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BACK PAIN IN PARKINSONISM: EXPLORING PAIN GENERATORS AND INTERVENTIONAL MANAGEMENT: A CASE SERIES

Ismi Adhanisa Hamdani [1,3,5,6,9], Lie Anto [2,4,5,6,9], Irma Savitri[5,6,9], Tiara Aninditha[5,6,9], Nurul Fadli[5,7,9], Yusak Mangara Tua Siahaan[8], Dyah Tunjungsari[6,9], Henry Riyanto Sofyan[5,6,9]

[1]Department of Neurology, Rumah Sakit Umum Daerah KH Idham Chalid, Ciawi, Bogor, Indonesia
[2]Department of Neurology, Rumah Sakit Umum Daerah Kabupaten Bintan, Riau, Indonesia
[3]Department of Neurology, Universitas Tarumanagara, Jakarta, Indonesia
[4]Department of Neurology, Universitas Maritim Raja Ali Haji, Tanjungpinang, Riau, Indonesia
[5]Fellowship MIN KNI, Rumah Sakit Umum Pusat Nasional Cipto Mangunkusumo, Jakarta, Indonesia
[6]Department of Neurology, Rumah Sakit Umum Pusat Nasional Cipto Mangunkusumo, Jakarta, Indonesia
[7]Department of Neurology, Rumah Sakit Universitas Indonesia, Depok, Indonesia
[8]Department of Neurology, Siloam Hospitals Lippo Village, Karawaci, Tangerang, Indonesia
[9]Department of Neurology, Universitas Indonesia, Jakarta, Indonesia

ARTICLE INFO

Corresponding author :
Ismi Adhanisa Hamdani
Department of Neurology, Rumah Sakit Umum Daerah Idham Chalid, Ciawi, Bogor, Indonesia

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ismi_fkui@yahoo.com

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ABSTRAK

Nyeri merupakan gejala non-motorik umum pada parkinsonisme, sering terabaikan meski berdampak klinis signifikan. Bentuk tersering adalah Penyakit Parkinson (PP), diikuti parkinsonisme sekunder dan atipikal seperti PSP. Dua kasus menunjukkan nyeri muskuloskeletal-spinal sebagai keluhan utama: pasien PP dengan nyeri punggung atas yang membaik setelah blok saraf erector spinae, serta pasien PSP dengan nyeri faset lumbal yang berkurang setelah injeksi steroid intraartikular. Nyeri pada parkinsonisme dapat bersumber dari otot, tulang, sendi, diskus, ligamen, fasia, maupun saraf, sehingga memerlukan evaluasi cermat. Kedua kasus menunjukkan efektivitas intervensi injeksi nyeri pada pasien yang tidak responsif terhadap terapi konservatif. Penilaian dan manajemen nyeri yang tepat sangat penting untuk meningkatkan kualitas hidup pasien parkinsonisme.

ABSTRACT

Pain is a common non-motor symptom in parkinsonism, often overlooked despite its significant clinical impact. The most common form is Parkinson's disease (PD), followed by secondary and atypical parkinsonisms such as PSP. Two cases presented with musculoskeletal-spinal pain as the chief complaint: a PD patient with upper back pain that improved after an erector spinae nerve block, and a PSP patient with lumbar facet pain that improved after an intra-articular steroid injection. Pain in parkinsonism can originate from muscles, bones, joints, discs, ligaments, fascia, or nerves, requiring careful evaluation. Both cases demonstrate the effectiveness of pain injection interventions in patients unresponsive to conservative therapy. Appropriate pain assessment and management are crucial to improving the quality of life of parkinsonism patients.

INTRODUCTION

Pain affected people with parkinsonism more frequently and with greater severity than age-matched controls.[1,2] Parkinsonism is a syndrome characterized by cardinal motor deficits—bradykinesia, rigidity, tremor, and unstable posture. Idiopathic PD is the most common etiology of parkinsonism, accounting for roughly 80% of these cases.[3]

Other spectrums of parkinsonism include atypical parkinsonism or Parkinson plus (such as PSP, multiple system atrophy, corticobasal degeneration, Lewy Body dementia) and secondary parkinsonism due to drugs, vascular, toxin, or brain infection. More than two-thirds of individuals with PD experience chronic or recurring pain, with prevalence ranges from 24% to more than 85% (mean 67.6%).[1,2,4-9] Pain in PD is heterogeneous and frequently intensified by rigidity, bradykinesia, and motor fluctuations.[4,6,10-12]

