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Doctors in Training

Managing Bone Health in Hospital Inpatients With Parkinson's Disease: What You Need to Know

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Abstract

Parkinson's disease (PD) increases the overall risk of fracture approximately 1.5- to 2.5-fold, and the risk of hip fracture two- to three-fold, because an increased tendency to fall is superimposed on reduced bone strength. Hospital admission is a period of particular vulnerability: acute illness, immobility, delirium and disruption to the strict timing of dopaminergic medication all compound an already elevated fracture risk, while bone health is frequently overlooked in favour of the presenting complaint. This article outlines a practical approach for doctors in training managing inpatients with PD. It covers why fracture risk is so high, including the contribution of non-motor symptoms and of the admission itself; how to screen for falls risk and assess fracture risk on admission, including suggestions of how to adapt the Fracture Risk Assessment Tool (FRAX) for people with PD; why accurate and timely PD medication administration and careful medication review matter for bone health; and how swallowing, renal function and cognition should shape the choice of osteoporosis treatment. Attention to vitamin D and calcium status, multidisciplinary team involvement and safe discharge planning are highlighted as achievable, low-cost interventions that any trainee can institute during a routine ward round.

Keywords: Parkinson's disease; osteoporosis; falls; fracture prevention; inpatient care; fracture risk assessment

1. Introduction

Parkinson's disease (PD) is the second most common neurodegenerative disorder and the fastest-growing neurological condition worldwide [1,2]. Doctors in training will meet patients with PD on almost every general medical, surgical, orthopaedic and elderly care ward, usually admitted for a problem unrelated to their neurological diagnosis. PD increases the overall risk of fracture approximately 1.5- to 2.5-fold, and the risk of hip fracture two- to three-fold, compared with people without PD, with the greatest risk in those with more severe disease [1,3,4]. Hip fracture in an older, frail patient with PD carries a high risk of prolonged hospitalisation, loss of independence and death.

Despite this, bone health in PD is easily overlooked. It is rarely the presenting complaint, so it competes for attention with the acute problem that has brought the patient into hospital, and bone health assessment after a PD-related admission or fracture is inconsistently performed. A recent systematic review of clinical practice guidelines found that, although all recognised bone health as a concern in PD, recommendations on fracture risk screening were inconsistent and only one addressed whether osteoporosis medication is acceptable to, and tolerated by, people with PD [2]. Guidance on PD medication in hospital, meanwhile, focuses mainly on preventing motor deterioration rather than on its consequences for falls and fractures. The relevant advice is therefore scattered across falls, osteoporosis and PD guidelines.

This article brings that guidance together into a practical approach for the inpatient setting. It explains why fracture risk is so high in PD and how hospital admission adds to it; how to assess falls and fracture risk on admission; how to reduce falls risk during the stay; how to choose osteoporosis treatment when swallowing, renal function, or cognition are impaired; and how to plan a safe discharge. Approached in this way, an admission becomes a genuine opportunity to identify and address a modifiable risk before a fracture happens.

2. Why Does Parkinson's Disease Increase Fracture Risk?

Fracture risk in PD results from two interacting problems: an increased tendency to fall and reduced bone strength [1]. Falls are common in PD: more than two-thirds of patients fell during one 8-year cohort study [1]. Postural instability, shuffling gait and freezing episodes are the most well-known causes, and impaired gait and balance, together with a history of previous falls, are the most consistent predictors of future falls [5]. Motor fluctuations related to medication timing add a further, often under-recognised, hazard. As a dose of levodopa wears off, rigidity and bradykinesia return and the patient becomes suddenly and unpredictably less mobile and more prone to falling [6].

Non-motor symptoms can also contribute substantially to falls risk and are easily missed on a busy ward. Autonomic dysfunction causes orthostatic hypotension, urinary urgency and nocturia [7]. Orthostatic hypotension in

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PD is also associated with poorer global cognition and executive function, and the two problems often coexist in the same patient [7]. Cognitive impairment, which becomes more common as the disease progresses, can reduce awareness of hazards and make it harder for a patient to remember to use a walking aid or call for help [1]. Sleep disturbance, depression, anxiety, and psychosis also occur and can reduce daytime activity, often lead to the prescription of sedating medication, and predispose to delirium during acute illness [8].

