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Psychosis in *Borrelia burgdorferi* infection – part II: case reports

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Summary

In the second part of the article case reports of three patients were presented. All patients were treated for *Borrelia* infection and all of them developed psychotic disorders. History of each patient was different, as well as the probability of causal relationship between neuroborreliosis and psychosis.

The first case concerns a female patient with no former psychiatric history, who developed psychotic episode secondary to neuroborreliosis. Psychotic disorders resolved after antibiotic treatment. In the second case, a male patient was previously twice hospitalized in psychiatric ward due to psychosis. During the third psychiatric hospitalization suspicion of Lyme neuroborreliosis (LNB) was propounded. Patient's health state improved after combined therapy with antibiotics and antipsychotic drugs. Direct causal relationship between neuroborreliosis and psychosis is ambiguous. The third case concerns a male patient who was initially treated for LNB and tick-borne encephalitis in an infectious diseases ward. During the hospitalization he developed the first in life episode of mania with psychotic features. Second episode of psychosis occurred after a tick bite and a vaccination against tick-borne encephalitis. In this case, coexistence of many diverse factors results in many difficulties in estimating of the probability of the relationship between LNB and episode of mania with psychotic features.

Referring to the part I of the article, diagnostic difficulties and differential diagnosis were discussed, as well as the causal relationship between borreliosis and psychosis in each case was estimated.

Key words: *Lyme neuroborreliosis*, psychosis, case reports

1. Introduction

In the second part of the article presents case reports of three patients. They were hospitalized in distinct hospitals. Two of them were initially treated for psychotic disorders, but during diagnostics suspicion of Lyme neuroborreliosis (LNB) was propounded. Third patient was initially treated for acute LNB and thick-borne encephalitis

in an infectious diseases ward. During hospitalization the patient developed manic psychosis. Diagnostic difficulties, uncharacteristic course of LNB and doubts in differential diagnosis in each case were discussed.

2. Case I

A 46-year old female was admitted to the psychiatric hospital due to following symptoms: lack of logical verbal contact, insomnia, bizarre behavior (she was looking for her deceased brother, talked to him) and probable auditory hallucinations. Symptoms occurred for the first time in the patient's life and began 2 days before admission to the ward. The patient had never been under psychiatric care before.

In an admission room, she was agitated, vulgar, highly irritable, periodically illogical, behaved inadequately and presented with short-term memory impairment. The patient's family reported that she developed flu-like infection with appetite loss and weakness a week earlier. On the day of admission the patient was consulted by a neurologist. Computed tomography (CT) was performed, showing no abnormalities.

During the stay in a psychiatric ward, the patient presented with fluctuant consciousness disorders, disorientation in time and space. She wandered about the ward, had depressed mood and decreased drive. She confirmed recurrent auditory hallucinations. She did not disclose any delusions. She ate meals and took medications as recommended. She complained about memory and concentration impairment, headaches and backaches.

Due to poor tolerance of drugs, numerous attempts with many different antipsychotic medications were made. She was treated with haloperidol 6 mg/day, olanzapine 10 mg/day and perazine 50 mg/day. Additionally, she received diazepam up to 20 mg/day, clorazepate up to 50 mg/day. Moreover, she received mianserin 30 mg/day, zopiclone 7.5 mg as rescue medicine, piracetam, donepezil, biperiden, and group B vitamins. No improvement was achieved. Patient was still disoriented, presented with echolalia, thought disorders, anxiety, stereotypic movements, bizarre behavior (she hid under other patients' beds, packed her belongings in the middle of the night, climbed window sills, stroked walls).

Once again she was examined by a neurologist, but no neurological deficits were found. In order to exclude organic cause of the symptoms, following were performed: magnetic resonance (MRI) of the head, electroencephalography (EEG), abdominal ultrasonography (USG), chest X-ray, and another CT of the head. MR imaging showed multiple angiogenic or demyelinating changes in the right parietal lobe and bilaterally in the white matter of the brain. EEG was inconclusive due to many muscle aberrations. Only in aberrations-free parts of the recording features of disorganization of background rhythm in alpha-beta waves were visible.

