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Case report

Interrupting the trajectory of frailty in dementia with Lewy bodies with anabolic exercise, dietary intervention and deprescribing of hazardous medications

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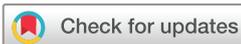
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SUMMARY

An 87-year-old man with dementia with Lewy bodies, living in residential aged care, exhibited rapid functional decline and weight loss associated with injurious falls over 9 months. Independent clinicians (geriatrician and exercise physiologist) assessed him during an extended wait-list period prior to his commencement of a pilot exercise trial. The highly significant role of treatable factors including polypharmacy, sarcopenia and malnutrition as contributors to frailty and rapid functional decline in this patient are described. The results of a targeted intervention of deprescribing, robust exercise and increased caloric intake on his physical and neuropsychological health status are presented. This case highlights the need to aggressively identify and robustly treat reversible contributors to frailty, irrespective of advanced age, progressive 'untreatable' neurodegenerative disease and rapidly deteriorating health in such individuals. Frailty is not a contraindication to robust exercise; it is, in fact, one of the most important reasons to prescribe it.

BACKGROUND

Frailty is a medical syndrome of increased dependency, vulnerability to stressors and excess mortality that is driven by diminished strength, endurance, neuropsychological and physiological functions.¹ The prevalence of frailty increases with age and chronic disease burden. However, there are also significant reversible components to this syndrome that are critical to diagnose and treat specifically. Morley *et al* describe the aetiology of frailty as the interaction among four central factors: sarcopenia, malnutrition, atherosclerosis and cognitive impairment.² Extrinsic factors such as decreased physical activity and immobilisation, insufficient dietary energy and protein intake, texture-modified diets and the iatrogenic effects of medications may add to pathophysiologic stressors including cachexia from chronic disease, cardiac and skeletal muscle dysfunction, sedation, apathy, depression, loneliness, delirium, psychosis, anorexia of ageing, dysphagia, poor dentition, impairment of vision, smell and taste. All of these stressors exert pressure on these four key components of frailty and

are modifiable to a varying degree.²⁻¹⁰ Additionally, malnutrition, cognitive impairment and atherosclerosis can all exacerbate sarcopenia by contributing to a negative energy balance, reduced drive to exercise and a significant reduction in physical capacity, respectively.¹¹

In the residential aged care environment, there is a confluence of these risk factors for frailty, resulting in a five-fold higher incidence than among community-dwelling older adults.¹²⁻¹³ Guidelines for aged care facilities traditionally focus on safety/falls reduction,¹⁴ which is the leading cause of accidental death in these facilities.¹⁵ Many recommendations for falls reduction, such as deprescribing high-risk medications and offering challenging balance exercises, may improve frailty as well.¹⁴ However, a safety focus can also lead to undesirable practices such as the use of restraints and immobilising chairs, which may reduce falls risk but actively exacerbate underlying frailty; thereby leaving the individual even more vulnerable to injurious falls and adverse outcomes.¹⁶ Recently released guidelines on frailty clearly recommend anabolic interventions such as progressive resistance exercise and increased protein/energy intake as first-line treatments to prevent and treat frailty.¹⁷ Evidence for the efficacy of this approach in frail populations has been available since the 1990s¹⁸ but has not yet become routine practice within residential aged care. The potential for remediating frailty is significant, especially for those living with dementia, who experience the highest levels of frailty in this setting.¹⁹ Notably, the aetiology of frailty in individuals with dementia is reported to have minimal correlation with the burden of disease pathology in the brain,²⁰ suggesting that the higher incidence of frailty cannot be attributed to normal disease course and may be related to factors more amenable to intervention.

The following case provides a rare, longitudinal insight into the aetiology and progression of frailty in a patient of advanced age with an aggressive neurodegenerative disease: dementia with Lewy bodies (DLB). We highlight the critical importance of differentiating disease progression from remediable causes of frailty, and the positive outcomes of a comprehensive intervention

of deprescribing, increased protein–energy intake and robust anabolic exercise.

