Table 1 Comparison of demographic data, clinical features, comorbidities and treatment efficacy among reported case series

	US*	US†	France‡	Hong Kong§
No of cases	93	96	52	82
Mean age of onset (years)	60.9 (range 36 to 84)	52.4 (SD 16.9) (range 9 to 81)	–	62.1 (SD 12.9) (range 7 to 80)
Gender (male)	87%	87.5%	–	82%
Sleep-related injury	–	79.2%	–	81%
Injuries to self	32%	–	–	49%
Assaults on bed partner	64% (53/83)	–	–	57%
Dream enactment	93% (62/67)	87.5%	–	98%
Neurodegenerative diseases at baseline	53%¶	22.9%	75%¶	19.5%
Comorbid sleep disorder				
OSAS (AHI>10/h)	34%	–	–	61%
PLMD (PLMI>20/h)	47%	61%	–	33%
Narcolepsy	4%	14%	–	1%
Psychiatric illnesses (lifetime)	25.8%	9.4%	–	33%
Treatment efficacy of Clonazepam				
Completely or partially successful	87% (33/38)	90%	90%	87% (62/71)

In our study, completely successful cases of Clonazepam treatment included RBD patients without any further behavioural symptoms, while partially successful cases included the patients with residual shouting and/or talking, and minimal sleep movements.
*Olson *et al*.[1]
†Schenck *et al*.[2]
‡Sforza *et al*.[3]
§Present study.
¶The prevalence of neurodegenerative diseases for these two studies was based on cross-sectional data.
AHI, apnoea and hypopnoea index; OSAS, obstructive sleep apnoea syndrome; PLMD, periodic leg movement disorder; PLMI, periodic leg movement index.

treatment, as other drugs such as melatonin or pramipexole were not available in the local drug formulary. In our series, clonazepam was administered in 71 patients (86.6%) with a mean dosage of 0.65 (SD 0.47) mg initially and 1.4 (1.37) mg for the last prescription. The dosage of clonazepam was increased gradually in two-thirds (64%) of the patients. In contrast to Schenck and Mahowald's findings,[4] there was a significant increase between the starting dose and the last follow-up dose in our series (paired t test, $p<0.01$). Despite an increase in dosage, no obvious subjective tolerance and dependence were reported. Most patients responded well to clonazepam, and there was a significant reduction in SRI after treatment (pretreatment 80.8% vs posttreatment 5.6%; $p<0.05$). Only a minority reported adverse effects including intolerable daytime somnolence ($n = 5$), transient and reversible increase in liver enzyme ($n = 1$).

The similarities across all existing studies suggested an universal mechanism for the clinical and pathophysiological progression of RBD across different ethnicities studied to date. Male predominance, older population, a high percentage of SRI, aetiological association with neurodegenerative disorders, typical dreams content and excellent response to Clonazepam were comparable in all existing RBD series. In addition, the gender differences in our RBD patients were more related to the age of onset and diagnosis (similar to that of underlying neurodegenerative disorders like Parkinson disease), rather than the differences of clinical severity and presentation. Nonetheless, there was a relatively lower prevalence of neurodegenerative diseases in our cases. Emerging evidence suggested RBD as a preceding feature of neurodegenerative disorder for approximately 12–13 years.[1 5] However, the mean duration of follow-up in our series was slightly shorter (about 9 years after RBD onset). As a result, neurodegenerative disorders might not yet fully emerge in some of our RBD patients. Alternatively, there could be an underdiagnosis of subtle appearance or early stage of neurodegeneration in our RBD patients. Nonetheless, the prevalence rate of Parkinson disease and dementia in our RBD patients at baseline was about 13.4% and 10.9%, respectively, much higher than that of the local general population (0.5% and 4% respectively).

The comorbidity of psychiatric disorders was rarely addressed in RBD patients. We reported that one-third of our cases (33%) had lifetime histories of psychiatric disorders particularly depression, which was slightly higher than that of the local older population. Similar figures of lifetime histories of psychiatric disorders had been reported in Olson's study (25.8%), while Schenck's series revealed a slightly lower rate (9.4%). Although psychiatric disorders have rarely been implicated in the aetiology of RBD, certain psychotropics such as selective serotonin reuptake inhibitors might be a precipitating factor for the development of RBD.[5 6] Considering the prescription history of psychiatric medication in our series, there remained a possibility of psychotropic-related RBD in some cases. Further studies are required to delineate the relationship between typical and drug-related RBD.

In our study, patients' medical and psychiatric histories were based on the case-note review and the computerised record. Accordingly, systematic assessments of patients' physical condition and cognitive functioning were not fully obtained. Further systematic follow-up of our series was warranted to examine the progress of RBD development.

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CORRECTION

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P C A Vroomen, M Uyttenbogaart, G J Luijckv. *J Nerol Neurosurg Psychiatry*, 2006; **77**:799. ''Misleading conclusions on rt-PA treatment in the very elderly''. There is a spelling error in one of the authors names, G J Luijckv should be GJ Luijckx.