Chronic pain in PD is strongly linked with depression, sleep disorder and reduced quality of life.[4,8,11-20] Management should begin with optimization of anti-parkinsonian therapy and Deep Brain Stimulation (DBS) in few selected cases, the use of analgesics and adjuvants according to the pain phenotype, physical therapy, and when conservative measures are insufficient, consideration of targeted interventional procedures such as diagnostic or therapeutic blocks and botulinum toxin for dystonic pain.[4-6,10,13,19,20] Although expert consensus supports comprehensive multimodal approaches, high-quality controlled trials of specific pain therapies in parkinsonism remain limited.[5,6,19] This retrospective case series describes two patients in neurology pain clinic at Cipto Mangunkusumo Hospital Jakarta, with parkinsonism and spinal pain who responded to interventional pain procedure after inadequate response to conservative therapy. Informed consent was obtained from all participants included in the study.

CASE SERIES

Case 1: A 69-year-old male with clinically probable advanced PD (3-year duration), Hoehn & Yahr stage 4, total Unified Parkinson's Disease Rating Scale (UPDRS) score of 57 with no on/off phase (moderate-severe disability), controlled hypertension and DM, presented with severe upper-back pain since two weeks ago. He began developing right-sided resting tremor three years ago, followed by slowness over the next two years and increasing gait difficulty, stiffness, frequent crying during the past year. He currently uses a wheelchair for mobility.

He was referred to our hospital, and due to progressiveness of PD symptom, we escalate and change treatment from trihexyphenidyl 2 mg 2 times daily and levodopa 100 mg/benserazide 25 mg 1 tablet twice daily to Levodopa 100 mg/Carbidopa 25 mg/Entacapone 200 mg (LCE) 1 tablet three times daily and pramipexole 0,375 mg once daily. Pain assessment revealed acute onset (<3 months), upper back pain with predominant involvement of the left paraspinal region and did not radiate. The pain characterized as nociceptive and mechanical, accompanied by stiffness, without burning sensation, paresthesia, or electric shock–like features. The pain was persistent and aggravated with movement and axial activity.

Pain is severe with NRS 8/10 on movement. Pain is PD-unrelated because it was not started or became more severe after the initiations of PD symptoms, the pain did not worsen in relation to rigidity, bradykinesia, tremors, there was no chorea or dyskinesia, and the pain did not improve or show temporal relationship with the administration of antiparkinsonian therapy. Physical examination revealed a shuffling gait, rigidity, right hand resting tremor, and postural instability. Intention and postural tremors were also clearly observed. There was marked left-sided thoracic paraspinal rigidity, and he did not fulfill diagnostic criteria for sacroiliac joint and facet joint syndrome. Lumbar spine radiographs demonstrated disc degeneration at L2–L3 and bilateral facet joint degeneration at L5–S1, with parasyndesmophytes along the anterior lumbar vertebral bodies, raising suspicion for axial spondyloarthropathy. Brain MRI showed generalized cerebral atrophy with bilateral periventricular white-matter changes consistent with chronic small-vessel disease (Fazekas grade 2), loss of mesencephalic “swallow-tail” sign of nigrosome-1—an appearance seen

in PD—and suspected agenesis of left A1 segment of the anterior cerebral artery, with left ACA supplied from the right internal carotid artery via the anterior communicating artery.

Initial pain management with celecoxib 2x200 mg led to no improvement. Considering the musculoskeletal predominance and the contributory role of rigidity and axial motor features, a regional interfascial block called Erector Spinae Plane (ESP) Block was selected. We did ultrasonography (USG) guided injection of 10 ml mixture of lidocaine 2% 4 cc, dexamethasone 5 mg/1cc and Dextrose5% into interfascial or space underneath a group of back muscles of Erector Spinae that spread in this plane and numbs multiple nearby nerves. The procedure produced immediate clinical improvement (NRS 2/10). The patient also was given an additional levodopa 100 mg/benserazide 25 mg once daily. On follow up 2 weeks and three months post injection, patient reported the pain still in NRS 2/10.

Case 2: A 61-year-old male with PSP DD/vascular parkinsonism (2-year duration), ischemic stroke, coronary artery disease, hypertension, stage 3 chronic kidney disease, vascular cognitive impairment, heart failure with ejection fraction 45%, controlled DM, diabetic foot ulcer and polyneuropathy presented with chronic low-back pain with mixed nociceptive neuropathic features with NRS 7/10 with movement. The patient uses a wheelchair for mobility. Pain assessment demonstrated a chronic (>3 months) and progressive pain localized to the lower back, involving bilateral lumbar region with predominant involvement at the L4–L5 level.