CASE PRESENTATION

An 87-year-old man diagnosed with mild DLB 1 year prior by a geriatrician and living in a residential aged care facility since diagnosis was screened for a pilot exercise trial.²¹ He satisfied both the 2005 and 2017 criteria for the diagnosis of probable DLB including the onset of dementia prior to motor symptoms and the presence of two or more core features: fluctuating cognition and alertness, well-formed visual hallucinations and spontaneous parkinsonism features.^{22 23} He resided in a room by himself and his wife lived in the community, visiting daily. He had a recent history of recurrent falls and had lost 7% of his body weight in the 12 months since moving into the facility. Medical history included osteoporosis with hip fracture 5 years prior, chronic obstructive pulmonary disease (COPD), gout, dyslipidaemia, hypertension, macular degeneration, depression and a history of excessive alcohol consumption prior to admission. Medications included mirtazapine, aspirin, perindopril,

atorvastatin, tiotropium bromide, paracetamol and allopurinol. A texture-modified diet had been implemented due to concerns surrounding dysphagia and potential aspiration. The patient was observed to rapidly deteriorate in health status, precipitated by two injurious falls over 9 months while enrolled in a wait-list control period for the exercise trial. Evaluations were conducted to identify potential aetiologic factors in his rapid functional decline.

INVESTIGATIONS

The study geriatrician and exercise physiologist undertook external investigations during an extended, 9-month wait-list period due to the ill health of the patient prior to intervening. **Table 1** presents a timeline of relevant investigations and adverse events. A key limitation to investigations involved the lack of dietary assessment and nutritional biochemistry, as the study geriatrician was not the primary physician for the patient and therefore was not authorised to order these in his residential aged care setting. A complete biochemistry panel would have allowed for further evaluation of malnutrition, impaired cognition

Table 1 Investigations and patient timeline

Time point/event	November 2016, initial assessment	December 2016	Early February 2017, reassessme	February 2017	August 2017, reassessment
Review of systems	Patient reports constant hunger, feelings of isolation and dizziness on standing. Negative for pain, depression, Cardiovascular symptoms or dyspnoea		Patient still reports constant hunger, and feelings of isolation increased. Negative for dizziness, pain, depression, CVD Sx or dyspnoea		Patient reports hunger, increased isolation. Negative for pain, troubling hallucinations, depression, CVD Sx or dyspnoea.
Clinical course and physical examination	Weight loss >5 kg in 12 months and reduced muscle bulk diffusely. Kyphotic posture c/w Hx of hip fracture (2012). Ecchymosis widespread, acute ankle sprain. Cerebellar ataxia w. standing and walking c/w Hx excessive alcohol use.		Sepsis and seizure following fall. Further weight loss of 6.5 kg in 2 months, diffuse wasting. Ecchymosis still present. Decreased alertness c/w delirium, gait stability and strength/function.		Further weight loss of 11 kg in 5 months and severe, diffuse wasting. Healed rib fracture secondary to fall, immobilised with restraints. Decreased alertness. Ecchymosis still present. Unable to stand. Tardive dyskinesia, trunk dystonia to the right side, increased limb rigidity. Fetal posture in the supine position with hamstring contractures.
MMSE	21/30		17/30		13/30
SPPB	5/12		4/12		0/12
MNA-SF	10/14 (at-risk)		4/14 (Malnourished)		0/14 (malnourished)
Weight/BMI	74.5 kg/23 kgm ⁻²		68 kg/21 kg/m ²		57 kg / 17.7 kg/m ²
Orthostatic BP and HR	Symptomatic—dizziness		not symptomatic		
Lying (0 min)	120/50 mm Hg, 50 bpm		118/50 mm Hg, 56 bpm		Unable to perform due to immobility
Standing (1 min)	110/50 mm Hg, 62 bpm		126/60 mm Hg, 70 bpm		
Standing (3 min)	108/48 mm Hg, 70 bpm		128/460 mm Hg, 68 bpm		
BIA SMI	8.43 kgm ⁻²		7.8 kgm ⁻²		N/A
Grip strength (85 year+percentile. ²⁶)	L—25 kg (45th percentile) R—26 kg (40th percentile)		L—20 kg (25th percentile) R—17 kg (10th percentile)		L—17 kg (15th percentile) R—17 kg (10th percentile)
Pathology	TC: 4.7 mmol/L, LDL: 2.5 mmol/L, Vit. D: 58 nmol/L		No pathology results available for review by the research team		No pathology results available for review by the research team
Recommendations to facility doctor	Review need for aspirin, perindopril, atorvastatin, and mirtazapine. Recommend vitamin D—1000 IU/day. Flagged as high falls risk, malnourished and sarcopenic		Increase energy intake by an addition of high protein supplements and larger meal portions. Review needs for aspirin		Discussed in the Intervention section
Implemented recommendations	Removal of perindopril and atorvastatin. Vitamin D was not added, aspirin and mirtazapine not removed		Food portions not increased, high energy supplements prescribed (1080 kJ/day)		

