

CASE REPORT

Prolonged Psychogenic Non-epileptic Seizures Manifested Immediately after Minor Sport-related Head Injury in a Young Judo Athlete : A Case Report

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Abstract : Psychogenic non-epileptic seizures (PNES) are often associated with a history of mild traumatic brain injury, which may require differentiation with posttraumatic epilepsy, but usually there is a sufficiently long interval between an episode of trauma and their manifestations. Herein we report a case of a young female judo wrestler who experienced PNES immediately after minor head injury sustained during sport contest. It can be speculated that pathophysiological background of PNES in athletes may differ from general population and that disease in such cases may be related to stress of competition and fear to lose it. Early application of the video-electroencephalographic monitoring allows for timely diagnosis and initiation of appropriate treatment of PNES. *J. Med. Invest.* 73:265-269, February, 2026

Keywords : *Differential diagnosis, Immediate posttraumatic seizures, Psychogenic non-epileptic seizures, Sport-related head injury, Video-EEG monitoring*

INTRODUCTION

Psychogenic non-epileptic seizures (PNES) are generally considered as nonspecific umbrella category used for a variety of atypical neurophysiological responses to emotional distress, physiological stressors, and danger (1). They are often associated with a history of mild traumatic brain injury (TBI) and may be easily misdiagnosed as posttraumatic epilepsy (2-7). Nevertheless, usually there is a sufficiently long interval between an episode of trauma and manifestation of PNES. Herein we report a case of a young judo athlete who experienced prolonged convulsions soon after minor head injury sustained during sport contest, which were initially diagnosed as status epilepticus, while subsequent video-electroencephalography (EEG) monitoring effectively allowed for correct diagnosis of PNES.

CASE REPORT

A 20-year-old woman moved from a provincial town to the capital of Japan after graduation from high school and got a job as a member of the business team in a strong industrial company, where judo training was actively promoted and which she joined as well. She had no history of psychiatric disorders, psychological abnormalities, or mental trauma during childhood, and had regular educational background. A bit later, during judo contest within the regional qualifying tournament for the National Judo Championship, she hit her forehead against the opponent's head, but did not experience significant problems, continued to compete, and won this contest. In approximately 30 minutes, she took part in the next round competition and lost it in extra

time. Subsequently she noted a feeling of extreme fatigue and headache during that contest, and upon its completion for the first time in her life she experienced prolonged convulsions in both upper limbs, while remained fully conscious and responsive.

She was referred by ambulance to the emergency hospital, where clinical examination, including computed tomography and magnetic resonance imaging (MRI) did not reveal any TBI (Fig. 1). Nevertheless, convulsions of her upper limbs accompanied by head jerks continued, and were considered as status epilepticus. She was given levetiracetam (1500 mg/day) and lacosamide (200 mg/day), but they did not change her condition. Therefore, on the 4th day after an accident, she was transferred to the tertiary Epilepsy Center of our University Hospital for additional examination and control of seizures presumed to be caused by sustained head injury.

At admission, her neurological examination and repeat brain MRI did not reveal any abnormalities, but convulsions without impairment of consciousness in her both upper limbs, as well as head jerks persisted while sporadically their pattern was changing. The blood test did not disclose any abnormalities, beside mild decrease of total cholesterol and phospholipids (Table 1). Examination of cerebrospinal fluid was not done. Antigen tests did not reveal acute viral infections. Video-EEG monitoring was initiated, but no epileptiform activity was defined (Fig. 2). The diagnosis of PNES was strongly suggested and confirmed by subsequent video-EEG monitoring, which continued for several days until discharge of the patient from the hospital on the 10th day after an accident. By that time, all antiseizure medications (ASM) were stopped, and the patient was informed that she does not have epilepsy. Specialized psychiatric examination did not reveal any abnormalities requiring treatment.

Subsequently, her convulsions steadily disappeared, but relapsed when she tried to resume her judo training. Thereafter, she decided to quit sport, to retire from her work, and to return to her parent's house at hometown.