Clinically, it presented as neuropathic mechanical low-back pain with background neuropathic features, supported by a DN4 score of 6/10. The pain was aggravated by lumbar extension, flexion, rotation, and physical activity. The pain was associated with referred discomfort limited to above the knee, without radicular pain, without predominant nocturnal or rest pain. The pain was not associated with the initiation of parkinsonism symptoms, did not worsen in relation to rigidity, bradykinesia, and tremor. No other abnormal movements were observed. Physical examination revealed asymmetric bradykinesia (left > right), rigidity (left > right), upward gaze palsy, dysphagia, dysphonia, dysarthria, and postural instability.

The pain consistent with bilateral facet joint pain syndrome at level lumbar 4-5, fulfilling diagnostic criteria including facet joint tenderness, pain worsens with extension, flexion, and ipsilateral rotation, and referred leg pain limited to above the knee. Lumbar spine MRI revealed multilevel lumbar intervertebral disc bulging with associated central canal and neural foramina narrowing. There are fatty marrow changes at the anterior superior and inferior vertebral corners from L1-L5, suggestive of an underlying spondyloarthropathy. Bilateral facet arthropathy is present from L1–L2 through L5–S1, with facet joint effusions at L3–L4 and L4–L5, and ligamentum flavum hypertrophy from L3–L4 to L5–S1. Initial pain management with natrium diclofenac (2×50 mg), gabapentin (2×300 mg), and amitriptyline (1x12.5 mg nightly) and physiotherapy led to only mild improvement. In accordance with multimodal management principles and consideration of diagnostic/therapeutic blocks for suspected facetogenic pain, an USG guided lumbar facet joint intraarticular injection with 1 ml mixture of dexametason 2,5mg/0,5 ml and lidocaine 2% 0,5 ml was performed, producing immediate reduction in pain intensity (NRS 3/10) that stable until 2 weeks post intervention, and but the pain soon came back again. Patient also received initiation of levodopa 100 mg/benserazide 25 mg twice daily.

Table 1. Pain Assessment, Intervention, and Follow Up

	Case 1 : 69-year-old male	**Case 2 :** 61-year-old male
	Diagnosis & Current Treatment	
Diagnosis	Clinically probable PD (3-year duration), advanced PD / Hoehn & Yahr stage 4, moderate-severe disability, controlled hypertension & DM	PSP DD/vascular parkinsonism (2-year duration), ischemic stroke, coronary artery disease, hypertension, stage 3 chronic kidney disease, vascular cognitive impairment, heart failure (ejection fraction 45%), DM, diabetic foot ulcer & polyneuropathy
Medication	Acute upper back pain / thoracic spine pain, left paraspinal rigidity - Levodopa 100 mg/Carbidopa 25 mg/Entacapone 200 mg (LCE) 3x1 - Pramipexole 0,375 mg 1x1 - Celecoxib 200 mg 2x1	Chronic low back pain due to bilateral lumbar facet joint arthropathy at level L4-5 -Natrium diclofenac (2×50 mg), gabapentin (2×300 mg), amitriptyline (12.5 mg nightly) - Physical therapy.
Pain Assessment		
Onset	Acute (<3 months)	Chronic (>3 months)
Provocative /Palliative	Provoked with movement and axial activity. No relief from oral analgesics	Provoked by lumbar extension, flexion, rotation, and physical activity. Minimal relief from oral analgesics
Quality	Nociceptive, non-cancer Pain Detect 4, DN4 3	Mixed nociceptive neuropathic, non cancer Pain Detect 14, DN4 6
Region/ Referral	Upper back pain with predominant involvement of left paraspinal region. No radiation / referral	Low back pain involving bilateral lumbar region with predominant involvement at the L4–L5 level
Severity	NRS 8/10 with movement	NRS 7/10 with movement
Temporal	Persistent with movement	Persistent with movement
PD related	No	No
Physical Examination	Marked left-sided thoracic para-spinal rigidity Motoric strength 5/5/5/5 Normal physiologic reflex, negative pathologic reflex Normal sensory	Facet joint tenderness, pain worsens with extension, flexion, and ipsilateral rotation, referred leg pain limited to above the knee. Motoric strength 5/5/5/5 Reduced physiologic reflex, negative pathologic reflex Hypestesi stocking and gloves
Functional Disability	ODI INA 40% (severe disability) Good social and family support ADL partially dependent	ODI INA 50% (severe disability) Good social and family support ADL partially dependent
	Pain Intervention	
Pain Intervention	Erector Spinae Plane (ESP) Block USG Guided Using 10 ml mixture of lidocaine 2% 4 cc, dexamethasone 5 mg/1cc and Dextrose5% Additional: Levodopa 100 mg / benserazide 25 mg 1x1.	Lumbar facet joint intraarticular injection USG Guided Using 1 ml mixture of dexamethasone 5mg / 0,5cc and lidocaine 2% 0,5cc Initiation of Levodopa100 mg/benserazide 25 mg 2x1. Additional: duloxetine 60 mg 1x1
Follow Up 3 months	NRS 2/10, improvement persist until 3 months.	NRS 3/10, improvement persist until 2 weeks