Measured in kg/m², <9.5 kg/m² is considered Sarcopenic.²⁷

December 2016, adverse event: patient sustained minor elbow wound from injurious fall in facility. Infected wound led to sepsis, resulting in seizure and hospitalisation. Sodium valproate added to prescription. Patient restrained and catheterised and subsequently developed UTI and delirium.

February 2017, adverse event: patient experienced injurious fall within facility resulting in several fractured ribs and required hospitalisation. Physical restraints implemented in the reclining chair. Following medications added to prescription over 5-month period; risperidone, buprenorphine (patch), oxycodone, paracetamol. Supra-pubic catheter placed. Several UTIs reported. Completely immobile and highly sedated.

BIA SMI, bioelectrical impedance skeletal muscle index; BMI, body mass index; BP, blood pressure; HR, heart rate; LDL, low density lipoprotein; MMSE, mini-mental state exam; MNA-SF, Mini-nutritional Assessment Short Form; SPPB, short physical performance battery; Sx, symptoms; TC, total cholesterol; UTI, urinary tract infection.

and strength through indices such as serum albumin, electrolyte abnormalities, vitamin B₁₂ or iron deficiencies, thyroid hormone imbalances and diabetes. Physical assessment revealed the predominant DLB features in the patient, which were non-threatening hallucinations, orthostasis, drooling, mild rigidity in all limbs with a full range of motion, ataxia and postural instability. Notably absent were dystonia, dyskinesia, depression and dysphagia.

DIFFERENTIAL DIAGNOSIS

The aged care management plan for the patient appeared palliative and reactionary in approach to falls, pain management and psychosis, and suggested that the decline in function was viewed as an inevitable accompaniment to his DLB. However, the external research team elucidated many core, modifiable factors potentially contributing to the frailty through their investigations.

Natural disease course of DLB

DLB is an aggressive form of dementia with a lifespan postdiagnosis of 3–8 years.²⁴ The rate of cognitive decline in prospective studies is two to four points on the Mini-mental State Exam (MMSE) every year postdiagnosis.²⁴ The disease is characterised by acute fluctuations and a progressive decline in function over time, and is not relapsing–remitting in nature. The patient at baseline had mild cognitive impairment, was only mildly rigid with full range of motion and did not have depression, troubling hallucinations, dystonia or dyskinesia. Poorer prognostic outcomes are mainly precipitated in DLB by neuropsychiatric symptoms and hospitalisations from falls and bronchopneumonia,²⁵ similar to the general ageing cohort. However, this patient presented with a much more rapid cognitive deterioration

of eight points over 9 months. Despite no change to his non-threatening hallucinations, the antipsychotic risperidone was added to his medication regime, resulting in an acute worsening in extrapyramidal symptoms including tardive dyskinesia, trunk dystonia and rigidity. The rapid onset of these symptoms suggest iatrogenesis/delirium rather than the natural, degenerative disease progression.

Sarcopenia

The patient was sarcopenic at baseline, worsening at follow-up time points. Both isometric handgrip and appendicular strength were below age-matched thresholds at baseline²⁶ and deteriorated over the 9-month period in concert with declining physical activity levels. Body composition analysis confirmed a skeletal muscle mass (SMM) index below the sarcopenia definition of <9.5 kg/m²,²⁷ deteriorating from 8.43 to 7.8 kg/m² in the first 8 weeks of acute illness/sepsis, associated with a 6.5 kg overall weight loss. This equates to an average weekly loss of muscle mass of 250 g/week during that period, which is significantly higher than estimated in young, healthy bedrest models of muscle atrophy of 100–200 g/week,²⁸ and consistent with the literature reports of 25% of body weight loss being SMM.¹¹ This muscle loss likely continued (although not directly measured) with the further 11 kg of weight loss in the subsequent 5 months concomitant with restraint use, immobilisation and recurrent infections. Additionally, a significant decrease in gait speed, transfer and balance function accompanying muscle loss, progressing to non-ambulatory, chair/bed-bound status confirmed severe sarcopenia. Finally, the use of atorvastatin and vitamin D deficiency were also potential contributors to muscle weakness throughout the early stages of the decline.²⁹