Blood analysis on admission revealed only slight normocytic anemia. In following blood tests there was leukocytosis 15 thou./μL with elevated percent of neutrophils and monocytes. In this situation, ELISA test towards borreliosis was performed. It was positive for IgM antibodies (22 BBU/ml; norm < 11 BBU/ml [Biomedica Borrelia Unit/ml]) and negative for IgG antibodies. After a consultation with an infectious diseases specialist, the patient was transferred to an infectious diseases ward with suspicion of meningitis in Lyme borreliosis. Treatment with ceftriaxone was implemented. Due to lack of information, it is unknown whether lumbar puncture was proceeded and neither are known results of presumptive cerebrospinal fluid (CSF) investigation. After five days, the patient returned to the psychiatric ward due to persisting agitation, bizarre behavior and auditory hallucinations. During 3 weeks of treatment her health state steadily improved. Antipsychotic medications were gradually discontinued. Consciousness and psychotic disorders resolved; mood and drive were stabilized. There was a reduction in somatic complaints and an improvement in general well-being.

MRI of the head was repeated and it revealed unspecified focal bilateral subcortical alterations in the parietal and frontal lobes, which were probably a result of angiogenic demyelination, hence to microangiopathy. Control CSF analysis showed: protein 27 mg/dl, cytosis 7.0 cells/μl (segments – 4%, lymphocytes – 96%), erythrocytes 2.0/μl. Investigation of CSF for *Borrelia burgdorferi* (*Bb)*-specific antibodies was not performed for unknown reasons.

The patient was discharged from the hospital in good general condition, without disturbances of consciousness or acute psychotic disorders. Further treatment at a neurological and infectious disease outpatient clinic was recommended at discharge.

3. Case II

A 27-year old male patient was admitted to a psychiatric hospital after a consultation in a general hospital where he was transferred after a sudden change of behavior. His family reported that patient unexpectedly became highly agitated, was aggressive towards family members and behaved in a bizarre way (he wanted to jump from the moving train, lied down on a floor). After examination by a neurologist in the general hospital and performing CT of the head, which was negative, the patient was transported to the psychiatric hospital.

In the past, the patient was twice hospitalized in a psychiatric ward: 3 years and 1 year before the described episode. In both cases, he was admitted after a sudden change of behavior. He also reported that 2 years after the first psychotic episode, he was diagnosed with borreliosis. He was treated with doxycycline and after then he discontinued antipsychotic medications.

After the admission to the psychiatric hospital, communication with the patient was difficult due to selective answers. He presented with inadequate and bizarre be-

havior (he looked around the room or clenched his eyelids). His speech was quiet and slurred. He seemed to have auditory hallucinations. He did not disclose any delusions. Periodically, he was irritable and agitated. His statements were inconsistent, and many answers were illogical. Nevertheless, he was uncritical to his health state.

In the ward, he was initially highly agitated and aggressive towards other patients and objects. He showed persecutory and grandiose delusions. He required isolation and intravenous drug administration. He received: olanzapine 20 mg/day, haloperidol 10 mg/day and clorazepate up to 60 mg/day.

After few days, the patient's mental state slightly improved. He became calm and cooperated willingly; he took oral medications. However, disorientation and short-term memory impairment persisted.

In blood tests, D-dimer concentration was elevated up to 762 ng/ml, as well as activity of aminotransferases (AST 53 U/l, ALT 45 U/l). Complete blood count, blood clotting test and urine tests were without abnormalities. In EEG, performed during treatment with olanzapine 20 mg/day, recording was correct with slight influence of the drug.

Diagnostics towards LB recurrence was performed. Western-Blot (WB) serum test was performed with the following results (positive result > 7 points; borderline = 6; negative < 6):

- positive for IgM antibodies: 9 points;
- negative for IgG antibodies: 5 points.

In addition, consulting neurologist found bilateral facial palsy.

After a consultation with an infectious diseases specialists, the patient was transferred to an infectious diseases ward for further diagnostics and treatment. There, serology investigation was repeated using ELISA and WB method. It revealed positive result for IgM antibodies (ELISA: 72.79 RU/ml [Relative Units/ml]; WB: 8 points) and borderline result for IgG (ELISA: 19.65 RU/ml; WB: 6 points) in serum. CSF serology was negative both for IgM and IgG antibodies. CSF test showed cytosis 2/μl, protein concentration 0.63 g/l, glucose concentration 3.01 mmol/l.