Bone mineral density (BMD) is reduced in PD for several reasons. Reduced mobility and weight-bearing activity, sarcopenia, weight loss, and poor nutrition are common as the disease progresses, and reduced sunlight exposure contributes to vitamin D insufficiency [1]. It has also been suggested that levodopa may impair bone quality by raising serum homocysteine, but a recent systematic review concluded that, despite plausible mechanisms, evidence of a causal link between antiparkinsonian medication and low BMD is lacking [9]. Crucially, this is not a reason to reduce dopaminergic treatment. Low BMD only partly explains the excess fracture risk and PD remains an independent predictor of fracture after adjustment for femoral neck BMD, probably reflecting falls and muscle weakness [3]. Together, this means that the same fall that another older adult might walk away from is much more likely to result in a fracture in a patient with PD.

Hospital admission can independently worsen both sides of this equation (Fig. 1). On the falls side, acute illness, unfamiliar surroundings, delirium, and missed or delayed doses of dopaminergic medication all make a fall more likely. People with PD are particularly vulnerable to delirium: in one prospective cohort, delirium affected 67% of people with PD compared with 39% of older adults without PD, and was associated with higher mortality, new dementia and institutionalisation, yet hypoactive delirium is frequently missed [8]. On the bone side, immobility is a potent stimulus to bone loss: unloading the skeleton causes immediate loss of both trabecular and cortical bone, with increased bone resorption and reduced bone formation [10]. Hospitalised older adults spend a significant amount of time sitting or lying in bed, and around one in five admissions is followed by a new loss of independence in activities of daily living, with length of stay the strongest predictor [11]. Missed doses of PD medication, which have been estimated to prolong admission by an average of 4 days [6], therefore add to immobility and deconditioning. In this way, an admission for an unrelated problem can drive a vicious cycle of increasing falls, reduced BMD, further admissions and repeated fractures (Fig. 1).

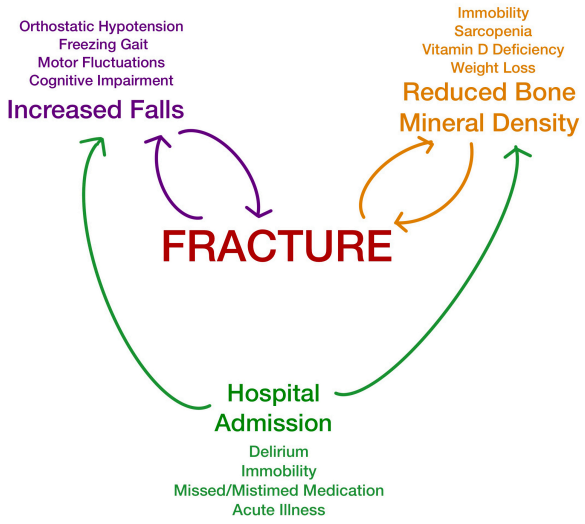

Fig. 1. The vicious cycle linking falls, reduced bone mineral density and fracture in hospital inpatients with Parkinson's disease. Hospital admission can independently worsen both falls risk and bone fragility, while a fracture itself further increases immobility, falls and bone loss, perpetuating the cycle. Drawn using Procreate (Version 5.4.14; Savage Interactive Pty Ltd., Hobart, Tasmania, Australia).

3. Assessing Bone Health and Falls Risk on Admission

Every patient with PD admitted to hospital should be assumed to be at high risk of falling, regardless of age. The National Institute for Health and Care Excellence (NICE) recommends a multifactorial falls risk assessment for older people and for people aged 50 and over at higher risk of falling, and people in hospital do not need to meet any further criteria to receive it [12]. This should cover falls history, cognition, continence, vision, footwear, mobility, lying and standing blood pressure, and medication.