Malnutrition

The patient was at risk of malnutrition at baseline according to the Mini-nutritional Assessment (MNA)³⁰ and developed severe malnutrition during the follow-up. Anorexia of ageing is a contributing factor to be considered, however, the patient always reported hunger, not loss of appetite. By contrast, there were multiple modifiable factors potentially driving insufficient energy intake.³¹ First, the prescription of anti-cholinergic and/or sedating medications (oxycodone, buprenorphine, risperidone, sodium valproate, mirtazapine, tiotropium) likely decreased the patient's opportunity to eat and contributed to the dysphagia known to be present in DLB³² as well as reduced saliva production, impairing mastication and swallowing along with the absence of lower teeth. A pureed diet was prescribed due to fear of aspiration. Texture-modified diets have been observed to significantly reduce the daily energy and protein intake in older adults¹⁰ as does eating in isolation with minimal social engagement.⁵ Additionally, there were potentially underlying cachexic processes from the recurrent infections, COPD diagnosis as well as statin and ACE-inhibitor prescriptions.⁷ Finally, the patient reported feeling hunger between meals and readily ate all food provided, which suggests the energy or protein content of food provided was not satiating or sufficient. Furthermore, no assessment of caloric intake or calculation of energy requirements for maintenance (approximately 30 kcal/kg/day) or weight increase (35 kcal/kg/day) or protein needs (1.2 g/kg/day) was conducted by the aged care facility despite severe and persistent weight loss.^{33–35}

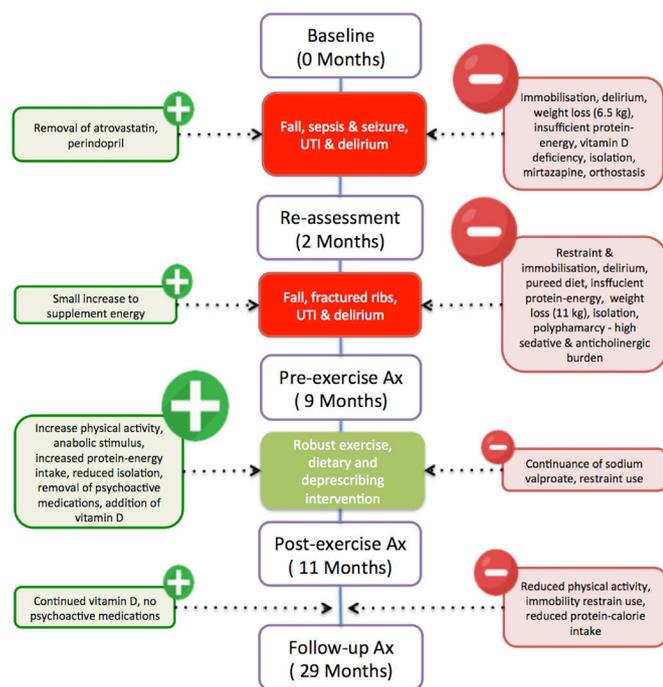


Figure 1 Contribution to frailty trajectory. Timeline illustrating the contributions of various negative and positive factors to the development of frailty and rehabilitation within the patient over the course of 29 months with inclusion of adverse events. The size of the plus/minus images indicates the relative magnitude of the effect on patient health at each stage. UTI, urinary tract infection.

