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Does the Etiology, Phenomenology and Motor Subtype of Delirium Differ When It Occurs in Patients With An Underlying Dementia?: A Multi-Site, International Study

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Abstract

Objectives: To compare the etiology, phenomenology and motor subtype of delirium in patients with and without an underlying dementia. **Methods:** A combined dataset (n = 992) was collated from two databases of older adults (>65 years) from liaison psychiatry and palliative care populations in Ireland and India. Phenomenology and severity of delirium were analysed using the Delirium Symptom Rating Scale Revised (DRS-R98) and contributory etiologies for the delirium groups were ascertained using the Delirium Etiology Checklist (DEC). Delirium motor subtype was documented using the abbreviated version of the Delirium Motor Subtype Scale (DMSS4). **Results:** Delirium superimposed on dementia (DSD) showed greater impairment in short term memory, long term memory and visuospatial ability than the delirium group but showed significantly less perceptual disturbance, temporal onset and fluctuation. Systemic infection, cerebrovascular and other Central nervous system etiology were associated with DSD while metabolic disturbance, organ insufficiency and intracranial neoplasm were associated with the delirium only group. **Conclusion:** The etiology and phenomenology of delirium differs when it occurs in the patient with an underlying dementia. We discuss the implications in terms of identification and management of this complex condition.

Keywords

delirium, dementia, cognitive impairment, delirium motor subtypes

Introduction

Delirium occurring in the presence of an existing dementia is known as Delirium superimposed on Dementia (DSD). It is a common and under recognised condition,[1] with estimated prevalence rates of between 22% and 89% in elderly hospital and community populations.[2] It contributes significantly to patient and carer distress,[3] worsening of cognition[4] and risk of mortality.[2]

The diagnosis of DSD can be challenging, especially in those with advanced dementia who have multiple existing cognitive deficits[4,5] and in conditions such as Lewy Body Dementia that have significant clinical overlap with delirium in terms of symptomatology.[6] Generally, the acute

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onset of cognitive symptoms including impaired attention, confusion and arousal in conjunction with non-cognitive symptoms such as hallucinations, delusions and altered sleep wake cycle have been shown to be good detectors of delirium in patients with existing dementia.[7-10] However, less is known as to whether DSD differs in terms of its presentation and etiology from delirium occurring in patients who do not have an underlying dementia. These differences, if present, are important to understand in aiding the diagnosis and management of this condition, which comes with a high rate of mortality and morbidity.[3,4]

Despite an increased focus on this area in recent years, studies comparing the etiology and phenomenology of DSD to delirium have shown mixed outcomes. This may be due in part to the use of a range of different tools and this research taking place in different clinical settings (e.g. palliative, ICU, surgical). Some research has shown that DSD is more severe with a greater degree of agitation, hyperactivity, perceptual disturbance and disorientation when compared to those with delirium only[11-13] while others have failed to replicate these findings.[7,10,14] Meagher et al. (2010)[7] found that measures of orientation and attention were significantly worse in those with DSD when compared to delirium alone. In contrast, Leonard et al (2016)[10] found similar cognitive profiles across all delirium (DSD and Delirium only) groups but that these groups were distinguished from controls and those with dementia in performing poorer on tests of attention and vigilance. However, thus far large-scale studies with participants from different clinical settings comparing the phenomenology of DSD to delirium in terms of cognitive and non-cognitive symptoms have been lacking.

Dementia or pre-existing cognitive impairments are known risk factors for delirium.[15,16] It has therefore been postulated that those with dementia require a lower level of physiological insult to develop delirium when compared to younger patients without cognitive impairments[13] but this has yet to be properly established. It is important to understand how etiologies trigger delirium superimposed on dementia and the nature of these etiologies as they may be treatable and help to lower morbidity and distress.[17] However a recent review[5] has pointed to a lack of research in investigating contributory etiologies in DSD and we aim to address this issue in the current study.

Many recent studies have looked to break delirium down into motor subtypes as a way of facilitating more accurate diagnosis and management.[18,19] Hypoactive delirium has been associated with older age, increased medication exposure, increased risk of being missed or misdiagnosed and a greater risk of morbidity and mortality, while hyperactive delirium has been linked to younger patients and carries a better prognosis.[18] It is therefore important to establish whether motor subtype differ in those with an underlying dementia, as this is a vulnerable group where delirium assessment and management can prove particularly challenging.