A basic bone health history is quick to take and easy to miss: ask about previous fragility fractures, height loss, back pain suggestive of vertebral fracture, dietary calcium intake and time spent outdoors. Where it has not already been done, fracture risk should be estimated using the Fracture Risk Assessment Tool (FRAX), which calculates the 10-year probability of hip and major osteoporotic fracture from clinical risk factors, with or without femoral neck BMD [13]. PD is not itself a FRAX input. The 2024 UK National Osteoporosis Guideline Group (NOGG) guideline highlights PD as a condition that increases hip and non-vertebral fracture risk independently of BMD, so standard FRAX scores underestimate true risk [14]. NOGG therefore recommends entering 'yes' for rheumatoid arthritis as a proxy for PD, while acknowledging that this only partly accounts for the excess risk, and increasing the calculated probabilities by 30% in people who have had two or more falls in the previous year [14].

PD-specific approaches build on this guidance. The BONE PARK algorithm, first published in 2015 and since revised, has been updated as BONE PARK 2, which was developed from a cross-sectional study of people with PD and related parkinsonism [15]. It combines the NOGG ad-

justments described above (rheumatoid arthritis as a proxy for PD, the falls adjustment and, where available, measured femoral neck BMD) with the age-specific NOGG intervention thresholds. It advises treatment when dual-energy X-ray absorptiometry (DXA) is not feasible, and the probability calculated without BMD exceeds the intervention threshold, and specialist input for people at very high risk [15]. BONE PARK 2 is intended to make fracture risk assessment in PD more practical and more accurate, but it has not yet been validated against fracture outcomes, so there is not yet evidence that it predicts fracture better than FRAX [15]. It is best regarded as a structured, guideline-based framework rather than a validated risk score. A simplified practical approach, based on NOGG guidance and BONE PARK 2, is summarised in Box 1 (Ref. [14,15]).

Box 1. A simplified approach to fracture risk assessment in inpatients with Parkinson's disease (PD), based on National Osteoporosis Guideline Group (NOGG) guidance and the BONE PARK 2 algorithm [14,15].

1. Previous fragility fracture? If yes (especially hip or vertebral), consider osteoporosis treatment. Seek specialist advice if very high risk, for example, a recent vertebral fracture or two or more vertebral fractures.

2. Calculate the Fracture Risk Assessment Tool (FRAX) score. Enter 'yes' for rheumatoid arthritis as a proxy for PD. Add femoral neck bone mineral density (BMD) if a recent dual-energy X-ray absorptiometry (DXA) result is available.

3. Adjust for falls. If there have been two or more falls in the previous year, increase the major osteoporotic and hip fracture probabilities by 30%.

4. Compare with the age-specific NOGG intervention threshold. Low risk: optimise calcium, vitamin D and falls prevention. Intermediate risk: arrange DXA, or, if DXA is not feasible, offer treatment if the probability is above the intervention threshold. High risk: offer treatment. Very high risk: seek specialist advice.

5. Apply clinical judgement. Consider treatment at a lower threshold in frequent fallers and in patients who are frail, have advanced disease or cognitive impairment, or lie close to a threshold.

6. In every patient, check calcium, 25-hydroxyvitamin D and renal function, address falls risk, and document the bone protection plan in the discharge summary.

Whichever approach is used, the result should be interpreted in the light of disease-specific features that FRAX does not capture. The falls adjustment applies a fixed 30% increase, which is likely to oversimplify the risk in very frequent fallers [15]. Marked postural instability or freezing, more advanced disease, cognitive impairment, sarcopenia, muscle weakness, and recent immobility all suggest that the true risk is higher than the calculated figure [4,15]. These features should lower the threshold for treatment. DXA should be arranged where it would change management. It may be impractical in patients with severe tremor, dyskinesia, rigidity, or contractures, and should not delay treatment for those who already qualify for it. When the hip and spine cannot be measured reliably, a forearm measurement can be used instead [14]. In practice, bone health screening after a PD-related admission or fracture is inconsistently performed, and doctors in training are well placed to make sure it is not forgotten before discharge.

Checking serum calcium, phosphate, 25-hydroxyvitamin D, and renal function as part of admission blood tests is a simple, low-cost step. It can identify correctable contributors to bone fragility in many patients with PD, and the results will be needed when choosing osteoporosis treatment [14].