Cognitive impairment and atherosclerotic processes

The patient was euipidaemic and normotensive, with no history or current symptoms of cerebrovascular or cardiovascular

Reminder of important clinical lesson

Medication review				
Medication	Indication	Action recommended	Timepoint withdrawn	Adverse effects from withdrawal
perindopril (8 mg Mane)	None	Discontinue	2 months	Nil reported
atorvastatin (10 mg Mane)	None	Discontinue	2 months	Nil reported
aspirin (100mg Mane)	None	Discontinue	9 months	Nil reported
oxycodone (2.5 mg QDS)	None	Discontinue	9 months	Nil reported
paracetamol (500 mg QDS)	None	Discontinue	9 months	Nil reported
mirtazapine (15 mg Nocte)	None	Discontinue	9 months	Nil reported
sodium valproate (200mg/5ml Mane)	None	Discontinue	-	-
risperidone (0.25 mg BD, 0.5 mg PRN BD)	None	Discontinue	9 months	Nil reported
buprenorphine (5 mg patch QW)	None	Discontinue	9 months	Nil reported
albuterol (unclear, Mane)	COPD	Use as required	-	-

Recommended treatments				
Medication	Indication	Action recommended	Timepoint implemented	Adverse effects
vitamin D supplement (1000 IU Mane)	Osteoporosis	Commence	9 months	Nil reported
Increase supplemental intake	Malnutrition	Increase by 5,040 kJ	9 months	Nil reported
Pureed diet	None	Re-evaluate need	9 months	Nil reported

Exercise Intervention	
Weeks 1-3	
Exercise rehabilitation performed within the residential facility involving weighted knee extension, chair leg press, triceps and biceps curls, shoulder raises, and seated rowing to promote shifting of centre of gravity. Additionally, standing and subsequently walking were trained until able to transfer into transport	
Weeks 4-8	
High intensity progressive resistance training was performed on pneumatic machines (Keiser Sports Fitness, Ltd., Fresno, Ca, USA) involving leg press, knee extension, triceps extension with sensory cueing. Further functional movements included walking, sit-to-stand and transferring. Performed for 1 hour sessions in the clinical gym for 3 days/week with dietary snack provided after each session.	



Figure 2 Intervention components.

disease, and no current alcohol or smoking (ex-smoker—discontinued 10 years prior) suggesting that atherosclerosis was not a recent contributing factor to progressive frailty or cognitive impairment. Apathy and depression are common predictors of malnutrition and cognitive performance in DLB,^{4 8 36} yet were absent in this patient at all assessment points. The high sedative burden of his medication regimen (five drugs with this side effect) was likely contributing to his cognitive impairment,^{6 37} especially considering the extra-pyramidal side effects observed (tardive dyskinesia, rigidity, trunk dystonia). His significantly reduced level of alertness and loss of postural control and communication ability witnessed just after administration of risperidone and sodium valproate was consistent with a drug-related delirium. The rapid eight-point decrease in his MMSE over a 9-month period, which is a similar decline to that reported in postoperative literature,^{9 38} was likely precipitated

by recurrent and persistent delirium from multiple episodes of infection and malnutrition, in addition to the central nervous system side effects of several medications noted above.

Summary

The final diagnosis we provided to the facility staff was an exacerbation of underlying frailty due to sarcopenia, malnutrition, polypharmacy and isolation/immobility, with all factors potentially treatable with targeted, robust interventions and changes in clinical care. All of these factors were inseparably involved in the decline (figure 1). For example, sedating drugs reduced the ability to eat, exacerbated sarcopenia and impaired strength and functional mobility, increasing fall risk further. Thus, without a comprehensive approach to all implicated factors, recovery would be sub-optimal or impossible.

TREATMENT

Deprescribing

Over the course of observation, deprescribing with rationale was recommended to the facility doctor at several time points (figure 2) to reduce iatrogenic influences of polypharmacy based on consensus criteria.³⁹ Primarily, most drugs were recommended for removal through lack of indication or being prescribed beyond the duration of treatment, which are stand-alone reasons to deprescribe. Additionally, specific contraindications included duplicate drug classes, ACE inhibitors with low/normal BP, anticholinergics in patients with dementia/delirium, long-acting with short-acting opioids for breakthrough pain, overall increased anticholinergic burden and inappropriate antipsychotics use for several categories including prescription for Parkinsonian disorders, behavioural symptoms in dementia and in patients with falls risk. There was also one indication for starting a prescription, namely adding vitamin D for known osteoporotic fractures of hip, spine and ribs and low lab value in an institutionalised patient.