The aims of the current study are twofold: 1. To compare delirium superimposed on dementia to delirium only in terms of phenomenology, etiology and motor subtype. 2. To compare the phenomenology of the delirium superimposed on dementia and the delirium only group to those with dementia only and cognitively intact controls.

Methods

Subjects and Design

This study used a retrospective cross-sectional design and combined dataset of existing related databases from two countries, Ireland[20-23] and India.[24-27] All research took place between 2008 and 2020 inclusive. Raters for data included in this study were trained by an expert (DM), through training workshops in both Ireland and India using well validated assessment methods for these clinical populations as outlined below. Research was conducted across palliative care and liaison psychiatry settings. Only patients over the age of 65 were included in our analysis. Table 1 shows demographic and clinical data for the sample.

Palliative Care

This group consisted of consecutive referrals to a liaison psychiatry service in a hospice setting that were subsequently diagnosed as having delirium, dementia, DSD or cognitively normal controls at the time of assessment. Research took place at Milford Hospice in Limerick, Ireland.

Liaison Psychiatry

This group consisted of consecutive referrals to liaison Psychiatry services for those aged >65 years in two Irish Hospitals; University Hospital Limerick and University Hospital Galway and to a liaison Psychiatry service at the Postgraduate Institute for Medical Research (PGIMER) in Chandigarh, India.

Assessment

Overview of Training in Rating Scales and Assessment. Raters for data included in this study were trained by an expert (DM), through training workshops in both Ireland and India. He was involved in developing several of the tools used in this study (See below). Data between the studies was combined and harmonised as outlined below.

Delirium Diagnosis

Delirium was diagnosed according to DSM-IV[28] criteria using all available clinical information including patient

Table 1. Demographic and Clinical Data for Overall Population and Sub Populations.

	Total sample (n = 992)	Palliative care (n = 249)	Liaison Psychiatry (n = 743)	*P*-Value
Age (Mean, SD)	77.2 (7.3)	75.9 (6.2)	77.7 (7.6)	.00
Sex (%Female)	474 (48%)	120 (48%)	354 (48%)	.83
Dementia (Total, %)	346 (35%)	87 (35%)	259 (35%)	.98
Psychotopics (%)	548 (55%)	152 (61%)	396 (53%)	.30
Total medications (SD)	8.6 (4.6)	9.2 (3.9)	8.4 (4.8)	.03

P value <.05 for statistical significance, SD = Standard deviation.

assessment and collateral information from nursing staff, family and the patient's medical records.

Dementia Diagnosis

Dementia status was determined according to (i) a pre-existing diagnosis, or (ii) on the basis of detailed history and examination taking in aspects such as the patient's functional ability, neuroimaging and cognitive decline prior to assessment and was made in accordance with DSM IV.[28]

The Short Form of the Informant Questionnaire on Cognitive Decline in the Elderly (Short IQCODE)[29] was also used to aid in dementia diagnosis. The Short IQCODE comprises a 16-item informant questionnaire comparing the individual's ability on day-to-day items with their performance ten years ago. It has been validated across a wide range of populations and educational abilities.[30] Items are scored by the rater from 1 = much improved, 3 unchanged to 5 = much worse. Items include aspects of daily life such as the ability to remember conversations that happened the previous day, address and telephone numbers and where things are kept. Scores are added up and divided by the total number of questions (n = 16). The resultant score ranges from 1-5. All databases in this analysis that used the IQCODE score to aid in dementia diagnosis used a cut off of ≥3.5 to signify likely dementia. This was then combined with all relevant clinical information to make a diagnosis.

Delirium Symptomatology and Severity

Delirium severity and symptomatology were measured using the Delirium Rating Scale Revised-98 (DRS R98).[31] The DRS-R98 is a 16-item clinician rated scale used to rate the severity of delirium both overall and on a broad range of neuropsychiatric and cognitive symptoms. It is made up of 13 severity items and 3 diagnostic items. Each item is rated 0 (absent/normal) to 3 (severe impairment), with descriptors attached to each severity level. The severity scale (items 1-13) range from a score or 0-39 with larger scores indicating more severe delirium. A score of over 15 points typically indicates delirium and 18 points indicates delirium when dementia is present. The DRS-R98 can be divided into non-cognitive (items 1-8) and cognitive (9-13) subscales based on construct validity. It has been validated across clinical settings and has been shown to have a high interrater reliability, sensitivity and specificity at detecting delirium.[7,10,21,23,32] It has also been used to determine differing symptomatology and severity across delirium motor subtypes.

