonalnim trakama u likvoru (CSF) sa dobrim oporavkom nakon terapije kortikosteroidima.

PRIKAZ SLUČAJA

Muški pacijent, 39 godina star, je hitno hospitalizovan zbog prolazne konfuzije i afazije (koja se razvila iznenada) sa kasnijom amnezijom za taj period. Bio je normotenzivan sa normalnim srčanim statusom i normalnim neurološkim statusom u vreme hospitalizacije. Angiografija višeslojnog skenera je bila normalna. Na elektroencefalografiji (EEG) urađenoj istog dana, registrovana je intermitentna delta-teta aktivnost u levom frontotemporalnom delu mozga (Slika 1). Sledećeg dana došlo je do spontanog i skoro potpunog oporavka govornih smetnji, ali je bilo poteškoća oko sećanja imena a konfuzija je i dalje postojala. Magnetna rezonanca glave i ultrazvuk karotidnog duplekss, napravljeni drugog dana, bili su normalni. Pacijent je imao glavobolje, smetnje u koncentraciji i pamćenju i povišena anti-TPO antitela u poslednjih nekoliko godina, iako je sve vreme imao normalan hormonski status štitne žlezde.

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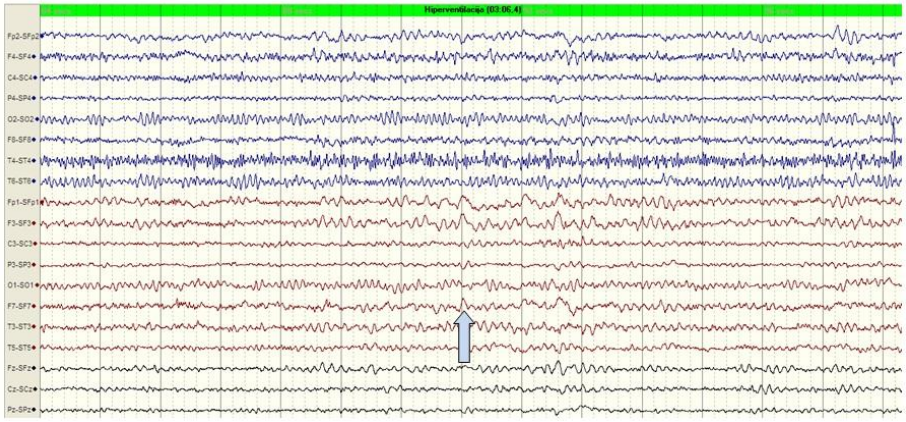
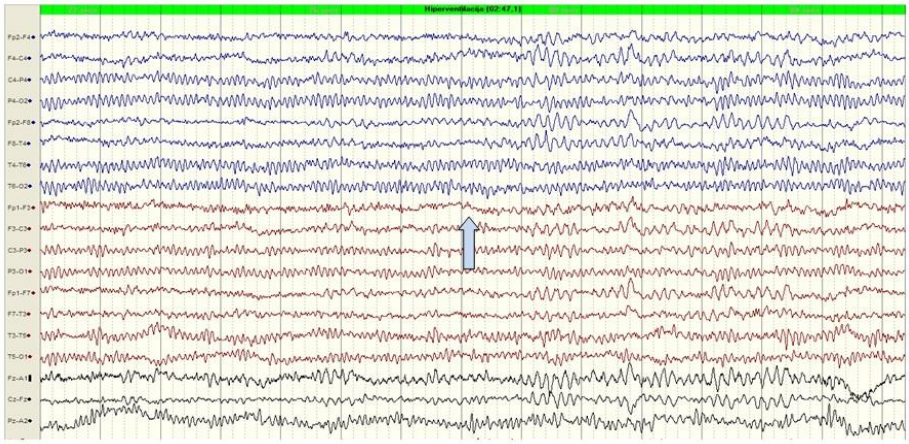
Slika 1. EEG prvog dana hospitalizacije: tokom hiperventilacije teta (oko 6 Hz) i delta (2-3 Hz) aktivnost preko levog frontotemporalnog regiona.