4. Reducing Falls Risk During Admission

Falls prevention in hospital is a team effort, but there is plenty a doctor in training can influence directly:

- Review the drug chart for anything that could worsen parkinsonism or mobility. Dopamine-antagonist antiemetics and antipsychotics such as metoclopramide, prochlorperazine and haloperidol should be avoided in PD, as they can precipitate a marked and dangerous deterioration in mobility; domperidone or ondansetron can be considered as alternatives for nausea, bearing in mind the risk of QT prolongation with ondansetron usage. Haloperidol should be avoided even in delirium; if an antipsychotic is unavoidable, quetiapine is usually preferred, ideally with specialist advice [8].
- Undertake a structured medication review. People with PD are often prescribed medicines for insomnia, depression, anxiety, psychosis, pain and bladder symptoms. Benzodiazepines, Z-drugs, sedating antidepressants and antipsychotics, opioids and drugs with anticholinergic effects can cause sedation, confusion, orthostatic hypotension and impaired balance, and should be reduced or stopped where possible, with the ward pharmacist and, for PD-related prescribing, the PD team [8,12]. Melatonin is generally preferred if a hypnotic is needed [8].
- Measure lying and standing blood pressure, and review antihypertensives and diuretics that may worsen orthostatic hypotension, particularly if the patient reports dizziness on standing [12].
- Screen for delirium on admission and regularly thereafter, and use non-pharmacological prevention: reorientation, glasses and hearing aids, hydration, bowel care, sleep hygiene and early mobilisation [8].
- Refer early to physiotherapy and occupational therapy to maintain mobility rather than default to bed rest, and en-

sure walking aids and appropriate footwear are available at the bedside.
- Anticipate toileting needs, since urgency and nocturia are common in PD and unassisted trips to the bathroom are a frequent cause of inpatient falls.

Perhaps the single most important, and frequently mismanaged, aspect of inpatient PD care is medication timing. NICE recommends that adults with PD in hospital take levodopa within 30 minutes of their individually prescribed administration time [16], and antiparkinsonian medication should not be withdrawn abruptly, because of the risk of acute akinesia and neuroleptic malignant syndrome [17]. Omitted and delayed doses of critical medicines, including PD medication, were identified as a patient safety priority by the National Patient Safety Agency (NPSA) [18], yet they remain common, particularly outside normal drug round times, in the emergency department or when a patient is nil by mouth for investigations. In the 2022 UK Parkinson's audit, 58% of people with PD admitted to hospitals in England did not receive their medication on time every time [6], and a 2025 national emergency department audit found that only 38% of levodopa doses were given within 30 minutes of the expected time [19]. From a bone health perspective, mistimed medication matters directly. Missed or late dose is a period of worse rigidity and bradykinesia, and therefore a period of higher falls risk.

Encouraging self-administration where the patient is able (in the same audit, 78% of self-administered doses were given on time [19]), prescribing PD medication as time-critical, seeking early specialist advice on alternative routes (such as a nasogastric tube or transdermal patch) when the patient cannot swallow, and involving the ward pharmacist or PD nurse specialist early are all simple, high-yield interventions. These are also natural targets for quality improvement: multifaceted, multiprofessional projects to reduce delays in time-critical PD medication have been described [20], and an audit of medication timing, or of bone health assessment in inpatients with PD, is an achievable project for a doctor in training.

5. Choosing Osteoporosis Treatment in the Inpatient With Parkinson's Disease

Where osteoporosis treatment is indicated, because DXA confirms osteoporosis, because the FRAX-based risk exceeds the intervention threshold, or because a fragility fracture has already occurred, the choice of agent needs to take account of practical issues that are common in PD [14,15]. Swallowing difficulties affect more than one-third of people with PD and become more frequent as the disease progresses [21]. Oral bisphosphonates must be swallowed whole on an empty stomach, and the patient must stay upright for 30–60 minutes after the dose. Both requirements can be unrealistic in an unwell inpatient with dysphagia, significant motor fluctuations, or cognitive impairment, and adherence to oral osteoporosis treatment is often poor in older people taking several other medications [22]. In this situation, intravenous zoledronic acid, given once yearly, or subcutaneous denosumab, given six-monthly, avoid the need for tablet swallowing and prolonged positioning, and are recommended alternatives in patients who cannot reliably take or tolerate oral bisphosphonates [14], as summarised in Table 1 (Ref. [14]).