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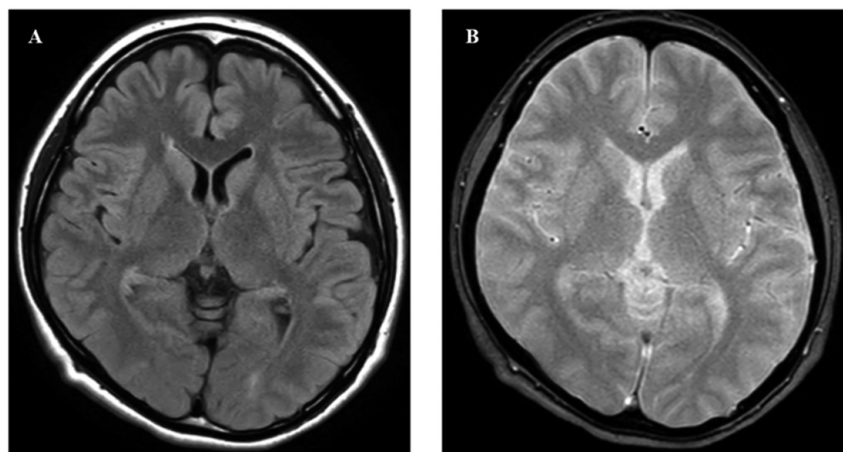


Figure 1. T1-weighted (A) and T2-weighted (B) MRI of the presented patient in few hours after minor sport-related head injury and manifestation of psychogenic non-epileptic seizures did not demonstrate any abnormal findings.

