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Review of literature

Catatonic syndrome: From detection to therapy

Syndrome catatonique : du dépistage à la thérapeutique

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ABSTRACT

Introduction. – Catatonia is a psychomotor syndrome which can include motor, mental, behavioral and vegetative symptoms. Exclusively associated with schizophrenia until the 1970s, catatonia still remains an under-diagnosed syndrome with significant morbidity and mortality.
Literature findings. – As a result of its different forms and developments, catatonic syndrome can be associated with many organic and psychiatric etiologies and confused with a variety of diagnoses. In addition to its organic complications, malignant catatonia can also be extremely severe. Several diagnostic scales are described, those of Bush and Peralta being the most widely used. Despite the recent development of the DSM-5, we can regret the lack of progress in the international classifications concerning both the recognition of the etiological diversity of this syndrome and in the clinical and therapeutic approaches to it. The diagnosis is based solely on clinical data, and needs to be completed by information from paraclinical settings, particularly with respect to detecting organic etiology. The first-line treatment is still based on the use of certain benzodiazepines or benzodiazepine-like agents such as lorazepam, diazepam and zolpidem. If the first or second line fails, or in case of malignant catatonia, electroconvulsive therapy is recommended. For the periodic form, no large-sample study has been performed on long-term treatment. A few case reports suggest the use of lithium in periodic catatonia, specifically to prevent recurrent episodes or at least to extend the inter-episode intervals. Other studies are in favor of the use of benzodiazepines, with disagreement between gradual discontinuation and long-term treatment. Concerning the management of catatonia in patients with schizophrenia, for whom first-line benzodiazepines are often insufficient, certain atypical antipsychotics such as clozapine or quetiapine appear efficient. These data are also applicable to children and adolescents.
Conclusion. – Often neglected by practitioners, catatonic syndrome remains a common entity of which it is important to be aware, especially in case of rapid installation of the symptoms. Diagnostic scales should be used and a lorazepam test should be performed to avoid delaying the diagnosis. Second-line therapy requires further study. This concerns in particular diazepam, anti-NMDA (N-methyl-D-aspartate) and rTMS (repetitive transcranial magnetic stimulation). Some specificities of catatonia, such as the periodic form and cases in patients with schizophrenia, also require further evaluations.

Mots clés :
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RÉSUMÉ

Introduction. – La catatonie est un syndrome psychomoteur qui comprend des symptômes moteurs, mentaux, comportementaux et neurovégétatifs. Exclusivement rattaché à la schizophrénie jusque dans les années 1970, ce syndrome reste sous-diagnostiqué malgré une morbi-mortalité relativement importante.

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Revue de la littérature. – Pouvant présenter différentes formes et évolutions, le syndrome catatonique s'associe à de multiples étiologies organiques ou psychiatriques et possède plusieurs diagnostics différentiels. En dehors de ses complications organiques, la catatonie maligne peut mettre en jeu le pronostic vital. Parmi de multiples échelles, celles de Bush et de Peralta sont les plus utilisées. Malgré les récentes évolutions du DSM-5, les classifications internationales ne décrivent encore que partiellement la diversité étiologique et les approches cliniques et thérapeutiques qu'elle revêt. Le diagnostic est exclusivement clinique mais peut être complété paracliniquement pour un étayage étiologique. Le traitement de première intention comprend quelques benzodiazépines ou agents apparentés qui sont le lorazepam, le zolpidem et le diazépam. En cas de forme maligne ou d'échec des traitements médicamenteux, l'électroconvulsivothérapie est recommandée. Pour la forme périodique, aucune étude à grande échelle n'étaye la prise en charge au long cours. Quelques études de cas suggèrent pour cette forme l'emploi du lithium pour prévenir un épisode ultérieur ou tout au moins pour élargir les intervalles de temps inter-épisodiques. D'autres études de cas suggèrent plutôt l'emploi de benzodiazépines au long cours tandis que d'autres rapportent l'intérêt d'une suspension progressive. Dans le cas du syndrome catatonique chez les patients atteints de schizophrénie, seuls quelques antipsychotiques atypiques comme la quétiapine et la clozapine semblent montrer une réelle efficacité. L'ensemble de ces données seraient applicables chez l'enfant et l'adolescent.
Conclusion. – Souvent négligé par le praticien, le syndrome catatonique reste une entité importante à connaître, en particulier lors d'une installation symptomatologique rapide. Au moindre doute, une échelle diagnostique peut être utilisée et un test au lorazepam réalisé. L'étude des autres traitements pharmacologiques devrait être approfondie et concerne le diazépam et les antagonistes des récepteurs au NMDA (N-methyl-D-aspartate). L'usage de la r-TMS (stimulation magnétique transcrânienne répétée) devrait lui aussi être davantage évalué. Il en est de même pour les spécificités des formes périodiques et de la catatonie chez les patients atteints de schizophrénie.