Delirium Etiology

Contributory etiologies were identified using the Delirium Etiology Checklist (DEC).[32] The Delirium Etiology Checklist is a 13-item checklist that is designed to document the etiological underpinnings contributory a delirium episode. Its thirteen categories are: drug intoxication, drug withdrawal, metabolic/endocrine disturbance, traumatic brain injury, seizures, infection (intracranial), infection (systemic), neoplasm (intracranial), neoplasm (systemic), cerebrovascular, organ insufficiency, other CNS and other systemic. Other CNS includes neurological conditions such as Parkinson's disease and Multiple Sclerosis while other systemic includes conditions such as post-operative state, immunosuppression or heat stroke. These categories are present to allow the rater to list contributory etiologies that do not fit under the other 11 items.

Each etiology is rated on a scale according to its likelihood of being contributory to the delirium. This scale ranges from 0 = ruled out/not present/not relevant to 4 = Definite cause. Raters can therefore document multiple etiologies and their level of contribution to a particular episode of delirium. For the purposes of this study, DEC items were broken down into two categories: present and possibly/probably contributory (scores of 3 or 4) and non-contributory (score 0-2). This also allowed for the total and mean number of likely contributory etiologies to be calculated and compared between delirium motor subtypes.

Delirium Motor Subtypes

Delirium motor subtypes were identified using the abbreviated version of the Delirium Motor Subtype Scale

(DMSS4).[33] The DMSS4 comprises two items taken from the original Delirium Motor Checklist (DMC)[34] and Delirium Motor Subtype Scale (DMSS)[35] denoting hyperactivity and two items denoting hypoactivity, with mixed subtype defined as the present of both hyperactive and hypoactive criteria. No subtype describes a presentation in which neither the criteria for hyperactive nor hypoactive delirium are fulfilled. It was developed for quick and accurate identification of delirium motor subtype in busy clinical practice and has shown a good concordance with the original DMC scale and the DMSS and has been widely validated across a variety of populations and clinical settings[8,10, 20-23].

Ethical Approval

Because of the non-invasive nature of the study, approval was given by the Limerick Regional Ethics Committee to augment patient assent with proxy consent from next of kin (where possible) or a responsible caregiver for all participants in accordance with the Helsinki Guidelines for Medical research involving human subjects. All of the Indian studies took place at Postgraduate Institute of Medical Education and Research, Chandigarh in North India. Subjects were patients referred to an inpatient psychiatric liaison service and diagnosed with delirium as per the DSM-IV criteria. The studies were approved by the Ethics Review Committee of the Institute. Written consent was obtained from the primary caregivers of the patients and patients themselves wherever possible in accordance with the Helsinki Guidelines for Medical Research involving human subjects.

Statistical Analysis

Statistical analysis was conducted using the SPSS v25 package. Continuous normally distributed variables are reported as means plus standard deviation, while categorical variables are reported as counts and percentages. Normally distributed data were analysed using Analysis of Variance (Anova), whereas non-parametric data were compared using Kruskal Wallis and chi-squared tests. Adjusted residuals were used for post-hoc analysis of chi-squared tests, with a level of greater than 1.96 (2.0 is used by convention) used to indicate statistical significance. (Agresti, 2003).[36] Dunn multiple comparison test was used for post-hoc analysis of the Kruskal Wallis test, while the Bonferroni procedure was used during post hoc analysis for ANOVA.

Results

The sample comprised 992 individuals, 518 (52%) male and 474 (48%) females. The mean age (standard deviation) of the sample was 77.3 (7.2). Demographics of each of the four groups is outlined in table 2.

There was a significant difference between groups in terms of age, with dementia and delirium superimposed on dementia groups being significantly older than delirium only and cognitively intact control groups. The delirium only group had the greatest percentage of males, whereas pure dementia had the greatest percentage of females. Those with dementia only had the highest mean number of medications prescribed, whereas delirium superimposed on dementia had the highest DRS-R98 total scores, severity scores and scores for cognition. This indicates a greater degree of impairment and greater symptom severity in these domains. The Delirium only group showed greater severity of non-cognitive symptoms (items 1-8), however this was not statistically significant.

Table 3 show a breakdown of DRS-R98 scores between each of the four groups included.

DSD and Delirium

DSD showed greater impairment on short term memory, long term memory and visuospatial ability than the delirium only group. The delirium only group showed significantly greater severity in perceptual disturbance, motor agitation, temporal onset and fluctuation than DSD. In contrast, both delirium and delirium superimposed on dementia scored significantly higher than dementia only and control groups on most DRS-R98 items.

