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CASE REPORT

Vocally disruptive behavior: A case report and literature review

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Vocally disruptive behavior (VDB) is a common and particularly difficult symptom to manage in dementia. VDB is usually considered collectively with agitation and aggression as a component of behavioral and psychological symptoms in dementia and is therefore poorly understood as an individual symptom. A review of the literature is described where VDB as a challenging behavior has been individually examined as a symptom. A case of VDB occurring in patient with dementia is described where the patient's repetitive vocalizations responded to treatment with pregabalin. This has not been previously reported in the literature. The prevalence of VDB, the factors associated with it and the current management guidelines for clinicians are outlined with a review of the drug treatment strategies for VDB. Pregabalin with its unique pharmacological profile and excellent tolerability should be considered as a possible treatment for VDB where drug treatment is indicated.

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Introduction

It is well recognized by clinicians who treat behavioral and psychological symptoms in dementia (BPSD) that repetitive vocalizations are a common and particularly challenging symptom to manage. Vocally disruptive behavior (VDB), the term commonly used in the literature to describe these symptoms, has received limited attention as an independent symptom on its own and is usually considered alongside agitation and aggression (Beck *et al.*, 2001; Nagaratnam *et al.*, 2003). VDB refers to verbal or vocal behaviors that are either repetitive, disruptive or inappropriate to the circumstances in which they are manifested (Cohen-Mansfield and Werner, 1997). These may include the persistent repetition of words and phrases, swearing, grunting and bizarre noise making such as weird laughing or crying (Nagaratnam *et al.*, 2003), and most commonly screaming (Locke and Mudford, 2010). There is a clear consensus that VDB is a common and particularly difficult clinical issue for patients and their carers. There is a dearth of research guiding successful intervention (Cohen-Mansfield and Werner, 1997; von Gunten *et al.*, 2008).

VDB may present in a variety of care settings and can be particularly challenging to manage in congregated residential settings. In nursing homes, inappropriate vocalizations can have a deleterious effect on other residents, their carers and visitors (Bang *et al.*, 2008). Formal care staff may feel stressed and avoid the resident, or move the resident to a more restricted level of care (Barton *et al.*, 2005). Other residents may, in response, become annoyed, stressed and agitated and vocalize disruptively themselves. This may lead to family dissatisfaction with the care milieu (Sloane *et al.*, 1997).

There is a lack of a uniform consensus definition for VDB in the research literature (CohenMansfield *et al.*, 1990; CohenMansfield and Werner, 1997; Dwyer and Byrne, 2000; von Gunten *et al.*, 2008; Locke and Mudford, 2010). This has led to the wide range of prevalence rates for VDB found in nursing home studies of between 10% and 30% (CohenMansfield *et al.*, 1990; Sloane *et al.*, 1997). Regardless of the definition, these behaviors invariably occur more often in advanced disease (Dwyer and Byrne, 2000; Beck *et al.*, 2001). As cognitive abilities deteriorate, vocalizations are more challenging to manage, as they become less verbal and therefore less intelligible, and less clearly related to specific needs or purposes (Magri *et al.*, 2007). Non-verbal (unintelligible) VDB are more prevalent and more disruptive than verbal forms (von Gunten *et al.*, 2011).

VDB in dementia may be associated with the underlying illness pathology, the individual patient characteristics and/or environmental factors. Attempts to examine associations of VDBs with specific brain

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regions and neurotransmitter systems have yielded few firm conclusions (Nagaratnam *et al.*, 2003). Neuroanatomical correlates implicated include the orbitofrontal and the dorsolateral pre-frontal cortex, interruption of the fronto-subcortical circuitry and thalamic lesions which mimic frontal lobe dysfunction (Nagaratnam *et al.*, 2003). Lesions in these areas can lead to disinhibited behavior and deficits in decision-making (Sloane *et al.*, 1997; Yusupov and Galvin, 2014). Neurotransmitters such as serotonin, acetylcholine and dopamine have also been implicated (Gauthier *et al.*, 2010). While the GABAminergic system has not specifically been associated with VDB, it has been linked with severe behavioral problems in dementia (Lanctôt *et al.*, 2004). It is likely that multiple neurotransmitter systems are involved, and that there is no straightforward connection with the neurobiology and pathology of dementing disorders (Tible *et al.*, 2018), which may explain why no single drug has proven effective (Nagaratnam *et al.*, 2003).