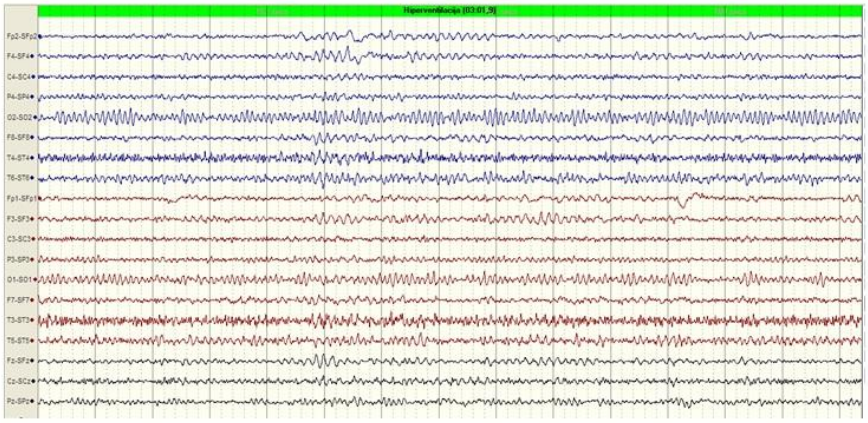
U laboratorijskim nalazima tokom hospitalizacije konstatovane su visoke vrednosti anti-TPO 127,2 IU/ml (0,0-5,7) i anti-TG 1405,47 IU/ml (0,0-4,11), praćene normalnim vrednostima tiroidnih hormona i normalnim ostalim laboratorijskim nalazima. . U sadržaju proteina likvora od 0,58 g/l (0,15 – 0,45), bio je prisutan koeficijent albumina od 7,79 (povišen 5,7) sa pozitivnom oligoklonalnom trakom. Izveštaj kliničkog psihologa nije pokazao ni kognitivnu efikasnost ni kognitivnu disfunkciju, ali je koncentracija bila narušena. Zbog subjektivnog osećaja slabosti u njegovoj desnoj ruci urađena je studija provodljivosti nerva (NCS) i otkrivena je blokada provodljivosti srednjeg nerva u desnoj kubitalnoj regiji. Registrovan je spontani oporavak na kontrolnom EEG-u (intermitentna theta aktivnost nad temporalnim regionima obe hemisfere) (Slika 2).

Slika 2. EEG nakon spontanog rešavanja govornih tegoba, a pre početka terapije kortikosteroidima: intermitentna aktivacija HV 6-7 Hz bilateralno

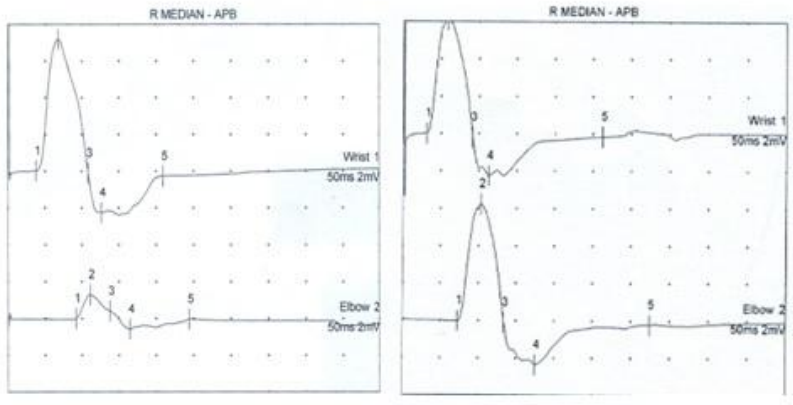


Takođe smo uradili neke dodatne testove kao što su: kulture likvora, autoimune analize, pregled lekova. Svi rezultati su bili normalni. Na osnovu prethodno navedenih nalaza, kod ovog pacijenta je postavljena dijagnoza HE i lečen je trodnevnom pulsnom terapijom kortikosteroidima (500 mg dnevno, iv) praćeno alternativnom oralnom terapijom kortikosteroidima tokom narednih 6 meseci. Glavobolja se značajno smanjila i konačno prestala, a nakon početka terapije nije bilo konfuzije i smetnji u govoru. Klinički oporavak je praćen, uz neurofiziološki oporavak koji je zabeležen na EEG-u (Slika 3) i NCS-u (Slika 4.). Nije bilo novih kliničkih simptoma niti nekih znakova u narednih 7 godina.