Pain Detect 0-12: no neuropathic pain; 13-18: possible neuropathic pain, 19-38: Neuropathic pain.
DN4 > 4: Neuropathic pain
ODI: Oswestry Disability Index

DISCUSSION

Globally, parkinsonism affected about 25.2 million people in 2050, and Indonesia is projected to rank 6th worldwide in PD cases by 2050, along with an increased aging population and life expectancy.[21] Wijaya & Pinzon (2018) in Indonesia reported pain was present in over 70.96% of patients with PD, comparable to most studies.[22] In PD, chronic pain affects up to 80% during the course of the disease, the rest 20% present at early motor stage. In case 1, pain is present during the middle motor stage. Unresolved acute pain management tends to become chronic, which should be prevented. Pain is said to be more prevalent in PD patients due to several factors. Even though pain is different from nociception phenomena, nociceptive processing in PD is influenced by basal ganglia–cortical circuitry and brainstem-spinal networks, consistent with a neuroanatomy-based framework for clinical reasoning about pain generators in muscle, bone, joint, disc, ligament, fascia, and nerve.[7,18,19] Beyond dopaminergic pathways, glutamatergic mechanisms likely contribute to central sensitization and clinical pain expression, explaining why PD as a chronic disease is also a risk factor for pain chronification in both cases.[14]

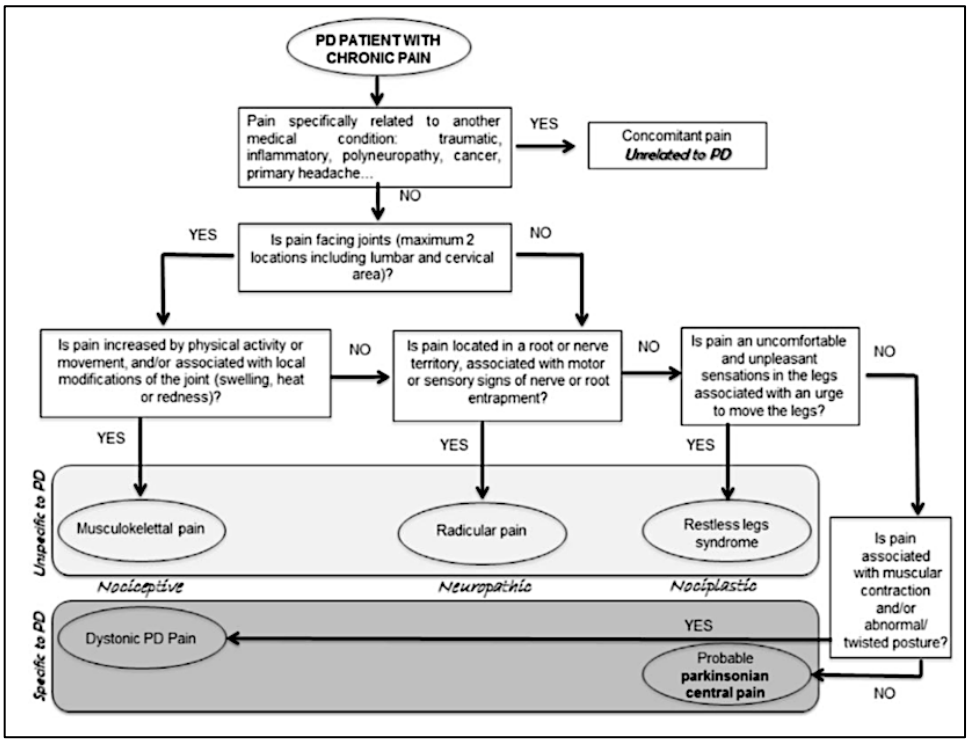
Pain in PD spans musculoskeletal pain, central pain, radicular/neuropathic pain, and dystonic pain, with musculoskeletal/spinal as the most common in clinical cohorts, as we found in both cases.[4,6,7,10] Musculoskeletal pain was found in about 45.2% of PD patients, followed by nocturnal pain (25.8%) and radicular pain (25.8%), and majority found in Hoehn & Yahr stage III. Pain is an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage and always a personal experience that is influenced by biological, psychological, and social factors, so even its subjective, we should try to characterized it by using comprehensive pain assessment, as described in both cases.