Other patient factors also shape the choice. Renal function determines which agents can be used: oral bisphosphonates are not recommended when creatinine clearance is below 30–35 mL/min, and zoledronic acid should not be given below 35 mL/min, whereas denosumab can be used in renal impairment but carries a higher risk of hypocalcaemia in advanced chronic kidney disease [14]. Patients with a recent vertebral fracture, two or more vertebral fractures or very low BMD may be at very high risk and should be considered for specialist referral, because anabolic agents such as teriparatide and romosozumab reduce fracture risk more rapidly and to a greater extent than some antiresorptive drugs [14]. Poor nutritional status should prompt dietetic review. Finally, likely long-term adherence and the patient's own priorities, discussed with them and, where appropriate, with family or carers, should inform the decision.

Denosumab has a discharge-planning implication that is easy to miss. Unlike bisphosphonates, its antiresorptive effect wears off quickly, and delaying or stopping treatment has been associated with a rebound increase in bone turnover and multiple vertebral fractures [23]. Any patient started on denosumab in hospital therefore needs a clear, documented plan for the next dose to be given on time in the community, ideally coordinated through the general practitioner (GP) or a fracture liaison service. There should also be an agreed exit strategy: if denosumab is to be stopped, for example because of declining health or difficulty attending appointments, it should be followed by a bisphosphonate, usually zoledronic acid, to reduce rebound bone loss [14,23]. For this reason, denosumab may be less suitable for patients in whom reliable long-term follow-up cannot be assured.

Calcium and vitamin D status should be corrected before, or alongside, starting any osteoporosis treatment, since antiresorptive therapy is most effective, and safest, once vitamin D deficiency has been addressed [14].

6. Multidisciplinary Care and Safe Discharge

Bone health in PD is a multidisciplinary problem, and hospital admission is often the first time it receives dedicated attention. Involve physiotherapy and occupational therapy for ongoing falls and mobility assessment, speech and language therapy where swallowing is impaired, the PD nurse specialist and ward pharmacist for medication optimisation, and a falls clinic or fracture liaison service for outpatient follow-up if one is not already involved.

Cognitive impairment, delirium, depression and sleep disturbance can interfere with rehabilitation, treatment ad-

Table 1. Summary of osteoporosis treatment options for inpatients with Parkinson's disease (PD) [14].

Agent	Route and frequency	Key advantage in PD	Practical caveat
Oral bisphosphonate (e.g., alendronate, risedronate)	Tablet, usually weekly. Sit upright for 30–60 min after the dose	Familiar, low cost, first line in most guidelines	Needs an intact swallow and the ability to sit upright, which is often impractical with dysphagia or marked motor fluctuations. Not recommended if creatinine clearance is below 30–35 mL/min
Zoledronic acid	Intravenous infusion, once yearly	Avoids swallowing and positioning requirements entirely; a single annual dose aids adherence	Requires creatinine clearance of at least 35 mL/min, adequate hydration and correction of vitamin D and calcium first. Can cause an acute-phase reaction after the first infusion
Denosumab	Subcutaneous injection, six-monthly	No swallowing needed, no renal dose adjustment, but severe hypocalcaemia is a significant concern in advanced chronic kidney disease	Rebound vertebral fracture risk if a dose is delayed or treatment stops without follow-on bisphosphonate, so needs a robust discharge follow-up plan. Risk of hypocalcaemia in advanced chronic kidney disease
Calcium and vitamin D (adjunct)	Daily oral supplementation	Corrects a common, reversible contributor to bone fragility in PD	Should be established before or alongside any antiresorptive agent, not used alone if osteoporosis is confirmed

herence and discharge planning, and may benefit from input from geriatric medicine or old age psychiatry where available [8]. In patients with cognitive impairment, obtain a collateral history from family or carers, screen for and treat delirium, and assess capacity for specific decisions such as starting long-term osteoporosis treatment, involving those close to the patient in best-interests decisions where capacity is lacking. Swallowing difficulties and cognitive impairment can both make oral osteoporosis treatment impractical [15], so a once-yearly infusion or a carer-supervised regimen may be more achievable than a weekly tablet for a patient with memory problems.