Slika 3. EEG urađen nakon završene terapije kortikosteroidima: tokom hiperventilacije nema promene u osnovnim aktivnostima



Slika 4. Blok provodljivosti srednjeg nerva i normalni nalazi nakon nekoliko meseci terapije kortikosteroidima



DISKUSIJA

HE je veoma retko, slabo shvaćeno i često pogrešno dijagnostikovano oboljenje. Za dijagnozu HE neophodna su abnormalna povišenja tiroidnih antitela: anti-TPO kod svih pacijenata i anti-TG kod polovine njih [7]. Kod ovog pacijenta oba antitela su umnožena više od stotinu puta, ali nije imao patoloških nalaza tiroidnih hormona u vreme postavljanja dijagnoze. Zanimljivo je da su početni viši titri antitela na TPO u serumu povezani sa zadovoljavajućim ishodima [8]. Međutim, kako je visok titar plazma anti-TG antitela otkriven kod pacijenata sa B hepatitisom, hepatitisom C, kod pacijenata sa dijabetesom tipa 1 i infekcijom Helicobacter pilori, specifičnost plazma anti-TG antitela u dijagnozi HE je niska [9]. Osim prisustva visokog titra anti-TPO antitela i kliničke manifestacije, ne postoje dobro utvrđeni dijagnostički kriterijumi za HE, pa je HE dijagnoza isključenja. Mora se imati na umu i isključiti široka lista diferencijalnih dijagnoza (npr. toksične metaboličke encefalopatije, meningoencefalitis, psihijatrijske bolesti, moždani udar, zloupotreba droga itd.). Kod našeg pacijenta tumor markeri, skrining lekova, laboratorijska analiza krvi i likvora i radiološka eksploracija mozga isključuju druge poremećaje koji se smatraju važnim u diferencijalnoj dijagnozi.

Prikazani pacijent je imao akutne smetnje govora, praćene konfuzijom i amnezijom, i rezidualnom glavoboljom nakon jednog dana spontane regresije simptoma. EEG nalazi su nespecifični za HE i najčešća EEG abnormalnost je usporavanje od blagog do teškog stepena, što se primećuje u više od 95% slučajeva. EEG nalazi se mogu koristiti za procenu odgovora pacijenata na terapiju steroidima [10]. Došlo je do značajnog

usporavanja EEG aktivnosti na dan nastanka deficita sa smanjenjem sledećeg dana posle spontane regresije simptoma bolesti kod prikazanog pacijenta. Po završetku lečenja i potpunog kliničkog oporavka, EEG se normalizovao.

Pregled likvora može biti potreban da bi se isključio infektivni ili drugi oblik autoimunog encefalitisa. Hiperpoteinorahija je prisutna kod 85% pacijenata sa HE i smanjuje se sa lečenjem bolesti [11]. Abnormalno povišenje tiroidnih antitela u likvoru nalazi se kod 62–75% dijagnostikovanih pacijenata sa HE, koje nema kod zdravih osoba. Tiroidna antitela u likvoru mogu da perzistiraju posle kliničkog poboljšanja [12]. Prikazani pacijent je imao hiperproteinorahiju i pozitivne oligoklonalne trake u likvoru, što je veoma retko prisutno kod pacijenata sa HE. Većina pacijenata sa HE ima normalan nalaz mozga na Magnetnoj rezonanci, što se vidi u slučaju prikazanog pacijenta [13].

Dobar odgovor na terapiju steroidima je obeležje dijagnoze HE. Preporučuje se lečenje prednizonom (1-2 mg/kg dnevno). Tretman bi mogao početi visokom dozom IV metilprednizolona (500–1000 mg/d), a zatim se nastavlja oralnim protokolom. Do 40% pacijenata doživljava potpunu remisiju nakon prvog kursa kortikosteroidne terapije [14]. Neki pacijentima bude bolje bez terapije steroidima. Spontana regresija simptoma kod pacijenta je postignuta nakon jednog dana, dok su preostali simptomi povučeni uključivanjem kortikosteroidne terapije. U slučajevima otpornim na kortikosteroide, treba dodati imunosupresivne lekove, kao što su azatioprin, ciklofosfamid i metotreksat ili imunomodulatornu terapiju kao što je davanje imunoglobulina intravenozno [15]. Pokazalo se da razmena plazme uklanja anti-TPO antitela i značajno ih smanjuje kod pacijenata sa HE, ali bez poboljšanja ni kliničkih ni neurofizioloških parametara [16]. Većina slučajeva ima dobre ishode.

Oligoklonalne trake u likvoru mogu biti od pomoći u dijagnozi HE s obzirom da je to još uvek slabo shvaćeno oboljenje. Takođe, brza dijagnoza HE i lečenje kortikosteroidima su važni za potpuni oporavak ovih pacijenata.