Exercise and nutrition

The study exercise physiologist commenced intensive anabolic exercise, and the facility was instructed to increase energy intake from 1080 to 5040 kJ/day through liquid supplements, in addition to implementing a high energy, high-protein diet and

Initial contact November 2016	Reassessment (2 months) Early February 2017	Reassessment (9 months) August 2017	Following intervention (11 months) November 2017
<i>allopurinol</i> 300mg Mane	<i>allopurinol</i> 300mg Mane	<i>oxycodone</i> 2.5mg PRN up to QDS	<i>sodium valproate (liquid)</i> 200mg/5ml Mane
<i>mirtazapine</i> 15mg Nocte	<i>mirtazapine</i> 15mg Nocte	<i>mirtazapine</i> 15mg Nocte	<i>albuterol</i> dose unclear - Mane
<i>aspirin</i> 100mg Mane	<i>aspirin</i> 100mg Mane	<i>aspirin</i> 100mg Mane	<i>vitamin D</i> 3000 IU Mane (Loading dose for 3 months)
<i>perindopril</i> 8mg Mane	<i>oxycodone</i> 2.5mg PRN up to QDS	<i>sodium valproate (liquid)</i> 200mg/5ml Mane	
<i>tiotropium bromide</i> 18mcg Mane	<i>tiotropium bromide</i> 18mcg Mane	<i>risperidone</i> 0.25mg BD	
<i>thiamine</i> 100mg Mane	<i>thiamine</i> 100mg Mane	0.5 mg PRN up to BD	
<i>atorvastatin</i> 10mg Mane	<i>paracetamol</i> 500mg QDS	<i>buprenorphine (patch)</i> 5mg QW	
<i>paracetamol</i> 500mg PRN up to QDS		<i>albuterol</i> dose unclear - Mane	
		<i>paracetamol</i> 500mg QDS	

Figure 3 Patient medication prescription timeline.

