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Short Communication

Psychiatric Aspects of Acute Pandysautonomia

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Summary. Five cases of acute pandysautonomia and one case of acute autonomic and sensory neuropathy are described with special reference to psychiatric symptoms. They originally presented as psychiatric disorders, such as hysterical neurosis, epilepsy, anorexia nervosa and hypochondriacal neurosis. Psychiatric symptoms arise from their autonomic nervous dysfunction and show emotional instabilities which are often regarded as hysterical overacting.

Key words: Psychiatric effects – Acute pandysautonomia

Introduction

Since acute pandysautonomia (APD) was initially described as an entity by Young et al. in 1969, a number of similar cases have been described (Okada et al. 1988). The disorder is characterized by severe sympathetic and parasympathetic impairment with relative or complete preservation of somatic motor and sensory functions. The causes of the syndrome are unknown; however, an immunological disorder similar to the Guillain-Barré syndrome has been suggested. The clinical course is often protracted with slow improvement. The site of the lesion is considered to be in the postganglionic fibres of both the sympathetic and parasympathetic nervous systems (Young et al. 1969; Okada et al. 1975). Preganglionic fibres or central sites of the autonomic nervous system (ANS) have also been thought to be disturbed in some cases (Okada et al. 1975; Okada and Shintomi 1990). On the other hand, the literature on the psychiatric aspects of acute pandysautonomia is very limited, although this illness is often misdiagnosed as one of various psychiatric disorders. The purpose of this study is to show how often psychiatric symptoms are observed in patients with acute pandysautonomia and to discuss the significance of the psychiatric aspects.

Case Reports

The clinical details of the six cases in this study are shown in Table 1. These cases have been reported previously (Okada et al. 1975, 1988, 1989; Okada 1989; Okada and Shintomi 1990), but here concise descriptions including psychiatric symptoms are presented.

Case 1 (Okada et al. 1975). A 37-year-old man suddenly began to suffer from pain in the upper abdomen and lower back. After 10 days, he showed additional symptoms, including disturbances of accommodation, mydriasis of pupils, orthostatic hypotension, fainting in an upright position, intermittent pains in the trunk, dryness of mouth, anhidrosis, chronic diarrhoea, and impotence. He looked younger than his age. He was referred to our clinic as suffering from hysteria 2 months after the onset of illness, because of overacting expression of lethargy or dullness and persisting complaints of pain. The neurological diagnosis was acute pandysautonomia.

Table 1. Clinical details of six patients with acute pandysautonomia who presented with psychiatric disorders

Patient no.	Age (years) sex	Main clinical features	Previous diagnosis	Neurological diagnosis[a]
1	37/M	Emaciation, diarrhoea, faintness, insomnia	Hysterical neurosis	APD
2	43/M	Faintness, fatigue, lack of appetite	Epilepsy[b]	APD
3	28/F	Low back pain, faintness, anhidrosis	Hysterical neurosis	APD
4	9/F	Faintness, vomiting, emaciation, diarrhoea	Anorexia nervosa[b]	APD
5	58/F	Faintness, vomiting, diarrhoea, emaciation	Hypochondriacal neurosis	APD
6	20/F	Lassitude, vomiting, faintness, diarrhoea	Hysterical neurosis	AASN

[a] APD, Acute pandysautonomia; AASN, acute autonomic and sensory neuropathy

[b] Diagnosis by psychiatrist; others, by physician

Case 2 (Okada et al. 1975). A 43-year-old man developed blurred vision, with difficulty in reading, dryness of the mouth, pain in the parotid region, vomiting and lack of appetite following complaint of fatigue and lumbago for 1 week. He also noticed lack of sweating below the neck, and impotence. Shortly thereafter, he began to have episodes of loss of consciousness when in the upright position. He was referred to our clinic as suffering from epilepsy about 3 months after the onset of illness. The neurological diagnosis was acute pandysautonomia.

Case 3 (Okada et al. 1988). A 28-year-old woman began to suffer from severe low back pain. After several days, she could not accommodate glaring light or near vision, and suffered from dry eyes and mouth. She also suffered from constipation and anhidrosis. After 3 weeks she was referred to our clinic as a hysterical patient, since she had had a fainting spell in hysterical fashion within 2 weeks of the onset of illness. The neurological diagnosis was acute pandysautonomia.