Within the context of PD, additional domains were considered using the framework of the King's Parkinson's Disease Pain Scale, to phenotype pain and track response.[8] We characterized pain as musculoskeletal pain when muscle and bone aching, with arthritic changes, myalgias in muscle and possibly worsened with movement, such as in both cases. Neuropathic / radicular pain characterized by numbness, tingling, burning, and restricted to affected nerve. A radicular neuropathic component is unsupported in either case, although neuropathic symptoms from diabetic polyneuropathy are present in Case 2 and absent in Case 1. The pain quality of nociceptive and neuropathic can be differentiated through history taking and supported by standardized questionnaires such as DN4 and pain DETECT. Central pain characterized by diffuse aching-burning, cramping or intermittent sharp pain and neuropathic pain but not restricted to affected nerve root. Akathisia characterized by restlessness and may fluctuate with dopaminergic treatment. Dystonia is prolonged, involuntary muscle contraction or forceful rigidity resulting in tissue damage, that may occur in any muscle group and most commonly present during "off" period. Central pain is possible in Case 2, particularly given recent initiation of levodopa, and reassessment after dopaminergic titration is planned; it is not evident in Case 1. Dystonic pain is unlikely in both cases because no dystonia was observed.

The next step for pain assessment in PD is to evaluate whether it is PD-related or not by using Parkinson's Disease Pain Classification System (PD-PCS) questionnaire, followed by assessing pain intensity, frequency, and impact on daily living. Pain is PD-unrelated if it was not started or became more severe after the initiations of PD symptoms, did not worsen in relation to bradykinesia, rigidity, tremors, there was no chorea or dyskinesia, and the pain did not improve or show temporal relationship with the administration of antiparkinsonian therapy. We can also observe whether the pain may fluctuate with medication changes or may worsen during "off" period. Both cases are PD-unrelated. But we must keep in mind, even pain is categorized as PD-unrelated or PD-unspecified such

as in musculoskeletal, radicular, and restless leg syndrome type, pain severity and distribution correlate and amplified with motor and non-motor symptoms. Motor symptoms such as rigidity and akinesia often exacerbating nociception and functional limitation, such as in case 1 where marked rigidity persist.[11,12] Non-motor symptom such as sleep impairment and mood symptoms (depression/anxiety) frequently co-exist and associate with higher pain severity indices, supporting integrated management.[13,20] So in both cases, first step of treatment is that we should still optimize antiparkinsonian therapy. We did adjustment of antiparkinsonian doses in case 1, and initiation challenge antiparkinsonian medicine in case 2, even though PSP usually has poor response to levodopa therapy. The negative impact of pain on quality of life is substantial, and discrete pain subtypes may differentially reduce health-related quality metrics.[8] In both cases, we don't do follow up on functional outcome as limitation in this study.

Figure 1. PD Patient with Chronic Pain Algorithm



Next step we should dissect pain generators in muscle, bone, joint, disc, ligament, fascia, and nerve. In first case, upper-back pain behaved mechanically, with other signs such as sacroiliac, piriformis, and facet were all absent, which supports interfascia generator. Sacroiliac joint provocative test (compression, distraction, Patrick/FABER, thigh thrust, Fortin finger, and Gillet) were all negative, and there was no midline discogenic tenderness. The second case illustrates how a predominantly nociceptive, non-radicular spinal pain can coexist with a background of neuropathic symptoms from diabetic polyneuropathy. The clinical picture pointed away from radiculopathy: straight-leg raising was negative, there was no dermatomal radiation, and neurological examination did not reveal root deficits. By contrast, bedside provocation consistently reproduced pain with extension, flexion, and ipsilateral rotation, there was focal facet tenderness, and referred discomfort remained above the knee. Lumbar MRI then provided an anatomic substrate—multilevel

facet arthropathy with effusions and ligamentum flavum hypertrophy, which supported a facetogenic generator.