Previous or concomitant diagnosis of psychiatric illness is considered one of the more important predictors of VDB (von Gunten *et al.*, 2011). Affective disorders (Dwyer and Byrne, 2000; von Gunten *et al.*, 2011), anxiety (Barton *et al.*, 2005), psychological distress especially where due to separation and attachment issues (Tible *et al.*, 2017, 2018), and psychotic symptoms (Sloane *et al.*, 1997) have been associated with VDB. In addition, pre-morbid personality characteristics such as introversion and rigidity may be relevant (von Gunten *et al.*, 2011; Tible *et al.*, 2018). The occurrence of aggressive behaviors prior to entering the nursing home is also associated with VDB (Cohen-Mansfield *et al.*, 1990). In patients with dementia, greater cognitive, functional, communication and physical impairments have all been associated with increased rates of VDB (Cohen-Mansfield *et al.*, 1990; Sloane *et al.*, 1997; Dwyer and Byrne, 2000; von Gunten *et al.*, 2011). Physical illness associations include pain and discomfort (Cohen-Mansfield *et al.*, 1990), delirium, seizures (Nagaratnam *et al.*, 2003) and sensory problems such as hearing deficit (von Gunten *et al.*, 2011).

Environmental factors associated with increased rates of VDB include over and under stimulation (Cohen-Mansfield and Werner, 1997; Sloane *et al.*, 1997; von Gunten *et al.*, 2011), and unmet care needs (Beck *et al.*, 2001). Other residents vocalizing can contribute to the risk of a person developing VDB (Cohen-Mansfield *et al.*, 1990). VDB may be precipitated by interventions assisting the patient with activities of daily living such as bathing, eating and toileting (Cohen Mansfield *et al.*, 1990). 'Sundowning'-associated increase in VDB may be related to overstimulation due to visitors and nursing staff changeover (Barton *et al.*, 2005). In the nursing home setting the person who typically repetitively vocalizes is a female, with advanced dementia, broken sleep and who requires full nursing care (Cohen-Mansfield *et al.*, 1990). Very often, however, there may not be any particular obvious trigger or temporal pattern evident when assessing this problem.

Repetitive vocalization behaviors are not exclusive to dementia sufferers. They may occur in a variety of other clinical contexts including intellectual disability (Matson *et al.*, 2011), autistic spectrum disorders (Lanovaz *et al.*, 2011), personality disorders and psychotic depression (Sloane *et al.*, 1997). They have been described in palliative care settings (Adams *et al.*, 2012) and in patients with acquired brain injury, frontal lobe impairments (Sloane *et al.*, 1997) and epilepsy (Enatsu *et al.*, 2011).

We present a case of VDB in advanced dementia, comorbid with a number of other risk factors and associations. We describe successful treatment with pregabalin which is a novel therapeutic approach.

Case report

Mrs M, an 88-year-old woman residing at home, was referred to our psychiatry of old-age service by her primary care physician for assessment and management of agitation, including loud regular vocalizations. Her husband was stressed by the agitation, and her formal care providers were considering resigning as a result of the VDB.

