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Imaging the Spectrum of Tremor: Integrating Clinical Classification With Radiologic Findings

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Abstract

Tremor is one of the most prevalent movement disorders encountered in clinical practice, with a broad spectrum spanning neurodegenerative, metabolic, vascular, viral, and functional etiologies. While many tremors are diagnosed clinically, neuroimaging plays a critical role in identifying underlying causes, distinguishing among overlapping symptoms, and guiding therapy planning. Despite this, comprehensive imaging-based resources designed for radiologists are limited - especially those grounded in real-world imaging examples and structured for educational use. This guide includes a case-based multimodality visual review of important tremor syndromes with radiologic relevance. Drawing on real-world cases presented during departmental teaching and conference settings, we provide a structured overview of rest and action tremors, highlighting distinctive imaging patterns across modalities, including MRI, CT, and DaTscan. Additional attention is put on interventional therapies such as deep brain stimulation (DBS) and MRI-guided focused ultrasound (MRgFUS), with discussion of post-treatment imaging interpretation and pitfalls. The review encompasses a spectrum of tremors - including Parkinson's disease, essential tremor, dystonic tremor, Wilson's disease, multiple system atrophy, cerebellar infarcts, and lithium toxicity - with matching imaging findings and pathological insights. This graphical reference serves as a practical and informative resource for radiologists, especially trainees, presenting high-yield imaging patterns, clinical correlations, and procedural concerns in the evaluation of tremors. By reinforcing pattern recognition and diagnostic confidence, this review intends to enhance radiologists' role in the multidisciplinary management of patients with tremor.

Categories: Nuclear Medicine, Neurology, Radiology
Keywords: datscan, deep brain stimulation, essential tremor, movement disorder, mri, neuroimaging, parkinson's disease, tremor

Introduction And Background

Tremor is one of the most common movement disorders encountered in clinical practice, posing a diagnostic difficulty because of its varied etiologies and overlapping clinical characteristics [1]. Broadly categorized into resting, action, and postural kinds, tremors can result from neurodegenerative, vascular, toxic-metabolic, or structural causes. Accurate categorization is vital, as management techniques and prognoses vary greatly across the spectrum [1,2].

Neuroimaging plays a vital role in the evaluation of tremor, notably in separating Parkinsonian disorders from mimickers such as essential, dystonic, or psychogenic tremors [3]. Structural modalities like MRI and CT assist in revealing underlying lesions, atrophy patterns, or secondary causes, while functional imaging - particularly the I-123 ioflupane DaTscan - offers valuable insights into dopaminergic circuit integrity [4,5]. Advances in MRI-guided focused ultrasound (MRgFUS) and deep brain stimulation (DBS) have also provided new imaging considerations in post-treatment assessment [6,7].

Despite their diagnostic value, imaging findings in tremor disorders can be modest or nonspecific, requiring careful integration of clinical context, drug history, and neuroanatomical connection. While textbook descriptions primarily focus on archetypal patterns, real-world cases frequently arrive with diagnostic ambiguity [8].

This article is a case-based, multimodality pictorial review of tremor syndromes, with an emphasis on radiologic features, differential diagnosis, and therapeutic implications. By covering both common and rare causes of tremors, this article serves as a high-yield visual reference for radiologists, neurologists, and trainees, providing practical tools to approach tremor evaluation with greater confidence and clarity.

Review

Imaging modalities overview

Neuroimaging serves as a complementary diagnostic tool in the evaluation of tremor disorders, particularly

when clinical findings suggest an underlying structural or neurodegenerative cause. It plays a vital role in identifying structural reasons, assessing functional dopaminergic activity, and guiding post-treatment evaluations. The three basic modalities in clinical practice are DaTscan, MRI, and CT, each offering distinct advantages depending on the clinical scenario (Table *1*).