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HASHIMOTO ENCEPHALOPATHY WITH POSITIVE OLIGOCLONAL BANDS IN CEREBROSPINAL FLUID

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Abstract: Introduction: Hashimoto encephalopathy (HE) is a rare and often misdiagnosed entity. Except high levels of the thyroid peroxidase (anti-TPO) and antithyroglobulin (anti-TG) antibodies, neurophysiological and psychological tests are beneficial for the diagnosis. The presence of oligoclonal bands in the cerebrospinal fluid (CSF) of these patients is very rare. We present a patient with HE and oligoclonal bands in CSF with good clinical response on corticosteroid therapy. Case report: Male patient, 39 years old, suddenly developed focal neurologic deficit. He had elevated anti–TPO and anti TG antibodies, impaired concentration on psychologist report and oligoclonal bands in CSF. Slowing of electroencephalography activity was normalized with full clinical recovery of the patients, after corticosteroid therapy. The patient is in clinical remission 5 years after establishing the diagnosis.
Conclusion: Oligoclonal bands in the CSF may be helpful in the diagnosis of HE considering that it is still poorly understood entity. Also fast diagnosis of HE and treatment with corticosteroids are important for a full recovery of this patients.
Key words: Hashimoto encephalopathy, cerebrospinal fluid, oligoclonal bands

INTRODUCTION

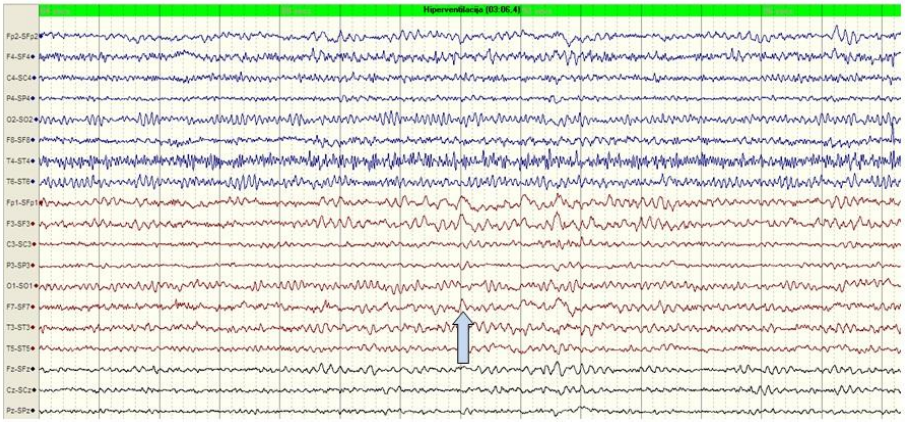
Hashimoto encephalopathy (HE) is a cerebral disorder in patients with autoimmune disease of the thyroid gland [1]. The first case of HE with high serum concentrations of thyroid peroxidase (anti-TPO) and antithyreoglobulin (anti-TG) antibodies, thyroiditis, cognitive decline and tremor was described by Brain in 1966 [2]. It is known today that prevalence of this disorder is 2/100 000, with female predominance (gender ratio 4:1). HE usually begins in 5th or 6th decade of life [3]. Possible clinical manifestation of HE are cognitive impairment, psychiatric manifestation (confusion, interference with memory, alteration of consciousness from somnolence to coma, amnesia, psychotic symptoms), seizures, focal neurological deficits, movement disorders, headache [4]. HE is considered as a vacuities of cerebral small blood vessels. This is supported by perivascular lymphocytic inflammation in the brain tissue, presence of unknown pathogenic autoantibodies and immune complexes in some HE patients, specific astrocyte binding of anti-TPO antibodies, and good response to steroid treatment as a hallmark of the diagnosis [5]. The presence of oligoclonal bands in cerebrospinal fluid is reported in only few HE cases [6]. We present a case of a patient with HE and oligoclonal bands in cerebrospinal fluid (CSF) with a good recovery after corticosteroid therapy.

CASE REPORT

Male, 39 years old, patient was hospitalized as an emergency case due to transitory confusion and aphasia (developed suddenly) with later amnesia for that period. He was normotensive with normal cardiac status and with normal neurological status at the time of hospitalization. Multislice scanner angiography was normal. On electroencephalography (EEG) performed on the same day, intermittent delta-theta activity in left frontotemporal part of the brain was registered (Figure 1). There was spontaneous and nearly complete recovery of speech disturbances next day, but difficulty in the nomination and confusion persisted. Magnetic resonance imaging of his head and carotid duplex ultrasound, made on the second day, were normal. The patient had headaches, concentration and memory disturbances and elevated anti –TPO antibodies in the last few