Before discharge, check that the PD medication regimen is unchanged from the patient's usual regimen (or that any change has been agreed with a PD specialist), that any new osteoporosis treatment and its follow-up dosing are clearly documented in the discharge summary, and that outstanding investigations such as DXA or vitamin D level are followed up in primary care. These are small, achievable tasks for a doctor in training, and they are often the difference between an admission that reduces a patient's future fracture risk and one that leaves it unaddressed until the next fall.

7. Where the Evidence is Limited

Much of the advice in this article is drawn from national guidelines, whose recommendations are largely based on studies in the general older population. Evidence specific to PD is more limited, and several recommendations rely on observational data, expert consensus or extrapolation. In particular, no randomised controlled trial has yet shown that osteoporosis treatment reduces fractures in people with parkinsonism; a large, home-based, placebo-controlled trial of zoledronic acid has been designed to answer this question [22]. People with advanced PD, severe cognitive impairment, dysphagia or frailty are underrepresented in osteoporosis trials, so the optimal treatment for these groups is uncertain, and decisions rely on individual judgement about benefit, treatment burden and life expectancy. PD-specific approaches such as BONE PARK 2 have not yet been validated against fracture outcomes [15], the 30-minute window for levodopa administration is based on expert consensus [16], and proposed mechanisms linking PD medication to bone loss remain unproven [9]. The recommendations here should therefore be applied as a pragmatic framework, distinguishing guideline-based advice from advice based on indirect evidence, and tailored to the individual patient and their goals of care.

8. Conclusion

People with PD admitted to hospital are at high risk of fracture because falls and reduced bone strength combine, and the admission itself can make both worse. Resident doctors are well placed to break this cycle with a few simple steps: treat every inpatient with PD as being at high risk of falling; give PD medication on time and review medicines that increase falls risk; assess fracture risk using FRAX adapted for PD; check and correct vitamin D and calcium; choose an osteoporosis treatment that the patient can realistically take; and make sure the plan, including follow-up of any injectable treatment, is clearly handed over at discharge. Although PD-specific evidence remains limited, many of these measures are supported by existing clinical guidelines or expert consensus and can be incorporated into routine inpatient care.

Key Points

• PD increases overall fracture risk approximately 1.5- to 2.5-fold and hip fracture risk two- to three-fold, through both falls and reduced bone strength; hospital admission can worsen both.

• All inpatients with PD should be treated as being at high risk of falling, irrespective of age, and given a multifactorial falls risk assessment that includes medication review, delirium screening and lying and standing blood pressure.

• Timely, accurate administration of dopaminergic medication (within 30 minutes of the individually prescribed administration time) is a patient safety priority that also reduces falls risk.

• FRAX underestimates fracture risk in PD: enter rheumatoid arthritis as a proxy, adjust for recurrent falls, and interpret the result in the light of disease severity, cognition, and falls frequency.

• Swallowing, renal function, and cognition should guide the choice of osteoporosis treatment; intravenous zoledronic acid or subcutaneous denosumab are alternatives when oral bisphosphonates are impractical.

• Vitamin D and calcium status should be checked and corrected, and discharge planning should ensure continuity of both PD and bone health medication, with a clear plan for repeat dosing, or a follow-on treatment, for any injectable osteoporosis therapy.

Curriculum Checklist

This article addresses the following Capabilities in Practice of the 2022 general internal medicine curriculum:

• Providing continuity of care to medical in-patients, including management of comorbidities and cognitive impairment.

• Is focused on patient safety and delivers effective quality improvement in patient care.

• Managing a multi-disciplinary team including effective discharge planning.

Availability of Data and Materials

Not applicable.

Author Contributions

BS and JH contributed to the conception and drafting of the manuscript. Both authors contributed to revising the manuscript critically for important intellectual content. Both authors have read and approved the final manuscript and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

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Conflicts of Interest

The authors declare no conflicts of interest.

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