Mrs M had a background history of advanced dementia of probable mixed etiology. She had vascular risk factors for dementia including atrial fibrillation and was on treatment with warfarin. She had been treated for depression during the early stage of her cognitive decline with a good response to citalopram. She had been taking donepezil for 10 years before it was discontinued when she progressed to advanced dementia. She also had a history of pulmonary embolus, osteoarthritis, mild renal impairment and recurrent urinary tract infections (UTIs). There was no history of any seizure disorder. She had a PEG tube *in situ*, which had been placed 1 year previously while she was an in-patient in a medical facility, due to recurrent aspiration pneumonia. She continued to regularly require treatment for aspiration pneumonia, as well as UTI. She was on the following psychotropic medication: citalopram 20 mg, zolpidem 5 mg and alprazolam 0.25 mg 'as required'.

Mrs M was assessed in her home in March 2016. She was living with her husband and was receiving formal care 24 hours a day in the home. She was unable to communicate verbally. The carers reported she had been screaming at night for months, usually from 2 to 4 a.m. The vocalizations were non-verbal. The presence

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or absence of her husband at night did not appear to alter the screaming. There was no associated aggression.

Informant history revealed that her depressive illness had relapsed after citalopram was withdrawn the previous year, and that she had responded well to its reintroduction. Her mood was now reported as normal, responding positively to her husband and carers by day and with no behaviors such as crying or resisting care that might indicate depressed mood. She did not appear to be responding to hallucinations. There was no evidence of any active infective process. She had been in receipt of long-term regular opiate analgesia for the relief of pain secondary to osteoarthritis and her pain was well controlled. She did not appear to be experiencing pain at the time of her assessment and her carers did not feel pain was a reason for her shouting. She was not constipated. 'As required' administration of alprazolam was effective in reducing the vocal behavior for short periods of time only.

Gabapentin was commenced at a dose of 100 mg nocte and the VDB reduced in frequency and severity. It had to be discontinued as she developed myoclonic jerking. Mirtazapine 7.5 mg was then introduced for its sedative and anxiolytic effect. However, Mrs M developed restless legs and more pronounced myoclonic jerking, and it was discontinued.

A trial of pregabalin 50 mg nocte was started and within 2 days her husband and the carers reported a significant reduction in agitation and shouting in both frequency and intensity. Objectively, Mrs M had also improved, appearing more alert and relaxed, and she did not appear to be sedated. Her sleep had improved significantly. After 2 weeks of treatment, the screaming had profoundly diminished. The improvement was maintained over the next 9 months, at which point she was discharged from the service.

Discussion

The consensus approach to the management of VDB (Sloane *et al.*, 1997) should comprise careful and thorough assessment with problems identified and then addressed individually. Barton *et al.* (2005) set out a hierarchical approach to management, where the patient's physical and mental health are considered first, before advancing through more complex approaches including considering the function or hidden meaning of the VDB. Tible *et al.* (2018) have proposed understanding VDB within the frame of phenomenological diagnosis, where specific aspects of the VDB may be understood as being related to the patient's life history. Including the patient's family or care team in the assessment process is essential in analyzing the possible meaning behind VDB (Tible *et al.*, 2017). Contributory factors to VDB need to be considered individually and as many factors may be associated, a single therapeutic approach is likely to have a limited efficacy (von Gunten *et al.*, 2008, 2011).

Management plans should be tailor made and individualized (Cohen-Mansfield and Werner, 1997; Magri *et al.*, 2007), with a clinical approach matched to the individual behavioral symptoms and needs of the patient (Gauthier *et al.*, 2010; Tible *et al.*, 2017). Thorough physical assessment is an essential first step in the management of VDB, particularly focusing on the assessment of pain or discomfort (Cohen-Mansfield and Werner, 1997).

Therapeutic approaches examined are traditionally categorized as psychosocial or biological. As with neuropsychiatric symptoms in dementia generally, non-pharmacological interventions should be attempted first, but are often underused (Sloane *et al.*, 1997; von Gunten *et al.*, 2011). Evidence for individual non-pharmacological interventions is generally weak, often due to heterogeneous research methods, but efficacy of these approaches is supported by long-standing clinical experience (Tible *et al.*, 2017). Trying non-pharmacological interventions prompts the consideration of secondary and modifiable causes for the VDB, and has the significant advantage of avoiding the side effects associated with psychotropic medication (Sloane *et al.*, 1997).