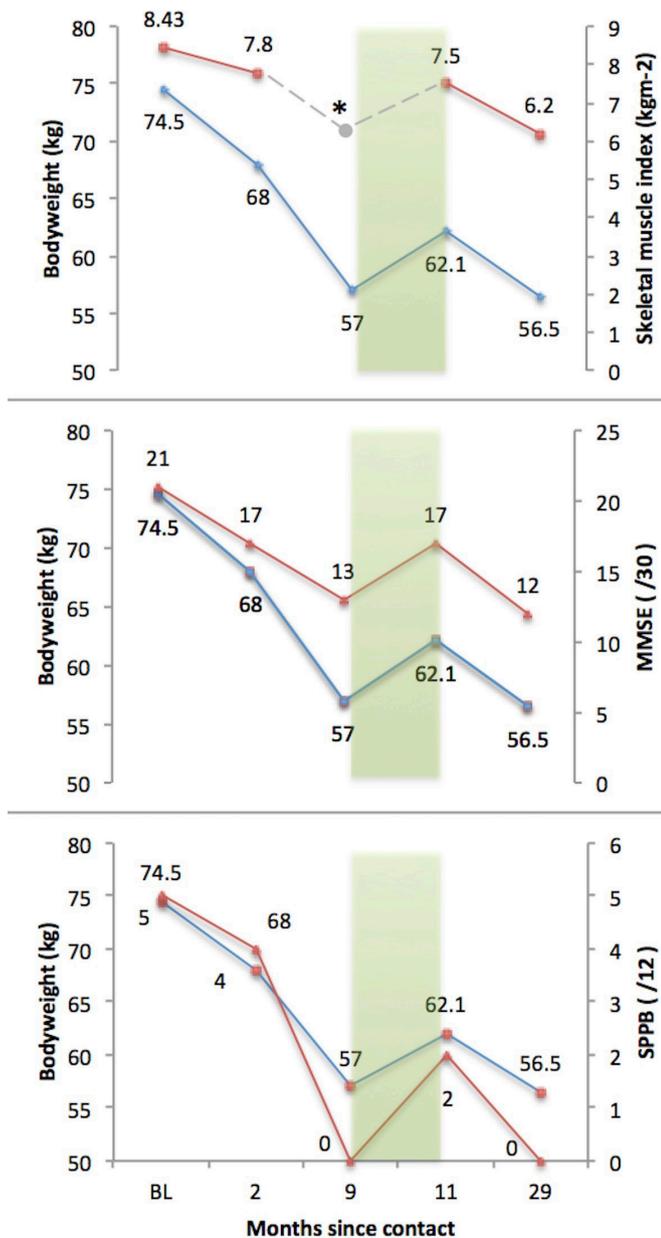


Figure 4 Frailty measures and outcomes from intervention. Graphs: relationship between body weight over time with (top) skeletal muscle index, (middle) cognition and (bottom) physical function. Intervention occurred between 9 and 11 months indicated in the green zone. Indicates no data collection, extrapolated from 29-month value of similar body weight. MMSE, mini-mental state examination; SPPB, short physical performance battery.

re-evaluating the need for a pureed diet as there was no evidence of aspiration. This was done in response to the severe and progressive undernutrition present, as evidenced by a weight loss of 17.5 kg, body mass index of 23 kg/m² decreasing to 17.7 kg/m², marked muscle wasting on physical exam, MNA score decreasing from 10/14 to 0/14, and complaints of hunger. Falls and infections are well-known co-morbidities associated with poor nutritional status in nursing home residents and were also present in this case. Protein and caloric supplementations significantly reduce mortality and complications as well as promoting weight change in undernourished patients.⁴⁰ Exercises were progressed in weeks 1–3 and administered in the facility to regain strength, standing and walking function to enable transport to the clinical



Figure 5 Rapid deconditioning and rehabilitation of patient. (A) Patient able to stand without hands at baseline assessment. (B) Weight loss of 8 kg and unable to stand without lifter after sepsis and delirium. (C) Further loss of 17.5 kg, immobile after rib fracture, deconditioning and sedation. (D) Weight gain of 5 kg, patient walking with contact guard after intensive rehabilitation for 8 weeks. BL, baseline.

gym for weeks 4–8. High-intensity progressive resistance training was performed on key muscle groups. All training throughout the 8-week period occurred as 3 days/week for 1-hour sessions and are described in figure 2.

OUTCOME AND FOLLOW-UP

The patient improved significantly following deprescribing and had no adverse consequences. A total of nine medications were assessed and recommended for immediate removal from the prescription across all time points. The prescription at each time point is detailed in figure 3. All medications except sodium valproate were ultimately removed, and high-energy supplements were prescribed (5040 kJ/day) in addition to his normal meal routine. The patient’s wife signed a waiver to allow the addition of some solid foods to his daily diet. The exercise intervention (figure 2) was tolerated well by the patient, and all 24 sessions were completed with no adverse events. Figure 4 displays key intervention results of the patient prior to, and following interventions. Figure 5 illustrates the rapid, clinically significant improvement in his overall function and health status, which included a transition from being chair-bound to ambulatory with contact guarding.

The patient was followed up 18 months after the study contact ceased, during which time no specific exercise was provided by the facility (2019). He had initially continued to walk with his wife’s support until he became too weak to stand with her assistance and was again wheelchair-bound. He was unable to stand even with assistance and had flexion contractures of both knees. He had lost all the weight that had been restored during the intervention period and weighed only 56.5 kg (17.5 kg/m², malnourished), with severe wasting of all skeletal muscles. Superficial lacerations were present on limbs. He was alert and conversational and had no extrapyramidal or psychotic symptoms, although his MMSE had declined to 12/30, which was a similar score to when he was last at this body weight prior to intervention. His only regular medications were paracetamol, vitamin D and a laxative solution.

Current care plan

Deprescribing has been successfully implemented, and although the presence of malnutrition was documented and dietary supplements prescribed by the facility, the continued weight loss suggests that actual energy and protein intake are far below needs. Actual documentation of his nutrient intake is needed via analysis of food/nutrients provided, portion consumed and

Reminder of important clinical lesson

intake relative to his energy and protein needs for weight gain/anabolism using standard metabolic equations.³³

The severe sarcopenia (low strength, SMM and functional mobility) he now manifests suggests that without robust anabolic exercise, this sarcopenia is insufficiently addressed by the deprescribing and nutritional supplementation. It should be noted that without anabolic exercise, nutritional supplements reduce habitual intake at meals, and thus do not actually augment total nutrient intake as anticipated.⁴¹ Thus, resistance training is the

critical missing component needed for treatment of both sarcopenia and malnutrition.