Oral amoxicillin at the dose of 2.0 g/day was implemented, while treatment with olanzapine 10 mg/day and haloperidol 10 mg/day was continued. Blood count, biochemical and urine tests were normal. After four days, the patient was discharged from the hospital in good general condition. Continuation of antibiotic and antipsychotic therapy was recommended. Afterwards, patient was twice controlled in the infectious diseases ward. During the first follow-up hospitalization MRI of the head was performed. It showed elevated signal rate in T2-weighted images of area to the back from the ventricular triangles. It was probably an image of uncompleted myelinization. There were no other abnormalities. During the second follow-up hospitalization CSF test was repeated and revealed: cytosis 1/μl, protein concentration 0.97 g/l, glucose concentration 3.24 mmol/l. Also, analysis for oligoclonal IgG bands was performed

and it was negative as well. The patient was discharged from the hospital in good general condition and he was recommended to continue therapy with olanzapine 10 mg/day. Medical records confirm that the patient took antipsychotic medications during the control hospitalizations in the infectious disease ward – i.e., for 4 months after discharge from the psychiatric hospital.

4. Case III

A 36-year old man, with no history of psychiatric treatment, with no somatic diseases, was transferred from a hospital emergency ward to an infectious diseases ward because of meningitis. He reported five tick bites in the period of 3 months before the admission. He did not notice erythema migrans.

In the emergency ward, the patient had fever of up to 39°C, complained about nausea, dizziness and diplopia, and was vomiting. He displayed psychomotor retardation. During a physical examination nuchal rigidity, Kernig's sign, ptosis, and spontaneous vertical nystagmus were present. CT of the head revealed 30 x 25 mm hypodensity area in the cortico-subcortical part of the left frontal lobe. MRI of the head revealed no abnormalities, except for asymmetry of the ventricular system (anatomical variant). CSF was clear with cytosis 247 cells/µl (mononuclear cells – 68.9%), protein 140.38 mg/dl (norm: 15–45 mg/dl). Leukocytosis with normal CRP was revealed in blood test.

On admission to an infectious diseases ward, patient complained about nausea, dizziness and vague sight. He was subfebrile and vomiting. In blood tests, leukocytosis 13.1 thou./µl with a predominance of granulocytes (76%) was present. On a neurological examination cranial nerves (III, V, VII) palsy was found, as well as discrete features of cerebello-piramidal syndrome. The patient received intravenous fluids and intense decongestant treatment (dexamethasone up to 32 mg/day and intravenous mannitol). An empirical treatment with acyclovir and intravenous ceftriaxone was implemented.

Serological test confirmed thick-borne encephalitis (positive IgM in serum and CSF) and acute neuroborreliosis without CNS infection (positive IgM in serum: ELISA: 143 RU/ml; WB: 8 points; negative IgG in serum: ELISA 8.81 RU/ml; WB: 0 points; in CSF: both IgM and IgG negative with ELISA method). Syphilis and HIV infection were precluded. No pathogens were grown in blood and urine samples.

The patient's general condition steadily improved – meningeal signs, nausea, vomiting and diplopia decreased. Control CSF analysis showed cytosis 280 cells/µl (lymphocytes – 86%), protein 0.83 g/l (norm: 0.12–0.60 g/l).

On 13th day of hospitalization, symptoms of hypomania began to be observed. After a psychiatric consultation, the patient received quetiapine 3 x 25 mg/day. MRI was repeated, but revealed no abnormalities. Once again the lumbar puncture was performed – CSF was clear with cytosis 23/µl (mononuclear cells – 97%), protein

0.45 g/l. Neurologic examination showed reduced features of palsy of cranial nerves III and VII, decreased menigeal signs and nystagmus when looking right. Analysis for oligoclonal IgG bands was performed and gave negative result. Steroids were continued.

In the following days, manic symptoms intensified – the patient presented with strong psychomotor and sexual agitation, disobeyed the rules, entered the rooms of quarantined patients, which was a threat to his and other patients' health. He was transferred to a psychiatric ward with the recommendation to continue the treatment with ceftriaxone 2 g/day and dexamethasone 6 mg/day (gradual discontinuation).

In the psychiatric ward, the patient behaved inadequately, reported derealization, confirmed former persecutory and grandiose delusions as well as delusion of reference. After a neurological consultation, dexamethasone was discontinued. Aripiprazole 15 mg/day and pregabalin 150 mg/day were implemented. The patient's mental state gradually improved and he was discharged from the hospital in normalized mood and drive, without psychotic symptoms.

Afterwards, the patient was twice controlled in the infectious diseases ward. Neurological symptoms resolved, except for ptosis. Immunology tests (ELISA) towards borreliosis were performed and revealed slightly elevated IgM antibodies (38.35 RU/ml) and negative IgG result (2.23 RU/ml) in serum. Both IgM and IgG results were negative in CSF. CSF analysis showed cytosis 55/µl (including lymphocytes – 94%) and protein 1.21 g/l. Treatment with intravenous acyclovir for 14 days and ceftriaxone 4 g/day for 18 days was implemented. In the following CSF test, cytosis was reduced to 16/µl and protein concentration decreased to 0.98 g/l. The patient was discharged in a good general state.