DSD and Dementia

DSD differed from dementia across a wide range of cognitive (#1-8) and non-cognitive items (#9-13) on the DRS-R98. In terms of cognition, DSD showed significantly greater impairment in orientation, attention, and short-term memory but there were no significant differences in measures of long-term memory and visuospatial ability. DSD showed significantly greater severity than dementia on all non-cognitive items (#1-8).

Table 4 outlines the principle etiologies of the DSD and Delirium groups.

Only those who had delirium present (n = 734) were included in the group assessed with the DEC. This group was made up of 402 males and 332 females, with a mean age of 76.7 (7.3). More than one contributory etiology was listed in 561 (76%) number of cases. In terms of listed etiologies, delirium superimposed on dementia was significantly associated with systemic infection (57% of cases), Cerebrovascular (24% of cases) and other CNS etiologies (18% of cases) when compared to the delirum only group. The delirium only group was significantly associated with metabolic disturbance (60% of cases), intracranial neoplasm (7% of cases) and organ insufficiency (33% of cases). A significantly

Table 2. Demographics and DRS-R98 Totals for Each Group.

	Delirium (n = 485)	Dementia (n = 97)	DSD (n = 249)	Controls (n = 161)	*P* Value
Age	75.1 (7.3)	79.8 (6.5)	79.8 (6.4)	78 (7.2)	<.001
Male (no, %)	272 (56%)	39 (40%)	130 (52.2%)	77 (47.8%)	<.001
Total Medications	6.9 (4.3)	10.7 (4.4)	9.4 (4.4)	10.3 (4.3)	<.001
DRS R98 (#1-16)	21.9 (6.8)	13 (5.8)	22.2 (6.5)	7.5 (5.1)	<.001
DRS R98 (#1-13)	16.9 (6.2)	10.6 (5.1)	18 (6)	6 (4.2)	<.001
DRS R98Cog (#9-13)	8 (3.1)	7.3 (3.3)	9.7 (2.9)	4.2 (3.1)	<.001
DRS R98 Non-Cog (#1-8)	8.9 (4.2)	3.2 (2.8)	8.4 (4.3)	1.9 (2.2)	<.001

DRS-R98 = Delirium Rating Scale Revised 98, No = Number, Cog = Cognitive, Non-cog = non-cognitive, DSD = Delirium superimposed on dementia, *P* value <.05 for statistical significance.

Table 3. Mean and Standard Deviation of Individual DRS-R98 Items (Greater Scores Indicate Greater Intensity).

	Delirium I	Dementia II	DSD III	Controls IV	Sig Post Hoc tests
1. Sleep disturbance	1.9 (.9)	.7 (.7)	1.7 (.9)	.6 (.7)	I > II, IV; III > II; III > IV
2. Perceptual disturbance	1.1 (1.2)	.3 (.8)	.8 (1.2)	.2 (.5)	I > II, III, IV; III > II; IV
3. Delusions	.5 (.9)	.2 (.6)	.5 (.9)	.1 (.4)	I > II, IV; III > II; IV
4. Liability	.9 (.9)	.3 (.6)	1 (.9)	.2 (.9)	I > II, IV; III > II, IV
5. Language	1 (.9)	.3 (.6)	1 (1)	.1 (.5)	I > II, IV; III > II, IV
6. Thought process	1.3 (1.7)	.7 (.8)	1.4 (1)	.4 (.7)	I > II; II > IV; III > II, IV
7. Motor Agitation	1.4 (1)	.3 (.7)	1.1 (1)	.1 (.4)	I > II, III, IV; III > II; IV
8. Motor retardation	.8 (.9)	.4 (.6)	.9 (1)	.3 (.5)	I > II, IV; III > II, IV
9. Orientation	1.7 (.9)	.9 (.7)	1.5 (.8)	.2 (.6)	I > II > I V; III > II, IV
10. Attention	2.1 (.9)	1.6 (1)	2.2 (.8)	.7 (.9)	I > II > IV; III > II, IV
11. Short-term memory	1.7 (.9)	1.8 (1.1)	2.2 (.9)	1.3 (1)	I > IV; II > IV; III > I,II, IV
12. Long-term memory	1 (.9)	1.4 (1)	1.7 (1)	.7 (.9)	I, II > IV; III > I, IV
13. Visuospatial	1.4 (1)	1.8 (1.1)	2 (1.1)	1.1 (1)	I > II > IV; III > I, IV
14. Temporal onset	2 (1.1)	.6 (.8)	1.6 (.8)	.3 (.6)	I > II, III, IV; III > II, IV
15. Fluctuation	1.2 (.6)	.4 (.6)	.9 (.7)	.1 (.3)	I > II, III, IV; II > IV; III > II, IV
16. Physical disorder	1.8 (.5)	1.4 (.6)	1.7 (.5)	1 (.5)	I > II > IV; II > IV; III > II, IV