There is no specific therapy for pain in PD or parkinsonism. Even though the National Institute for Health and Care Excellence (NICE) guideline stated that pain in PD is frequent, but did not provide specific pharmacological recommendations beyond standard care. Pain management is tailored to the individual's symptoms, preferences, and goals. It takes multimodal approach, with PD diagnosis should be reviewed every 6–12 months. Non-pharmacological interventions are the cornerstone of pain management in PD with physiotherapy & regular physical activity can overcome muscle stiffness, rigidity, balance, posture, and flexibility, which are primary causes of musculoskeletal pain. Occupational therapy, self-management techniques and psychological support are also equally important. Best practice in pharmacological interventions begins with optimization of dopaminergic therapy (to address rigidity, motor fluctuations, and dystonia), followed by analgesic tailored to pain subtype (NSAID or simple analgesia for nociceptive components; anticonvulsant or antidepressant for neuropathic features), rehabilitation, and psychosocial interventions.[4-6,10,19] Ensuring levodopa is taken on time, as per the NICE standard, helps maintain consistent symptom control and reduce pain. Uncontrolled pain warrants seeking advice from pain specialist. In patients with inadequate response to medication and therapy, carefully selected Interventional Pain Management (IPM) procedures such as diagnostic/therapeutic blocks, may be embedded within a multimodal plan. IPM has several advantages such as act as diagnostic beside therapeutic, act as local anesthetic that directly work on the affected site, reducing the use of oral analgetic, especially in elderly with polypharmacy and indicated in step 4 of modified WHO stepladder (Figure 2). In case 1, a targeted ESP block offered rapid improvement that persist until 3 months. The most probable primary mechanism of ESP block in reducing pain is a direct effect of local anesthetic via physical spread and diffusion to neural structures in the fascial plane deep to the erector spinae muscles and adjacent tissue compartments.

These include neural blockade and central inhibition from direct spread of local anesthetic to the paravertebral or epidural space; analgesia mediated by elevated local anesthetic plasma concentrations due to systemic absorption; immunomodulatory effects of local anesthetics; and an effect mediated through the mechanosensory properties of thoracolumbar fascia. Biological plausibility of this primary mechanism is confirmed by injectate spread to the ventral rami of spinal nerves (though quite variable) in most studies. In case 2, a targeted intraarticular facet joint injection offered rapid improvement, but not for a long time. In cases where intervention did not give satisfactory result, we should evaluate and re-diagnosis whether the pain come from same pain generator or not, evaluate our technique, or escalate to further treatment such as Medial Branch Block (MBB) diagnostic injection to medial branches nerve that carry pain signals form facet joint, continued by Radiofrequency Ablation (RFA) of MBB or escalate to surgery.

Although high-quality randomized data for many interventions for PD pain are limited[5,6,19], both patients in our case series experienced improvement and reduction in pain scores following intervention. Limitation of this study is only 2 cases presented and no long term follow up data.

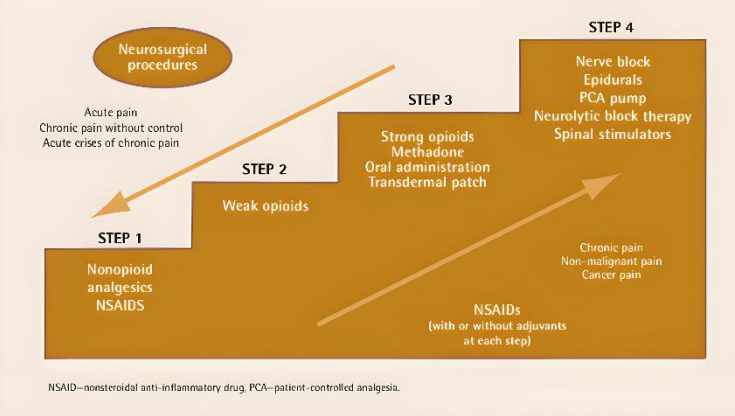


Figure 2. Modified WHO stepladder for chronic non cancer pain

CONCLUSION

Pain in PD is common yet underrecognized, clinically heterogeneous, and frequently disabling, warranting proactive, structured assessment and management. A "pain-generator" framework anchored in neuroanatomy and PD-specific pathophysiology can guide targeted therapy, including the selective use of diagnostic/therapeutic blocks when conservative measures fail. Incorporating standardized tools can improve phenotyping and outcome tracking in both practice and research. Larger prospective studies are needed to define the effectiveness and durability of interventional strategies for pain in parkinsonism.

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