Case 4 (Okada et al. 1988). A 9-year-old girl had severe epigastric pain and vomiting following a common cold for about 3 days. Several days later, she had fainting spells and a high fever, diagnosed as aspiration pneumonia. Afterwards, she showed disturbances of accommodation and mydriasis. She also had intermittent pain in the abdomen and vulva, pylorospasm, vomiting, diarrhoea, and urinary incontinence. She was misdiagnosed as having anorexia nervosa because of a weight loss from 32 kg to 19 kg. She appeared lethargic, dull or apathetic and began psychotherapy. She was referred to our clinic 1 year after the onset of illness. The neurological diagnosis was acute pandysautonomia.

Case 5 (Okada et al. 1989) A 58-year-old woman had constant diarrhoea, abdominal pain and vomiting which began 2 years after suffering from struma at the age of 26. She gradually showed autonomic nervous dysfunctions such as orthostatic hypotension, chronic diarrhoea, increased peristaltic movements of the intestine, atonic stomach, diminished tears, salivation and sweating, and pupillary disturbances. These symptoms continued chronically until she was referred to our clinic with a diagnosis of hypochondriacal neurosis at the age of 53. The neurological diagnosis was a type of acute pandysautonomia.

Case 6. (Okada 1989). A 20-year-old woman exhibited low abdominal pain, diarrhoea, fever, emaciation and general muscle pains and fainting spells. Within 1 month she developed various signs of autonomic nervous dysfunction such as orthostatic hypotension, diminished tears and salivation, dysuria, diarrhoea, vomiting, emaciation, and pupillary disturbances. She also exhibited areflexia of the extremities and hypalgesia in the lower body from the level of the mammary line. She seemed puerile and overacted when she complained of her symptoms. She received psychotherapy for a short time, 1 month before she was referred to our clinic. The neurological diagnosis was acute autonomic and sensory neuropathy (AASN) as previously reported by Colan et al. (1980).

Results and Discussion

Patients with APD or AASN show severe impairments of ANS; headache, insomnia, constipation, diarrhoea, dryness of mouth, anhidrosis, fainting spells, vomiting, urinary incontinence, impotence, or visual difficulties (Young et al. 1969; Okada et al. 1988; Colan et al. 1980). Such symptoms are extremely difficult for patients to bear. They all show emotional instability. Physicians most often regard these symptoms as hysterical overacting when patients complain about their suffering, as routine laboratory findings usually show no abnormalities. Therefore, physicians refer patients to psychiatrists. Psychiatrists also generally mistake them for psychiatric disorders. Before the patients in our papers were diagnosed as APD or AASN in our clinic, their disorders were all considered to be psychiatric, such as hysterical neurosis, epilepsy, anorexia nervosa and hypochondriacal neurosis (Table 1). Case 4 (presented as anorexia nervosa) and case 6 (presented as hysterical neurosis) had received psychotherapy. Swift treatment of the physical conditions of this syndrome can avert much suffering and save many patients from a psychological degeneration that may be protracted, if not incurable. At present, it is up to psychiatrists to diagnose these symptoms correctly as APD or AASN, because patients tend to be referred to them.

Therapy for this disorder has been entirely expectant, and no specific drugs have been used on a long-term basis with these patients. However, several cases have shown some response to treatment for the relief of some autonomic nervous symptoms. The male patient of Young et al. (1969) was treated by glucocorticoid therapy and noted a dramatic increase in salivation, moisture of other mucous membranes, and return of his general feeling of well-being. In some cases (Andersen et al. 1972; Hopkins et al. 1974; Okada et al. 1975; Inamdar et al. 1982), the injection of parasympathomimetic drugs such as carbachol, bethanechol or metacholine relieved pupil, bowel and bladder dysfunctions. With the aid of mineralocorticoid therapy, postural hypotension of some cases (Young et al. 1969; Yahr and Frontera 1975; Okada et al. 1975) became less symptomatic. Complete recovery has been reported in only one case by Young et al. (1969); however, the course of the illness in other cases is usually chronic with incomplete recovery.

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