Modality	Primary Role	Key Diagnostic Features	Clinical Utility	Pitfalls / Limitations
DaTscan (I-123 SPECT)	Functional assessment of presynaptic dopaminergic neurons	Symmetric vs. asymmetric uptake in the striatum	Differentiates Parkinsonian syndromes from essential tremor	Medication interference; SWEDD requires further clinical evaluation and does not necessarily indicate a false-negative scan; limited availability
MRI	Structural brain imaging	Hummingbird sign (PSP), hot cross bun (MSA), infarcts, DBS/MRgFUS findings	Identifies degenerative or secondary causes; treatment planning	May be normal in early disease; requires clinical correlation
CT	Rapid screening	Infarct, hemorrhage, mass, calcification	Useful in acute settings or MRI contraindications	Lower sensitivity, poor brainstem resolution

TABLE 1: Imaging Modalities in Tremor Evaluation

Overview of the primary imaging modalities used in tremor evaluation [7,9-12].

Abbreviations: DaTscan, dopamine transporter scan; SPECT, single-photon emission computed tomography; MRI, magnetic resonance imaging; CT, computed tomography; PSP, progressive supranuclear palsy; MSA, multiple system atrophy; DBS, deep brain stimulation; MRgFUS, magnetic resonance-guided focused ultrasound; SWEDD, scan without evidence of dopaminergic deficit.

DaTscan (I-123 Ioflupane SPECT Imaging)

DaTscan is a nuclear medicine technology that examines presynaptic dopaminergic neuron integrity by imaging dopamine transporter (DaT) density in the striatum [5]. It is particularly useful in distinguishing degenerative parkinsonian syndromes from essential tremor and other conditions without presynaptic dopaminergic deficits. However, DaTscan cannot reliably differentiate Parkinson's disease from atypical degenerative parkinsonian syndromes, such as multiple system atrophy (MSA) or progressive supranuclear palsy (PSP). Typical findings include symmetric comma-shaped uptake in the striatum on normal scans, whereas abnormal scans (e.g., Parkinson’s) indicate decreased or asymmetric uptake, notably in the putamen. Clinically, DaTscan offers good diagnostic support in early or unclear cases of Parkinsonism. Other functional imaging modalities may provide complementary diagnostic information. ^{18}F-FDOPA PET assesses presynaptic dopaminergic function, similar to DaTscan, although their diagnostic performance varies according to the clinical setting and indication. In contrast, ^{18}F-FDG PET evaluates cerebral glucose metabolism and may reveal characteristic metabolic patterns that help differentiate Parkinson's disease from atypical parkinsonian syndromes, including PSP, MSA, and corticobasal degeneration. These modalities are not routinely required for tremor evaluation but may be useful in diagnostically uncertain cases. DaTscan is typically normal in pure drug-induced parkinsonism, whereas reduced striatal uptake may suggest underlying degenerative parkinsonism. SWEDD (scan without evidence of dopaminergic deficit) describes patients with clinical features suggestive of Parkinson's disease but normal dopaminergic imaging. It represents a heterogeneous clinical population rather than a distinct tremor syndrome or necessarily a false-negative DaTscan. Certain medications, including bupropion, methylphenidate, and amphetamine-based stimulants, may interfere with dopamine transporter binding and affect DaTscan interpretation. However, not all antiparkinsonian or psychiatric medications significantly influence scan results, making medication review important before imaging [5,9].

MRI

MRI delivers high-resolution structural imaging of the brain and brainstem. It is crucial for detecting secondary causes of tremor and visualizing neurodegenerative processes. MRI findings may include midbrain atrophy (“hummingbird sign”) in PSP, the “hot cross bun” sign in MSA with predominant cerebellar features (MSA-C), cerebellar infarcts, demyelinating lesions, or structural changes following DBS or MRgFUS. These findings are supportive rather than pathognomonic and may be absent in early disease. Clinically, MRI is useful across a wide spectrum of tremors, especially when a structural or degenerative origin is suspected [6,7,10].

CT

CT is less sensitive than MRI, but remains effective in acute circumstances or when MRI is contraindicated. Its utility includes rapid examination for bleeding, infarction, masses, or calcifications. Its limitations include poor soft tissue contrast and poor brainstem resolution [11].

