CASE REPORT

Agitation after minor trauma: combativeness as a cardinal catatonic feature

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SUMMARY

Catatonia is a syndrome of motor dysregulation, usually associated with psychiatric, neurological, systemic and drug-related diseases. Retarded and excited types exist, both of which often go unrecognised in clinical practice. We describe a 64-year-old woman who gradually developed insomnia, started communicating less, complained of feeling restless and ended up injuring relatives. Initiation of symptoms followed a fibula fracture. The patient was diagnosed with excited-type catatonia with prominent combativeness because of minor trauma and rapidly recovered after lorazepam treatment instatement. Our case demonstrates that catatonia can follow minor traumatic injury and how excited-type catatonic features may go unrecognised in general practitioner and specialist settings. Moreover, we show that catatonia may be recurrent, necessitating long-term treatment and very gradual lorazepam tapering.

BACKGROUND

Catatonia often goes unrecognised and given its presentation in a variety of medical settings it should be brought under the attention of a wide range of practitioners, not in the least because of the spectacular results that may be obtained when the appropriate treatment is instated.

CASE PRESENTATION

A 64-year-old woman without a personal or family history of psychiatric, substance-related or neurological illnesses broke her fibula late November 2010. Treatment at the emergency department consisted of immobilisation, nadroparine 0.3 cc/24 h, and a prescription of diclofenac and acetaminophen. She gradually developed insomnia, communicated less, stopped undertaking her daily (social) activities and complained feeling 'restless' all day.

Her daughter brought her to a general practitioner (GP) and a neurologist late December but all physical, lab and further examinations were normal, including a computed axial tomography (CAT) and MRI of the brain. In the following months, two psychiatrists suspected an agitated depressive disorder and prescribed mirtazapine 30 mg, which was replaced after 4 weeks by fluoxetine 20 mg owing to the lack of efficacy, with the following concomitant benzodiazepine agonists: zopiclone 7.5 mg, zolpidem 10 mg and prazepam 20 mg, sequentially given, without result. Her condition worsened as she started yelling at and hitting relatives. When her daughter brought her into our outpatient psychiatry unit mid-March 2011, the patient could not sit still and walked around

restlessly, constantly asking the same question ('what's happening?'). Besides this verbal stereotypy, the following features were observed: limb mannerisms (the patient continuously grabbed the same part of her chair), negativism (the patient refused and resisted to the physical examination) and ambitendency (when the examiner asked the patient not to shake the hand that he extended to her, the patient hesitantly touched it). No waxy flexibility was observed.

INVESTIGATIONS

Brain CAT scan, brain MRI, EEG, urine analyses (leucocytes, protein, screening for illicit drugs including cannabis, amphetamines, opioids), and all blood lab tests (liver enzymes, ureum, creatine, estimated glomerular filtration rate, Na⁺, K⁺, Ca²⁺, C reactive protein, leucocytes, thrombocytes, follicle stimulating hormone, T4, thyroid stimulating hormone and creatine kinase) were normal.

DIFFERENTIAL DIAGNOSIS

- ▶ Mood disorder: depressive disorder, single episode, with catatonic features
- ▶ Psychotic disorder, not otherwise specified, with catatonic features
- ▶ Anxiety disorder, not otherwise specified
- ▶ Delirium owing to a general medical condition

TREATMENT

We suspected an excited-type catatonic disorder because of a traumatic limb injury and started the following oral lorazepam dosing schedule: 1 mg per dose with at least 2 h in between dosages, and 2 mg at night, with a maximum of 8 mg daily.¹ During a follow-up appointment within 40 h the patient felt completely recovered and none of the aforementioned motor and verbal features were observed. Keeping the lorazepam at 5 mg a day, all other psychotropics were tapered over 12 days. As recommended for catatonic disorders because of medical illness,¹ a very gradual (here: 1-year) lorazepam tapering schedule was then started.

OUTCOME AND FOLLOW-UP

The catatonic features recurred several weeks after lorazepam tapering (ie, more than a year after the diagnosis was established) and the patient was admitted to our clinic for 2 weeks. After lorazepam was reinstated, the patient again rapidly recovered.

DISCUSSION

Our case highlights some of the caveats and pitfalls associated with excited-type catatonia following

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non-psychiatric conditions in GP and specialist settings. When psychiatric disease owing to general medical conditions is suspected, distinguishing catatonia from delirium may prove intricate. Features that may discriminate between catatonia and delirium include stereotypies, negativism, and the rapid resolution of symptoms following lorazepam administration in the event of catatonia.² Moreover, several cases of delirium have been reported to worsen after initiation of treatment with benzodiazepines,³ for example, owing to aggravating circadian rhythm reversal. Diagnosing the syndrome in outpatient settings is cumbersome given the overlap with (other) psychiatric symptoms and the lack of epidemiological data regarding its prevalence and clinical features.⁴ In particular excited-type catatonia may go unrecognised as behavioural and depressive disorders are often suspected. Moreover, combativeness—a prime feature here—is rarely recognised as part of a catatonic syndrome. In addition, contributing to the underdiagnosis of catatonia is the frequent omission of automatic obedience, negativism and ambtendency testing during physical examination.⁵ Our case further illustrates that high-dose oral lorazepam may be effective within hours for excited-type catatonia, extending previous findings of lower doses sufficing in retarded catatonia.¹ Finally, early differentiation of catatonia from other disorders is pivotal as lorazepam constitutes the only thoroughly investigated and efficacious agent for all catatonia subtypes.¹ Other benzodiazepines often prescribed for agitation may therefore not adequately reverse catatonic features.

In conclusion, delayed recognition of motor dysregulation and overlap with psychiatric features may complicate the diagnosis and treatment of excited-type catatonia in all sorts of medical settings. Overdue initiation of catatonia treatment can negatively impact prognosis,¹ a feature we believe may have played a role in the relapse observed in this patient.

Studies are needed to estimate incidence rates of catatonia following the main categories of medical illness and optimise diagnostic and therapeutic decision-making for catatonia.

Learning points

- ▶ Both excited and retarded-type catatonias exist, the latter of which often goes unrecognised.
- ▶ Excited-type catatonia can result from minor trauma such as a fibula fracture.
- ▶ To adequately diagnose catatonia, automatic obedience, negativism, waxy flexibility and ambtendency must be looked for during a physical examination.
- ▶ Lorazepam is the only investigated efficacious agent for all catatonia subtypes.
- ▶ Catatonia may follow a relapsing-remitting course, necessitating long-term treatment and very gradual tapering.

Contributors JLL has treated the patient under supervision of JVH and has written the manuscript with EHP, JVH and ME. In addition, JVH reviewed the final version. All authors declare that they have read and approved the final manuscript.

Competing interests None.

Patient consent Obtained.

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