The Patient was referred to a psychiatric outpatient treatment. He was taking antipsychotic medications for 4 months after discharge. Afterwards, the drugs were discontinued by a psychiatrist.

10 months after the psychiatric hospitalization, he was readmitted to the psychiatric ward due to mania recurrence. In an admission room he presented with logorrhea, grandiose delusions and delusional interpretation of reality.

In the ward, the patient confirmed tick bite 6 weeks before the admission. Moreover, a month before the hospitalization he was vaccinated against tick-borne encephalitis.

During a neurological examination the patient presented with right ptosis, diplopia while looking up, down and right. Perazin 200 mg/day and valproate 900 mg/day were implemented. Once again ELISA test towards borreliosis was performed and revealed positive result for IgM (37.5 AU/ml) in serum and negative for IgG. After a consultation with an infectious diseases specialist, the patient was transferred to the infectious diseases ward with suspected recurrence of neuroborreliosis, for further diagnosis and treatment.

There, serum ELISA test was repeated. Again, IgM antibodies were positive (92.14 RU/ml) and IgG were negative (3.19 RU/ml). Western-Blot confirmation test was performed, and analogical results were obtained (IgM: 9 points; IgG: 0 points). Due to a lack of evident neurological deficits on a careful examination, consulting neurologist decided not to perform a lumbar puncture. The patient was discharged in a stabile general condition. He was recommended to continue the psychiatric treatment with perazine 200 mg/day and valproate 900 mg/day. The patient's further history is not known.

5. Discussion

Case reports of psychosis in LNB have been rather rarely presented in literature so far. Stricker and Winger [1] presented a case report of two patients with complex auditory hallucination and Lyme borreliosis. Symptoms resolved after treatment with antibiotics. No other psychotic disorders were present. In the case of one patient, *Borrelia* spirochetes were confirmed in CSF using PCR method. In the second case, LB was diagnosed on the basis of serological tests. In both cases brain lesions were revealed in MR imaging.

Hess et al. [2] described a patient with delusions, auditory and visual hallucinations, which resolved after antibiotic and antipsychotic therapy. The patient was diagnosed with LB after positive serological tests of CSF. Similarly, Roelcke et al. [3] presented a case of a 54-year old patient with schizophrenia-like symptoms (delusion of reference and delusion of control), which resolved after treatment with ceftriaxone 2 g/day. LB was confirmed with CSF tests.

Markeljević et al. [4] reported atypical symptoms in LNB. A 45-year old patient presented with disorientation, cognitive impairment, behavior disorders, tremor, myoclonic jerks, headaches, paresthesias, sight disorder, and psychosis. LB was confirmed with serological tests. After antibiotic therapy, psychotic symptoms disappeared.

In Poland, Helon et al. [5] presented a case of a 43-year old patient with LB who also revealed psychotic symptoms. However, in this case, psychosis did not resolve after antibiotic therapy. The patient required long-term antipsychotic treatment, also after discharge from hospital.

In Norway, Pasareanu et al. [6] described a case of a woman in her 50s who developed first in live manic psychosis with accompanying muscle pains. Antipsychotic treatment was ineffective. After confirmation of specific anti-*Borrelia* antibodies in serum, intravenous ceftriaxone at the dose of 2 g/day was implemented. Both manic and psychotic symptoms resolved in 14 days. CSF investigation towards specific anti-*Bb* antibodies was negative when antibiotics were implemented. Two months later, during next hospitalization, antibody index was positive, which confirmed the diagnosis of neuroborreliosis.

Diagnostic problems and impossibility of clear separation of endogenous from infectious psychosis were depicted by Bär et al. [7]. They described a case of a 61-year-old woman diagnosed with neuroborreliosis who developed psychotic disorders after *Borrelia* infection. In this case, hypochondriacal and somatic delusions resolved after antipsychotic treatment. Also Fallon et al. [8] underline diagnostic difficulties describing two cases of psychotic disorders in late LNB.

Mattingley and Koola [9] presented a case similar to the cases described above (case II and III). They reported on a 41-year old male patient with a past history of doxycycline treatment due to borreliosis who presented with manic and psychotic symptoms. Specific antibodies were confirmed in serum, but antibiotics were not implemented. The patient improved when antipsychotic drugs and mood stabilizers were introduced. Authors suspect that Lyme-induced neurodegenerative changes might be the cause of psychotic symptoms.