Non-pharmacological interventions should be person-centered (Magri *et al.*, 2007). Various approaches have been described including: one-to-one attention, playing a video of loved ones (Cohen-Mansfield and Werner, 1997), music (Locke and Mudford, 2010), sensory stimulation (von Gunten *et al.*, 2008), anxiety management techniques such as deep breathing and counting (Yusupov and Galvin, 2014). A number of behavioral strategies have been recommended including contingent reinforcement for quiet time (von Gunten *et al.*, 2008), graded desensitization (Adams *et al.*, 2012) or even direct sensory feedback using microphone and earphones (Sloane *et al.*, 1997).

Staff training and education are essential to ensure consistent application of individual interventions (Sloane *et al.*, 1997; Magri *et al.*, 2007). In addition, a successful approach requires a system for monitoring the effect of any intervention applied (Sloane *et al.*, 1997). Psychological and milieu therapies have been successfully applied (Dwyer and Byrne, 2000). Very occasionally, isolation of the vocalizer from other residents may be necessary (Sloane *et al.*, 1997), though this could also exacerbate VDB as perceived isolation may be a factor in the development of the problem in the first place.

Patients with higher levels of cognitive function respond better to psychological interventions than patients with more advanced dementia (Bédard *et al.*,

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2011). In the event of limited success of non-pharmacological measures, it is appropriate to consider the addition of psychotropic medication while continuing the non-pharmacological treatment.

Pharmacological treatment options need careful consideration of the risks and benefits prior to prescribing, while use should be limited in time and stopped after gradual reduction when symptoms improve (Tible *et al.*, 2017). The ideal choice of psychotropic drug would be an agent with high efficacy and few adverse effects (AEs), with treatment continuing for the shortest time possible (Gauthier *et al.*, 2010). The evidence base to guide clinicians regarding individual drugs is weak as the majority of studies are observational or based on case reports (Sloane *et al.*, 1997). Furthermore, much of the literature on medication in dementia relate to behavioral disturbances in general with little information on specific target symptoms like VDB (Nagaratnam *et al.*, 2003).

Low serotonin levels found in dementia have been linked to loss of impulse control, providing a rationale for the use of serotonin enhancing drugs such as SSRI's, trazodone and buspirone (Sloane *et al.*, 1997; Magri *et al.*, 2007) in this context. Antipsychotics have been studied more robustly and have been shown to confer significant benefit for the short-term treatment of neuropsychiatric symptoms generally, but they have the potential to cause serious AEs (Gauthier *et al.*, 2010) and may be poorly tolerated (Nagaratnam *et al.*, 2003). Studies examining anti-epileptics have suggested potential usefulness for VDB (Cooney *et al.*, 1996; Lanctôt *et al.*, 2004; von Gunten *et al.*, 2011). Patients with VDB in dementia often have a severity of cognitive impairment that excludes treatment with cholinesterase inhibitors according to current protocols (Barton *et al.*, 2005). A study examining VDB in institutionalized elderly patients found evidence of frequent prescribing of multiple agents with as many as five psychotropic drugs used in individual patients (von Gunten *et al.*, 2011). A number of case reports have described the effective use of ECT in patients with VDB in dementia which has not responded to less-invasive interventions (Bang *et al.*, 2008). The evidence base for ECT in this context is very limited and does not support its clinical use in managing VDB.

In our case report, there is a strong temporal relationship between the initiation of pregabalin treatment and the reduction in screaming within 2 days. This corresponds with the pharmacokinetic profile of pregabalin where a steady-state drug level is achieved within this time profile (Baldwin and Ajel, 2007). This supports the efficacy of the drug in this clinical context, particularly as there were no other changes in circumstances or treatments which could explain the improvement. Withdrawal and rechallenge of the drug to test its efficacy was not considered ethical in view of the very significant improvement and the previously unremitting nature of the VDB, and therefore was not attempted. There was no other plausible explanation for the very significant reduction in her repetitive vocalizations.