Additional or Emerging Imaging Techniques

Several other imaging modalities, while less commonly used in routine clinical settings, give complementary insights into certain instances or research settings [13-15]. These additional techniques are summarized in Table *2*. FDG-PET assesses cerebral glucose metabolism and can assist in identifying atypical Parkinsonism subtypes. MIBG cardiac scintigraphy may help distinguish Parkinson's disease and dementia with Lewy bodies from MSA based on cardiac sympathetic innervation. Its findings may support the proposed "body-first" hypothesis but cannot establish the site of disease initiation. Cardiac disease, diabetes, and certain medications may also affect MIBG uptake, limiting its diagnostic specificity. Susceptibility-weighted imaging (SWI) and diffusion tensor imaging (DTI) enhance visualization of certain brain regions or tracts. Functional MRI (fMRI) and MR spectroscopy are mainly utilized as research techniques in this context [13-16]. These technologies broaden the diagnostic toolkit and may gain broader clinical value as access and validation improve.

Modality	Use in Tremor Evaluation	Comment
FDG-PET	Assesses cerebral metabolism	Helps differentiate PSP (frontal hypometabolism), MSA (cerebellar hypometabolism)
MIBG Scintigraphy	Evaluates cardiac sympathetic innervation	Reduced uptake in PD; preserved in MSA; helps in differentiation
SWI (part of MRI)	Enhances substantia nigra visualization	Loss of the "swallow tail" sign in PD
DTI	Evaluates white matter tracts	Research use in cerebellothalamic and basal ganglia pathways
fMRI	Maps task-related activation	Investigational in tremor network studies
MR Spectroscopy	Evaluates metabolic patterns	Limited clinical role; occasionally used in ataxia or tumor workups

TABLE 2: Additional Imaging Techniques in Tremor Evaluation

Additional imaging techniques used in tremor evaluation [13-16].

Abbreviations: FDG-PET, fluorodeoxyglucose positron emission tomography; MIBG, metaiodobenzylguanidine; SWI, susceptibility-weighted imaging; MRI, magnetic resonance imaging; DTI, diffusion tensor imaging; fMRI, functional magnetic resonance imaging.

Case-based review of tremor syndromes

Tremor syndromes are a wide range of neurologic diseases defined by rhythmic, involuntary muscular contractions [1]. Clinically, tremors are classified as resting or action tremors, with postural tremor representing a subtype of action tremor [2]. Each group contains numerous etiologies, from neurodegenerative illnesses to toxic, metabolic, structural, and functional factors [2,17].

Neuroimaging acts as a significant supplement to clinical evaluation by revealing structural abnormalities, aiding differential diagnosis, and, in certain situations, directly influencing therapeutic decisions. This section presents a set of representative tremor syndromes with related imaging findings across modalities, highlighting high-yield diagnostic patterns and significant interpretive challenges.

To guide this review, we present a structured tremor classification flowchart (Figure *1*) that serves as a conceptual foundation for the case discussions that follow.

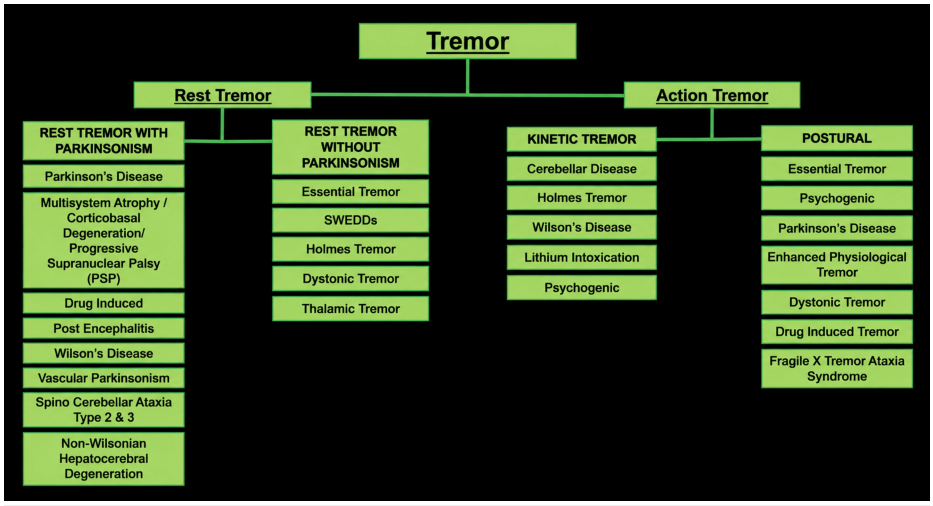


FIGURE 1: Classification of Tremor Syndromes

A hierarchical schema of tremor subtypes categorized by motor pattern (resting vs. action, including postural tremor) and associated etiologies.