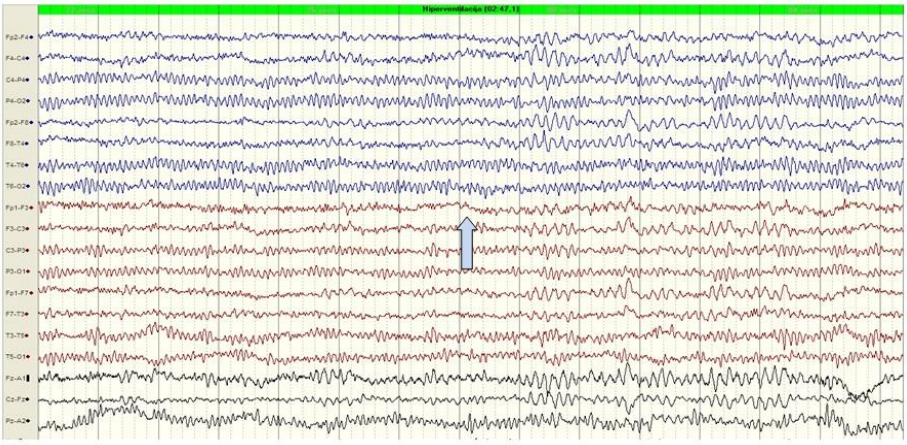
years, although he has had normal thyroid hormonal status all the time.

Figure 1. EEG on the first day of hospitalization: during hyperventilation theta (about 6 Hz) and delta (2-3 Hz) activity over left frontotemporal region.



In laboratory findings during hospitalization high values of anti-TPO 127.2 IU/ml (0.0-5.7) and the anti-TG 1405.47 IU/ml (0.0-4.11) were noted, followed by normal values of the thyroid hormones and normal remaining laboratory findings. In CSF protein content of 0.58 g/l (0.15 – 0.45), albumin coefficient of 7.79 (up 5.7) with positive oligoclonal band was present. Report of a clinical psychologist showed neither cognitive decline efficiency nor cognitive dysfunction, but concentration was impaired. Due to the subjective feeling of weakness in his right arm nerve conduction study (NCS) was performed, and conduction block of the median nerve in his right cubital region was detected. Spontaneous recovery on the control EEG was registered (intermittent theta activity over the temporal regions in both hemispheres) (Figure 2).

Figure 2. EEG after spontaneous resolution of speech problems, and before starting corticosteroids in the treatment: the intermittent activation of the HV 6-7 Hz bilaterally



We also performed some additional tests such as: CSF cultures, autoimmune workup, drugs screen. All of the results were normal. Based on previously mentioned findings, the diagnosis of HE was established in this patient and he was treated with three-day pulsed corticosteroid therapy (500 mg daily, iv) followed by alternative oral corticosteroid therapy during next 6 months. Headache reduced significantly and finally stopped, and there were no confusion and speech disturbances after start of the therapy. Clinical

recovery was monitored, supported by neurophysiological recovery recorded on the EEG (Figure 3) and NCS (Figure 4.). No new clinical symptoms or signs in next 7 years were reported.

Figure 3. EEG done after completed corticosteroid therapy: during hyperventilation no change in the basic activities

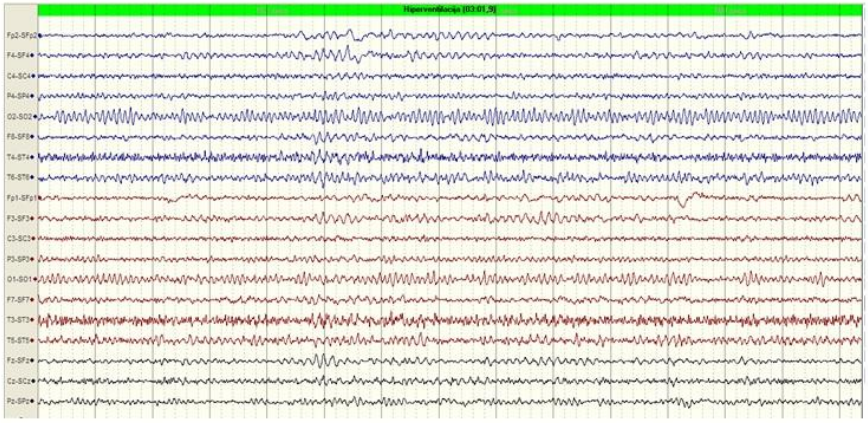
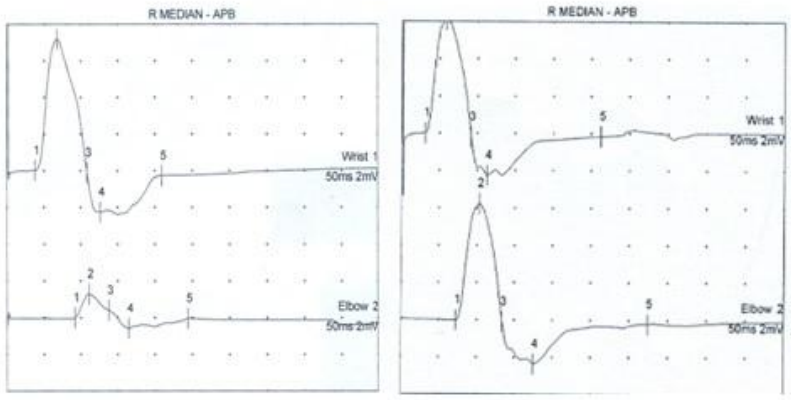