DSD = Delirium superimposed on Dementia, *P* < .01 indicates statistical significance for post hoc analysis.

greater proportion of the delirium superimposed on dementia group had antipsychotics prescribed but there was no difference in benzodiazepines between the groups. The mean number of etiologies was 2.4 (1.2), there was no significant difference in the mean number of etiologies between groups, with delirium having a mean 2.3 and DSD 2.4.

Delirium motor subtypes were documented in 707 patients (missing values = 27). Chi-squared test showed a significant association between delirium motor subtype and dementia status ($X^2 = 8.1$, df = 3, $P < .05$) and adjusted residual values showed that there is a significant association between DSD and hypoactive motor subtype (see table 5).

Discussion

This study aimed to compare delirium superimposed on dementia to delirium only in terms of phenomenology, etiology and motor subtype and to compare the phenomenology of the delirium superimposed on dementia group and delirium only groups to those with dementia only and cognitively intact controls.

Our findings demonstrate that delirium superimposed on dementia (DSD) differs from delirium without underlying dementia in terms of phenomenology, etiology and delirium motor subtype. In terms of non-cognitive symptoms, delirium occurring without an underlying dementia showed significantly greater levels of perceptual disturbance, motor agitation and fluctuation than those with DSD. It also showed higher scores in temporal onset, indicating that it was perceived to have come on more rapidly in this group. This is contrary to some previous research that has shown the opposite pattern or that there was no difference in non-cognitive neuropsychiatric symptoms between the groups.[10-13] However, a greater proportion of the DSD had at least one antipsychotic

Table 4. Etiology of delirium with and without dementia.

Total (%)	Delirium (n = 485)	DSD (n = 249)	X^2	*P* Value
1. Drug Intoxication	49 (10%)	26 (10%)	.3	.57
Adj, residual value	−.6	.6		
2. Drug withdrawal	28 (6%)	14 (6%)	.4	.84
Adj, residual value	−.2	.2		
3. Metabolic Disturbance	292 (60%)	97 (40%)	22.4	.00
Adj, residual value	4.7	−4.7		
4. Traumatic Brain injury	9 (2%)	9 (4%)	2.8	.09
Adj, residual value	−1.7	1.7		
5. Seizure	23 (5%)	18 (7%)	2.9	.09
Adj, residual value	−1.7	1.7		
6. Intracranial infection	9 (2%)	6 (2%)	.45	.5
Adj, residual value	−.7	.7		
7. Systemic infection	210 (43%)	142 (57%)	17.4	.00
Adj, residual value	−4.2	4.2		
8. Intracranial neoplasm	36 (7%)	5 (2%)	7.6	.00
Adj, residual value	2.8	−2.8		
9. Systemic neoplasm	112 (23%)	61 (24%)	1.3	.26
Adj, residual value	−1.1	1.1		
10. Cerebrovascular	59 (12%)	59 (24%)	20.4	.00
Adj, residual value	−4.5	4.5		
11. Organ insufficiency	160 (33%)	55 (22%)	5.6	.02
Adj, residual value	2.4	−2.4		
12. CNS other	34 (7%)	46 (18%)	27.9	.02
Adj, residual value	−5.3	5.3		
13. Other systemic	116 (24%)	50 (20%)	.35	.55
Adj, residual value	.6	−.6		
Antipsychotics prescribed	195 (40%)	193 (77%)	37.5	.00
Adj, residual value	−6.1	6.1		
Benzodiazepines prescribed	129 (26%)	57 (23%)	.15	.74
Adj, residual value	−.4	.4		

Adj. residual value = Adjusted residual value, DSD = Delirium superimposed on Dementia. $P < .05$ indicates statistical significance.

Table 5. A comparison of delirium motor subtypes in DSD and Delirium groups.