DISCUSSION

This is the first documented case of intensive progressive resistance training administered in a patient with DLB. The literature surrounding the effects of exercise is scarce and limited to several case reports,⁴² which evaluate aerobic, neuro-motor or functional training in clinically stable individuals. Additionally, this case provides a rare, longitudinal insight into the factors contributing to the rapid development of frailty in a patient with dementia in residential care and the key clinical assessments that guided effective rehabilitation.

However, the rapid decline reported in this scenario is likely to be a common but poorly documented occurrence in aged care facilities due to the high prevalence of risk factors. For instance, dementia is present in approximately 60% of all residential care patients.⁴³ One-third are sarcopenic,²⁰ one-half are currently, or at-risk of malnutrition³⁶ and over 60% are frail.⁴⁴ Additionally, almost one-half of all residents are prescribed at least one potentially inappropriate medication, of which neuroleptics are the most common.⁴⁵

This case highlights the concerning disparity between recommended optimal care for frailty and the reality of care for some patients in residential aged care facilities. The highest cause of accidental death in aged care facilities is falls, and as expected, the industry has evolved to prioritise safety and falls reduction. However, implementing immobilisation and restraints as a strategy to reduce falls further exacerbates the process of frailty, which is independently the most significant risk factor for falls.⁴⁶ Furthermore, the healthcare costs for frail patients far exceed the cost of non-frail patients, which places further strain on service delivery within residential aged care facilities.⁴⁷

Comprehensive geriatric assessment involving deprescribing, anabolic resistance training exercise and increased protein-energy intake are effective methods of mitigating the cycle of frailty.^{17 48 49} Importantly, withdrawal of anabolic interventions will likely precipitate a return to the previous level of functional decline. Extreme frailty is not a contraindication to comprehensive geriatric assessment and robust anabolic interventions. Conversely, it is one of the most important reasons to implement these treatment strategies.

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Patient's perspective

When my husband began to get worse, I felt like I was losing him. He was confused and upset, and his condition was bad. I would worry every time I would receive a call from the aged care facility, as I feared my husband might have injured himself once again. During this time, I felt like his quality of life was very poor. I couldn't talk normally with him and could not take him out for a coffee or a meal. I was very scared for his future, he had lost that much weight and was always sleeping, and the staff at the facility had told me to prepare for the worst.

A few days after his medications were removed, he was more awake and his humour and happiness came back. He began to know where he was, and it was beautiful to see him start doing the exercises to get him stronger and build muscle. Soon he was walking and we could get him into my car to drive him to the clinic, so he could do even more intense exercise. I even started training myself. My husband said that the exercise programme and walking again was a 'wonderful experience'. I kept walking him every day after the intervention finished, even though the staff was hesitant. We only stopped when he started to lose muscle again, which made it harder for me to stand him on his own. It would be great to see him able to do the robust resistance exercises again as he did in the study, as I think it is the only thing that can keep him strong enough to walk. The biggest challenge is finding the right facility and professionals that know how to train my husband like those in the study.

Learning points

- ▶ This case highlights the concerning disparity between recommended optimal care for frailty and the reality of care for some patients in residential aged care facilities
- ▶ The aetiology of frailty in dementia has many significant modifiable components that must be differentiated from the contributions of normal ageing and underlying disease.
- ▶ Anabolic interventions consisting of robust progressive resistance training, and increased energy and protein intakes are the most effective treatments for sarcopenia, malnutrition and frailty.
- ▶ Deprescribing in dementia when appropriate and observing patient condition offers an effective way of gauging iatrogenic contributions of polypharmacy to frailty, cognitive decline, sarcopenia and malnutrition.
- ▶ Highly clinically meaningful outcomes can be gained using resistance training in patients with advanced age, neurodegenerative disease and severe sarcopenia, including regaining of ambulatory status in previously immobilised, chair-restrained patients. However, withdrawal of anabolic exercise reverses these gains.

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