1. Introduction

Catatonia, which was initially described by Kahlbaum in 1874, is a psychomotor syndrome that may include motor, psychic, behavioral and neuro-vegetative symptoms. After it was initially included by Kraepelin in the category of early dementias, and then by Bleuler in a group associated with schizophrenia, it was not until the 1970s that the exclusive association with schizophrenia was called into question. This pathology, occurring in a wide range of psychiatric and medical conditions [1] with a prevalence estimated to be between 7.6% and 38% among psychiatric patients [1], is as yet under-diagnosed [1,2] with a frequent risk of inappropriate initial treatment and delay in instating the appropriate therapy [1]. In addition, between 15% and 30% of catatonic patients do not respond to the medications at present in use, in particular benzodiazepines, and require more efficient therapy [3]. We can deplore the insufficient advances to date concerning both the recognition of the etiological diversity of catatonia and the clinical and therapeutic strategies required, as well as the absence of any consensus in the international classifications (DSM, ICD) [4,5]. This situation could well favor misapprehensions among practitioners.

2. Present knowledge

The two clinical forms described are retarded/stupor catatonia and excitement catatonia. As the symptoms are rarely permanent over 24 hours, observation is required over an extended period. The alternation between the two forms is fairly frequent [1]. The intensity of symptoms distinguishes the malignant, potentially lethal form and the benign form [1]. Likewise, evolution can be acute, periodic or chronic [1]. According to Harvey et al. [6], the periodic form often goes unnoticed. It is characterized by the repetition of acute episodes of catatonia, alternating stupor or catatonic excitement indifferently. Although there may be cases of residual, inter-episode catatonic symptoms, these patients are generally asymptomatic between episodes [7]. Catatonic syndrome is also observed relatively frequently among children and adolescents [8]. The data presented in this article is also valid in this population, where the clinical criteria and treatment appear to be the same as in adult populations [8]. To date, no diagnostic scale specific to children and adolescents has been published, and no specific treatment in this population is described in the medical literature.

Among the risk factors, the use of a neuroleptic treatment and a history of neuroleptic malignant syndrome (NMS) can be noted [9]. Conversely, catatonia is a risk factor for NMS [10], which, according to some authors, could be a pharmacologically-induced subtype of catatonia [11]. Likewise, in presence of catatonia, the instatement of a neuroleptic treatment could increase the risk of NMS [1].

Numerous neurological pathologies can cause catatonia [6], and also certain infectious, metabolic, toxic, auto-immune and idiopathic pathologies [1,6]. In addition, a bout of catatonia can occur in association with NMS [12], with a serotoninergic syndrome [6], or with the discontinuation of a sedative treatment, among which lorazepam [13]. Catatonia can co-occur with a depressive syndrome, a manic or mixed state, a disorder in the schizophrenia spectrum, a severe obsessive-compulsive disorder or a disorder in the autistic spectrum [1]. Catatonic stupor in association with manic symptoms can make diagnosis more complex and indicate a mixed syndrome [14]. According to Rosebush and Mazurek [15], the distribution of concurrent or underlying diagnoses relates above all to affective disorders (46%) and schizophrenia (20%). Functional psychiatric illnesses such as catalepsy in conversion disorder are the main differential diagnoses [6]. Likewise, NMS can be confused with malignant catatonia [16], and melancholy stupor and anxious sideration can be confused with catatonic stupor [1].

Catatonia can be life-threatening, since in some forms it affects food and fluid intake, and motor function. The frequent organic complications include starvation, muscular contracture, decubitus ulcer, acute kidney or liver failure, sepsis, convulsions, pneumonia, pulmonary embolism, and adult acute respiratory distress syndrome [13,17]. Evolution towards malignant catatonia is characterized by three factors: severe rigidity, autonomic nervous system involvement and altered consciousness. Before the use of electroconvulsive therapy in this indication, most patients with malignant catatonia died, with a global mortality rate of over 31% [18].

From the etiopathogenic viewpoint, catatonia could result from hypoactivity of the GABA-A receptors and the dopaminergic system, and possibly from hyperactivity of the glutamatergic system

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