There is a paucity of literature on the use of pregabalin in people with dementia. It has been used successfully in the treatment of pain in patients with Lewy Body Dementia (Ukai *et al.*, 2017). Pain is generally under recognized and under treated in dementia. VBD and other non-verbal behaviors such as grimacing or restlessness may often be an expression of pain (Flo *et al.*, 2014). Pregabalin has been described in the management of refractory anxiety in dementia (Hulstaert, 2014). It is recognized that pregabalin is being increasingly prescribed off-license in low doses in people with dementia to treat agitation, particularly where it is thought to be secondary to anxiety (Crowther, 2013). The possible mechanisms of action in our patient include both pain relieving and anxiolytic effects.

When compared with the existing pharmacological treatments outlined above, pregabalin may offer several advantages including safety and tolerability. Pregabalin is an α2δ ligand similar in structure to gamma-aminobutyric acid (GABA) that is not active at GABA receptors (Tassone, 2007) or glutamate receptors and has no effects on the reuptake of 5-HT (Baldwin and Ajel, 2007). Pregabalin binds the α2δ subunit of voltage-sensitive calcium channels leading to reduction in neurotransmitter release (Stahl, 2004). A decrease in release of a wide range of neurotransmitters, including glutamate, norepinephrine, substance P and calcitonin has been described (Baldwin and Ajel, 2007; Tassone *et al.*, 2007) and is the mechanism of action thought responsible for the anticonvulsant, anxiolytic and chronic pain relieving actions of pregabalin (Stahl, 2004).

Pregabalin does not bind to plasma proteins and so does not displace protein-bound drugs. It readily crosses the blood–brain barrier and is minimally metabolized with renal excretion. Pregabalin lacks activity at the hepatic cytochrome P450 enzymes and has very few drug–drug interactions (Baldwin and Ajel, 2007). It displays linear gastrointestinal absorption leading to a predictable dose–response relationship (Semel *et al.*, 2010). The elimination half-life is approximately 6 hours, and steady-state concentrations are achieved within 2 days of initiation of therapy. Drug clearance may be lower in elderly patients due to age-related reduction in renal function (Tassone *et al.*, 2007).

AEs of the central nervous system such as somnolence and dizziness are the most commonly reported. These effects appear to be dose related and not related

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to age (Tassone *et al.*, 2007). The majority of AEs are mild to moderate in intensity and transient, with tolerance developing within the first 3 weeks of treatment (Montgomery *et al.*, 2008). Efficacy and safety of pregabalin in older patients are comparable to younger patients, and AEs can be minimized by initiating at low doses and titrating slowly (Semel *et al.*, 2010).

VDB deserves attention and research as a symptom in its own right, aside from BPSD generally. To our knowledge, there is no existing literature describing the use of pregabalin for VDB. Future research in VDB should be directed toward development of standardized approaches to assessment and therapy. There is a dearth of good quality research examining pharmacological agents for VDB, and a concerted effort will be required to address this deficit in our knowledge base. With its relatively benign side effect profile and favorable pharmacodynamic properties, pregabalin if effective would be an ideal drug to treat VDB where non-pharmacological measures have failed. The current evidence base to support its use is limited and further research is needed to establish its effectiveness before any recommendations can be made regarding its place in the drug treatment of VDB.

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Conflict of interest

The authors [BM, AB, SM, CC] have no conflicts of interest to disclose.

Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committee on human experimentation with the Helsinki Declaration of 1975, as revised in 2008. The authors assert that ethical approval for publication of this case report was not required by their local Ethics Committee. Written informed consent to publish the case report was obtained from the patient's next-of-kin.

Description of authors' roles

BM collected the data, reviewed the literature and wrote the article. AB and SM assisted with collecting the data and writing the article. CC assisted with reviewing the literature and writing the article.

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