	No subtype (n = 124)	Hypoactive (n = 188)	Mixed (n = 156)	Hyperactive (n = 239)	Total
DSD	43	78	47	70	238
Adj residual	.3	2.7	−1.1	−1.8	
Delirium	81	110	109	169	469
Adj. residual	−.3	−2.7	1.1	1.8	

DSD = Delirium Superimposed on Dementia, Hypoactive = Hypoactive delirium, Mixed = Mixed Delirium, Hyperactive = Hyperactive Delirium.

medication prescribed at the time of our study and this may partially explain the fewer perceptual disturbances and agitation when compared to the delirium only group at the time of assessment. DSD was also significantly associated with hypoactive delirium subtype and therefore more subtle perceptual disturbance may have been more difficult to detect in this group.

In terms of cognition, both delirium groups (Delirium and DSD) did not differ in terms of attention but DSD scored significantly worse in short term memory, long term memory and visuospatial functioning than the delirium only group. Impairment in attention is a key diagnostic criteria[25] for delirium and therefore it is expected that this would be present to a high degree in both groups. Memory impairment is generally more associated with dementia and this may be reflected in the worse scores of the DSD group. Therefore, the pre-existing cognitive impairments in those with dementia would be expected to worsen the scores of those with DSD in comparison to delirium but the two groups did not differ in terms of the core cognitive

features of delirium such as attentional difficulties. This supports the view that delirium is not fundamentally different in terms of its cognitive profile when it occurs with and without an underlying dementia.

This study supports the view that delirium is most often multifactorial, with an average of 2.4 etiologies listed per delirium case. There were no significant differences in the number of contributory etiologies in patients with DSD vs those with delirium. Systemic infection and cerebrovascular etiologies were more common in those with DSD, pointing to the importance of a thorough investigation for an infective cause when a patient with dementia starts to exhibit signs of delirium. Cerebrovascular etiologies such as transcient ischaemic attacks may also be cause of delirium in this group and it is important that risk factors for these conditions are managed in order to reduce the likelihood of delirium.

In keeping with previous research,[10] we found that DSD was distinguishable from dementia only on a range of non-cognitive neuropsychiatric (e.g. sleep wake cycle disturbance, perceptual abnormalities) and cognitive items when assessed with a detailed and well validated scale (DRS-R98) administered by trained raters. Three related cognitive domains: attention, orientation and short-term memory were significantly worse in the DSD group, but there was no difference between groups in visuospatial ability and long-term memory. This shows that DSD should be readily detectable in those with dementia but there is a need to develop reliable rapid tests that require minimal time and training for use in this group. There is also a need for research into delirium in different types of dementia and at different stages of dementia as these are factors likely to present particular challenges in the diagnosis is DSD.

It is important to note that our study has several limitations. Although the DRS-R98 is rated on the patient's presentation over the previous 24 hours, a longitudinal study may be preferable to our cross-sectional design when assessing a fluctuating condition such as delirium. We were unable to include type of dementia, which is important as some types of dementia such as Lewy Body Dementia differ significantly to others such as Alzheimer's disease in terms of symptomatology. The Delirium Etiology Checklist has good face validity but has yet to be fully validated across populations and is only gives a broad overview of contributory etiologies (e.g. systemic infection, metabolic disturbance) and more detailed assessment may have been valuable in this regard. The inclusion of a palliative care group may also have added heterogeneity to the sample and can be viewed as a limitation to the generalisability of the current study.

Overall, this study found that DSD differed from delirium occurring without an underlying dementia in phenomenology, etiology and motor subtype. To the best of the authors knowledge, this is the largest study to compare these groups to date and took place across two countries including patients from palliative care and liaison psychiatry settings. We found that delirium superimposed on dementia was more likely to present with worse memory and visuospatial ability, be the hypoactive motor subtype and be prescribed antipsychotic medication than the delirium only group, who were more likely to show greater perceptual disturbances and be the hyperactive motor subtype. Delirium superimposed on dementia was also easily differentiated from dementia only by a wide range of symptoms including non-cognitive and cognitive neuropsychiatric items. Our study also supports the notion that delirium is multifactorial and describes a pattern of the most frequent etiologies between those with DSD and delirium. It is important to understand the differences in delirium when it occurs in the context of an underlying dementia, as it can be difficult to detect and treat in a clinical setting. We believe that this study contributes to our knowledge of this complex area. Further research may focus on detecting DSD in more challenging cases of advanced dementia and lewy body dementia, where a strong degree of overlap in symptomatology exists. This can have a beneficial effect in terms of identifying this condition and lessening the morbidity and mortality with which it associated.

Declaration of Conflicting Interests

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Ethical approval

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