Wójcik et al. [10] emphasize the relevance of *Borrelia burgdorferi* infection diagnostics in the case of psychosis with concomitant flu-like symptoms and neurological disorders. They underline diagnostic difficulties that make it impossible to make a clear diagnosis. In literature, there can also be found a case report on a catatonic syndrome due to neuroborreliosis [11], as well as a case of delirium secondary to Lyme disease [12].

This article presented case reports of patients diagnosed with and treated for LB who also developed psychotic disorders. However, probability rate of actual neuroborreliosis in each case is different. Also, causal relationship between borreliosis and psychosis in each case should be estimated differently. Though, in all the cases diagnostic difficulties and uncertainty about final diagnosis are in a foreground.

The first case is an example of psychosis caused by infection. It is highly probable that it was LB which caused the described disorders. Arguments supporting this thesis are as follows:

1) when symptoms occurred, the patient was 46 years old, which is particularly untypical for the first episode of an endogenous psychosis;
2) long-lasting consciousness disorders and impairment of short-term memory are unusual symptoms of an endogenous psychosis;
3) poor response to antipsychotic drugs was present, despite appropriate doses and duration of the therapy;
4) history of flu-like infection two weeks before onset of the symptoms and uncharacteristic symptoms like headaches, fatigue, joint pains and backaches were present;
5) high rate of anti-*Borrelia* IgM antibodies was found in serum;
6) Psychotic and other symptoms resolved after antibiotic therapy, which is the main argument in favor of neuroborreliosis.

Thus, doubts about diagnosis of neuroborriosis concern the following aspects:

1) there is no information about erythema migrans, which is the most characteristic symptom of borreliosis. However, only in 40–50% of patients with LNB typical skin lesions are observed [13];
2) the result of CSF investigation from before antibiotics implementation is unknown. The analysis was not performed in the psychiatric ward and there is no information from the infectious diseases ward whether CSF was analyzed towards anti-*Borrelia* antibodies;
3) control CSF analysis was without abnormalities in only few days after completion of antibiotic treatment; the presence of specific antibodies in the cerebrospinal fluid was not determined;
4) the level of anti-*Borrelia* IgG antibodies was normal with simultaneous positive IgM result. It is typical for an early stage of infection, but false-positive result of IgM should also be taken into consideration;
5) there were no neurological symptoms characteristic for LNB, e.g., facial palsy. However, nonspecific joint pains occurred and resolved after antibiotic therapy;
6) there is no information whether CSF culture was performed in order to rule out other bacterial infections of the central nervous system.

Among above-mentioned doubts, the most important is lack of information about presence of the anti-*Borrelia* antibodies in CSF, which is crucial for diagnosis confirmation. Nevertheless, the whole clinical picture, especially effectiveness of antibiotic therapy, strongly supports the thesis of neuroborreliosis as a final diagnosis and as a direct cause of described psychotic disorders, similarly to the case reported by Pasereanu et al. [6].

Second case is more ambiguous and it is more difficult to judge whether LNB was the cause of psychiatric disorders. From the patient's history it is known that he was treated for neuroborreliosis in the past. However, medical documentation about it was inaccessible. Therefore, there is no precise information about the patient's signs, symptoms and implemented therapy for borreliosis.

In this case, psychotic disorders appeared abruptly and were intense – visual and auditory hallucinations were present as well as delusions. Similarly to the first case, consciousness disorders and short-term memory impairment occurred. Although antipsychotic drugs had incomplete effect, patient took them even after antibiotics were discontinued.

Diagnosis of neuroborreliosis should be made here more carefully than in the first case. In laboratory tests, anti-*Borrelia* IgM antibodies were found (both with ELISA and Western-Blot methods), whereas IgG result was negative in the first test and borderline in the second one. Such a result raises doubts about former borreliosis. No antibodies were found in CSF. There was only a slight anomaly in CSF cell count.

On the other hand, the significant argument for diagnosis of neuroborreliosis in this case is relatively high effectiveness of antibiotic treatment. Nevertheless, there is no data about erythema migrans history or joint pains. Although neurological examination revealed bilateral facial palsy, it was not confirmed later.