Figure 4. Conduction block of the median nerve and normal findings after several months of corticosteroid therapy



DISCUSSION

HE is a very rare, poorly understood and often misdiagnosed entity. Abnormal elevations of thyroid antibodies are required for HE diagnosis: anti-TPO in all patients and anti-TG in half of them [7]. In the present patient, both antibodies were multiplied more than hundred times, but he had no pathologic findings of thyroid hormones at the time of diagnosis. It is interesting that initial higher serum TPO antibodies titers are associated with more satisfactory outcomes [8]. However, as high titer of plasma anti-TG antibodies has been detected in patients with B hepatitis, C hepatitis, in patients with type 1 diabetes and Helicobacter pylori infection, specificity of plasma anti-TG antibodies in HE diagnosis is low [9]. Except presence of high titer of anti-TPO antibodies and clinical manifestation, there are no well-established diagnostic criteria for HE, so HE is a diagnosis of exclusion. A broad list of differential diagnoses must be kept in mind and ruled out (e.g toxic metabolic encephalopathies, meningoencephalitis, psychiatric disease, stroke, drug abuse etc). In our patient tumor markers, drug screening, laboratory of his blood and CSF and radiological brain exploration exclude other

disorders considered important in differential diagnosis.

Presented patient had acute speech disturbances, followed with confusion and amnesia, and residual headache after one day of spontaneous regression of the symptoms. EEG findings are nonspecific in HE and the most common EEG abnormality is slowing from mild to severe degree, which is observed in more than 95% of cases. EEG findings may be used for evaluation of patient`s response to steroid treatment [10]. There was severe slowing of EEG activity on the day of occurrence of the deficit with it`s reduction on next day after spontaneous regression of symptoms of the disease in presented patient. Upon completion of treatment and full clinical recovery, EEG normalized.

CSF examination may be needed in order to exclude infectious or other form of autoimmune encephalitis. Hyperpoteinorachia is present in 85% HE patients and decrease with the treatment of the disease [11]. Abnormal elevation of CSF thyroid antibodies is found in 62–75% of diagnosed HE patients, which are absent in the healthy individuals. CSF thyroid antibodies may persist after clinical improvement [12]. Presented patient had hyperproteinorachia and positive oligoclonal bands in CSF which is very rarely present in HE patients. The majority of patients with HE have normal MRI brain findings, as seen in the case of presented patient [13].

Good respond to steroid therapy is hallmark of diagnosis HE. Treatment with prednisone (1–2 mg/kg/ daily) is recommended. The treatment could begin with high-dose IV methylprednisolone (500–1000 mg/d) and then continues with oral protocol. Up to 40% of patients experience complete remission after the first course of corticosteroid therapy [14]. Some patients improve without steroid treatment. The patient's spontaneous regression of the symptoms was achieved after one day, while remaining symptoms retreated by the inclusion of a corticosteroid therapy. In cases resistant to corticosteroids, immunosuppressive medications should be added, such as azathioprine, cyclophosphamide and methotrexate or immunomodulatory therapy like given immunoglobulin intravenously [15]. Plasma exchange has been shown to remove anti-TPO antibodies and significantly reduces them in HE patients but without improvement either clinical or neurophysiologic parameters [16]. Majority of the cases have good outcomes.

Oligoclonal bands in the CSF may be helpful in the diagnosis of HE considering that it is still poorly understood entity. Also fast diagnosis of HE and treatment with corticosteroids are important for a full recovery of this patients.

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