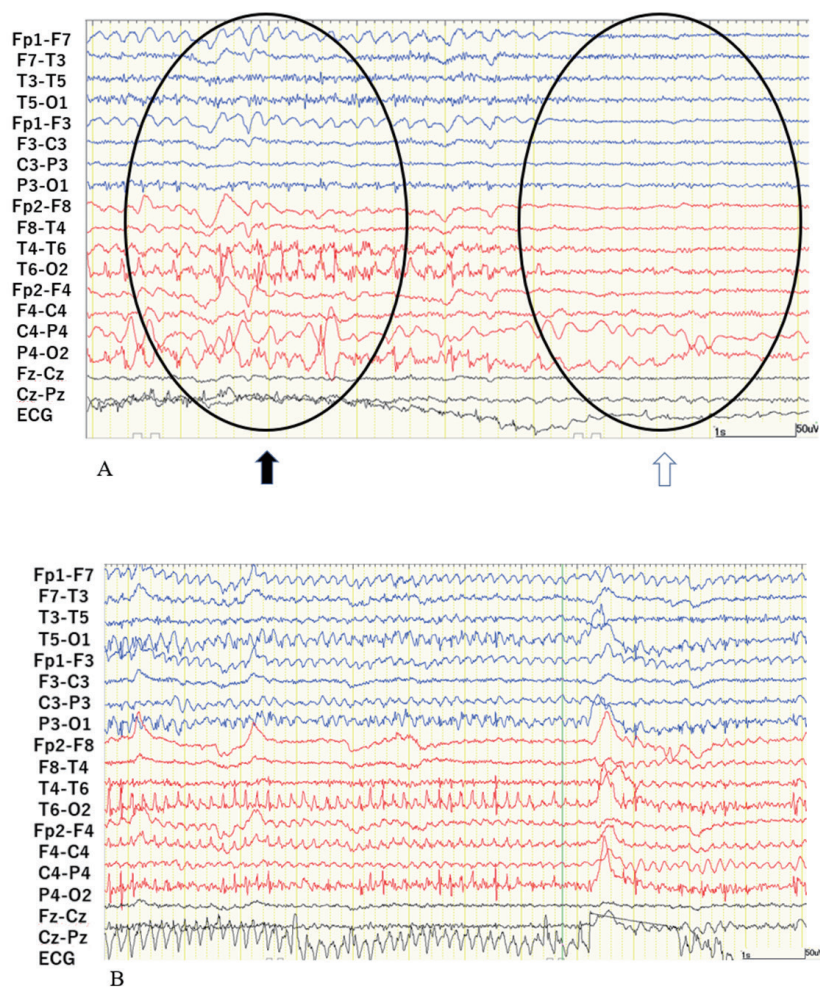


Figure 2. Findings during video-EEG monitoring of the presented patient (bipolar montage, time constant 0.1 s, high-pass filter 50 Hz). A : Background activity showing dominant, symmetrical, and reactive alpha rhythm bilaterally, with intermittent, rhythmic, uniform wobbles of approximately 1 Hz frequency, most probably caused by muscle artifacts due to incorporation of the electromyogram into the waveform, and with evident electrophysiological changes at shift from persistent convulsions in the patient's upper limbs with (solid arrow) without (open arrow) accompanying head jerks. B : Rhythmic spike-like slow-wave complex artifacts are appearing in both occipital regions (T5-O1 ; P3-O1 ; T6-O2 ; P4-O2), while other montages in close vicinity do not show obvious electrophysiological abnormalities. There is no evident epileptiform activity.

Table 1. Results of blood test in a 20-year-old woman with prolonged psychogenic non-epileptic seizures after minor sport-related head injury.

Parameter	Value	Normal values' range
Red blood cells ($\times 10^4/\mu\text{L}$)	419	386 - 492
Hemoglobin (g/dL)	13.0	11.6 - 14.8
Hematocrit (%)	38.3	35.1 - 44.4
MCV : Mean corpuscular volume (fL)	91.4	83.6 - 98.2
MCH : Mean corpuscular hemoglobin (pg)	31.0	27.5 - 33.2
MCHC : Mean corpuscular hemoglobin concentration (g/dL)	33.9	31.7 - 35.3
RDW-CV : Red cell distribution width - coefficient of variation (%)	12.8	12.2 - 14.8
NRBC : Nucleated red blood cells (%)	0.0	0 - 0.5
White blood cells ($\times 10^2/\mu\text{L}$)	62	33 - 86
Neutrophils (%)	66.2	41 - 68
Eosinophils (%)	1.9	0 - 7
Basophils (%)	0.5	0 - 3
Monocytes (%)	6.3	2 - 8
Lymphocytes (%)	25.1	25 - 45
Platelets ($\times 10^4/\mu\text{L}$)	22.3	15.8 - 34.8
PDW : Platelet distribution width (fL)	10.4	9.6 - 15.2
MPV : Mean platelet volume (fL)	9.8	9.2 - 12.0
Glucose (mg/dL)	88	73 - 109
Total cholesterol (mg/dL)	132	142 - 248
Triglycerides (mg/dL)	48	30 - 117
Phospholipids (mg/dL)	152	160 - 260
Total bilirubin (mg/dL)	0.7	0.4 - 1.5
Uric acid (mg/dL)	3.5	2.6 - 5.5
Lactate dehydrogenase (U/L)	158	124 - 222
Alkaline phosphatase (U/L)	61	38 - 113
Cholinesterase (U/L)	301	201 - 421
Amylase (U/L)	66	44 - 132
Creatine phosphokinase (U/L)	104	41 - 153
Magnesium (mg/dL)	1.9	1.8 - 2.4
Zinc ($\mu\text{g}/\text{dL}$)	84	80 - 130
Chloride (mmol/L)	105	101 - 108
Calcium (mg/dL)	8.9	8.8 - 10.1
Inorganic phosphate (mg/dL)	4.3	2.7 - 4.6
PT : Prothrombin time (sec)	11.9	9.8 - 12.1
PT/INR	1.01	0.88 - 1.10
APTT : Activated partial thromboplastin time (sec)	26.7	24 - 34
ESR (Erythrocyte sedimentation rate) at 60 minutes (mm)	6	3 - 15

DISCUSSION

Theoretically, any TBI can be associated with PNES. However, we were not able to identify specific reports on their interrelationships with sport-related head injury. Several characteristics of the presented case of a young female judo wrestler, who experienced first manifestation of PNES after minor head injury sustained during sport contest seem unique. First, PNES usually manifest on the medium- or long-term follow-up (in 81-89% of patients within the first year) after TBI (5, 6), whereas in our

patient they were observed immediately (i.e., within 24 hours) after traumatic accident. Second, the duration of convulsions in cases of PNES may be variable, but prolonged presentation during several days after injury mimicking status epilepticus is rather unusual. Such condition has been occasionally designated as PNES status (8). Third, PNES are often associated with underlying psychiatric disorders, psychological abnormalities, or mental trauma during childhood (1, 3, 8-10), but neither was a case in our patient.