In this case, following hypotheses should be considered:

1. Psychotic disorders were caused by infection, probably late neuroborreliosis, similarly to cases cited above [8, 9]. Supporting arguments are: rapid outburst of symptoms, consciousness disorders and short-term memory impairment, poor effect of antipsychotic treatment, positive serological tests, facial palsy, improvement after antibiotic treatment.
2. Patient could be a person predisposed to psychotic disorders and then endogenous psychosis episode was triggered by neuroinfection. Supporting arguments are: typical age of the patient for initial psychotic episodes, history of former psychiatric hospitalizations due to psychosis, partial improvement after antipsychotic medications.
3. It was a spontaneous episode of endogenous psychosis, while neuroinfection was an unrelated coincidence. Improvement then would be a result of la ong-term antipsychotic treatment.
4. Differential diagnosis should also include immune cross-reaction in the CNS. However, negative result of analysis for oligoclonal IgG bands rules out autoimmune encephalitis.

The most probable hypothesis is the first one or the second one. On the basis of available information, it cannot be settled which one is more reliable, since further patient's history is not known. Especially, there is no information, whether he continued antipsychotic therapy or whether he had further psychotic episodes.

In both cases long lasting consciousness disorders and short-term memory impairment are remarkable, as well as slight effectiveness of antipsychotic medications. Similar symptoms were described by Helon et al. [5]. Also, Fallon et al. [14] and Zajkowska et al. [15] underlined memory impairment in borreliosis.

The third case is the most complicated one. Unequivocal recognition of the cause of psychotic disorders is particularly difficult due to coexistence of many various factors. Differential diagnosis should include that:

1. The first manic episode was caused by steroids, as mania and psychosis resolved after dexamethasone was discontinued and antipsychotic medications were implemented. Although, iatrogenic ground does not explain the cause of the second manic episode. The patient had not taken any medications before second episode.
2. Symptoms of psychiatric disorders were caused by CNS infection – LNB. With such a suggestion the patient was transferred from the infectious diseases ward to the psychiatric hospital after the first psychotic episode. The fact that patient had

been taking intravenous ceftriaxone and for 18 days before psychotic symptoms occurred is an argument against this hypothesis. Moreover, there were no anti-*Borrelia* antibodies found in CSF. Nevertheless, this hypothesis was taken into consideration in the case of the second psychotic episode. Then, patient confirmed tick bite about 6 weeks before onset of the symptoms. In addition, positive result for IgM antibodies in serum made this hypothesis highly probable. Though, it was rejected after the consultation in the infectious diseases ward. No symptoms of neuroborreliosis were found (no neurological deficits or flu-like symptoms), hence lumbar puncture and antibiotic treatment were abandoned.

3. The patient's predisposition to psychotic or affective disorders should be taken into consideration as well. Then, tick-borne encephalitis or steroid therapy could be the triggers for psychiatric disorders. It is although impossible to proclaim which one of these factors was decisive in this case. Unrelated coexistence of manic disorders and CNS infection is also possible. Especially, the second episode of mania supports this theory. However, against are the following: a) untypical age for the first manic episode in the patient's life; b) no mood disorder history; c) no history of psychiatric disorders in the patient's family.
4. In differential diagnosis autoimmune encephalitis was also considered, but the CSF analysis towards oligoclonal IgG bands was negative. Nevertheless, there is a hypothetical probability that the second manic episode was a result of vaccination against tick-borne encephalitis, which patient had received a month before symptoms appeared. Then, theoretical cross-reaction between vaccinal antigens of tick-borne encephalitis virus and surface proteins of neurons could be suspected. However, there is no empirical evidence supporting such a theory.

Although the probability that the psychosis in the last case was caused by LNB is low, history of this patient shows how difficult and ambiguous diagnosis of infectious psychosis may be. The case of this patient clearly depicts that differential diagnosis in such a situation is highly complicated process and diagnosticians have to take into consideration many diverse factors. Moreover, precise indication of the cause may be even impossible.

6. Conclusions

Psychotic disorders may be one of many symptoms of infectious diseases. Borreliosis is tick-borne disease in which schizophrenia-like symptoms may occur [14]. However, due to diagnostic difficulties, confirmed diagnosis of neuroborreliosis as a cause of psychosis may be unattainable, which was illustrated with an example of the described cases and cited publications [7, 8, 10]. Case reports depict uncharacteristic nature of LB symptoms, as well as an impact of many accompanying factors on clinical picture.

Despite the fact that neuroborreliosis as an unambiguous cause of psychotic disorders is proclaimed only casuistically, it is a disease which should be considered in differential diagnosis. Especially, it ought to be kept in mind in the case of untypical psychotic disorders, occurring at abnormal age for endogenous psychosis, and in the case of patients who inhabit areas endemic for borreliosis.

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