On the other hand, our patient had multiple causes for a

psychological distress. She left her parents' home in a relatively young age and moved to megapolis, which was invariably accompanied by significant cultural and social changes in her life. She got good job in a prestigious company, and was seemingly encouraged to practice judo, which she might consider helpful for her professional growth, thus fear not to achieve desired results during competition and to lose it might be significant. After losing a contest in extra time (which indicates its highly competitive nature) during prestigious tournament she should experience psychological shock, which coupling with physical and mental exhaustion, as well as minor head injury sustained in previous contest, could trigger PNES. Hyperventilation during competition might have some provoking role as well, since its association with PNES is well recognized (1).

It can be presumed that under similar conditions any athlete, especially young one, can develop PNES, even in the absence of obvious predisposing factors. Appearance of PNES in children and adolescents after foot injury during swimming competition and following physical exercises was noted before (1). In such cases, pathophysiological background of the disease may differ from that in general population, which may have an impact on diagnostic strategy, treatment, and prognosis. If PNES manifest immediately after sport-related head injury, their differentiation with other non-epileptogenic conditions, such as concussive convulsions appearing at the time of head impact, convulsive syncope, and manifestation of sports' anxiety (11-13) may be needed. Obviously, seizures may be also manifestation of parenchymal brain injury; therefore, in case of their appearance hospital transfer and cranial imaging are strongly recommended (11).

Several clinical features may help to differentiate epileptic seizures and PNES, while precise diagnosis certainly requires confirmatory examinations (8). Some studies indicate that blood tests may be helpful on this way. Leucocytes and neutrophil counts, systemic inflammatory response and immune-inflammatory indices, levels of creatine phosphokinase, cortisol, lactate, norepinephrine, interleukin-6, S100-beta protein, brain-derived neurotrophic factor (BDNF), ceruloplasmin, pro-opiomelanocortin (POMC), neuropeptide Y (NPY), alpha-1-acid glycoprotein, malate dehydrogenase 2, ubiquitin carboxy-terminal hydrolase (UCH-L1), neurogranin, as well as RNA expression profiles, were all suggested as potential biomarkers useful for differential diagnosis of PNES, but obviously, their clinical utility should be tested further (8, 14-21). Currently, video-EEG monitoring is considered the "gold standard" option for early diagnosis of PNES and their differentiation with posttraumatic epilepsy (3, 22), as was re-confirmed in case presented herein. Its application allowed our patient to avoid unnecessary long-term therapy with ASM. Although prolonged video-EEG monitoring has become a routine tool for clinical evaluation of chronic epilepsy, this technique is rarely applied in acute period after TBI and usually not readily available in emergency room facilities. Therefore, if despite administration of ASM in a patient with TBI seizures persist, he or she should be transferred to a specialized epilepsy center, where prolonged video-EEG monitoring can be attained. It should be noted, however, that epileptic seizures and PNES may co-exist in the same patient, and interictal epileptiform activity was found on EEG in 10% of patients with PNES status (3, 8). It may complicate interpretation of electrophysiological findings and clinical diagnosis in general. It also indicates that such patients should be preferably evaluated in a specialized epilepsy center.

There are no guidelines on the optimal timing for return to competition after sport-related PNES. In our patient such an attempt resulted in recurrence of the disease. It was noted before, that relatively small proportion of individuals with PNES becoming seizure-free despite psychological therapy and

other treatment (1, 6). Factors of good prognosis include short diagnostic delay, early initiation of care after symptom onset, patient's comprehension of the disease psychogenic etiology, absence of comorbid epilepsy, absence of the history of TBI and psychiatric comorbidity, supportive environment, occupational activity, and care attendance (3).

CONCLUSIONS

Manifestation of PNES immediately after minor sport-related head injury is very unusual, but they still should be considered in differential diagnosis of presenting convulsions. Accumulation of data on sport-related PNES seems important task for future research.

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CONFLICT OF INTEREST DISCLOSURE

The authors have no personal or institutional interests in drugs, materials, or devices described in this paper.

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