Abbreviations: SWEDD, scan without evidence of dopaminergic deficit; PSP, progressive supranuclear palsy.

The following scenarios are intended to emulate the diagnostic thinking process radiologists experience in actual practice. By incorporating clinical symptoms, imaging patterns, and typical mistakes, each vignette reinforces fundamental ideas in tremor evaluation - making this section particularly relevant for resident teaching and board review.

Parkinson's Disease

Clinical overview: Parkinson's disease is the most prevalent neurological cause of resting tremor. It often appears with bradykinesia, stiffness, and resting tremor that commonly starts asymmetrically. Additional characteristics may include postural instability and micrographia. Tremor in Parkinson's disease is classically described as "pill-rolling" and may lessen with intentional movement.

Imaging findings: DaTscan shows asymmetric reduction of tracer uptake in the posterior putamen, often contralateral to the more clinically affected side (Figure *2*). The caudate may be relatively spared in early stages. MRI SWI may demonstrate loss of the "swallow tail sign" in the substantia nigra, a marker of nigrosome-1 degeneration. This finding may support the diagnosis of degenerative parkinsonism but is not specific to Parkinson's disease and may occur in other degenerative parkinsonian syndromes [4-6,9].

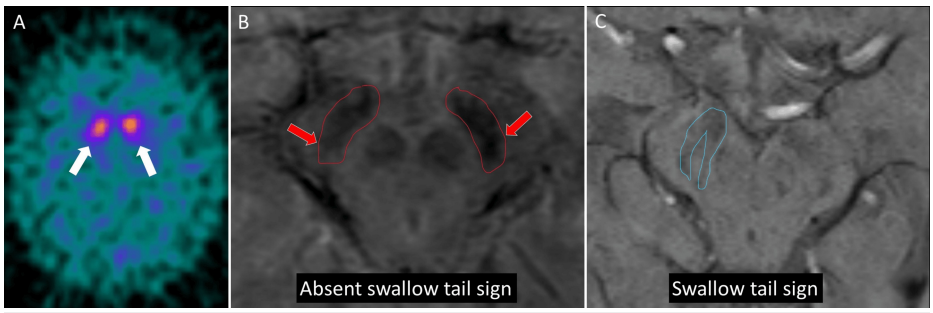


FIGURE 2: Parkinson's Disease Imaging Findings

(A) DaTscan showing asymmetric right putaminal uptake loss (white arrows). (B) SWI MRI demonstrating an absent swallow tail sign in the substantia nigra (red arrows). (C) SWI MRI demonstrating a normal swallow tail sign in the substantia nigra in a normal individual (blue outlined area).

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Diagnostic considerations: The differential diagnosis includes essential tremors (normal DaTscan), drug-

induced parkinsonism (typically normal DaTscan; reduced uptake may suggest underlying degenerative parkinsonism), vascular parkinsonism (stepwise progression with vascular burden), and atypical parkinsonian syndromes (e.g., MSA, PSP). False positives on DaTscan may result from drugs such as amphetamines or SSRIs. SWEDD represents a heterogeneous clinical population with normal dopaminergic imaging and should not automatically be interpreted as a false-negative DaTscan. DaTscan is particularly helpful in early or indeterminate parkinsonism, while MRI aids in ruling out secondary structural causes and provides ancillary signs when present [4-6,9,18].

Essential Tremor

Clinical overview: Essential tremor is the most common action tremor and one of the most prevalent movement disorders overall. It often appears as a bilateral postural and kinetic tremor, often affecting the wrists and forearms, with probable involvement of the head and voice. Unlike Parkinson's disease, essential tremor frequently spares the lower limbs and lacks accompanying stiffness or bradykinesia. Tremor may momentarily improve with alcohol and worsen with stress or weariness [4,15,18-20].

Imaging findings: DaTscan is typically normal in essential tremor, with symmetric comma-shaped uptake in the caudate and putamen (Figure *3*). This is a key distinguishing feature from parkinsonian syndromes. MRI/CT is often unremarkable, with no consistent structural abnormalities seen. Although subtle cerebellar atrophy has been reported in some cases of essential tremor, it is not an established or specific diagnostic imaging marker [4,15,18-20].

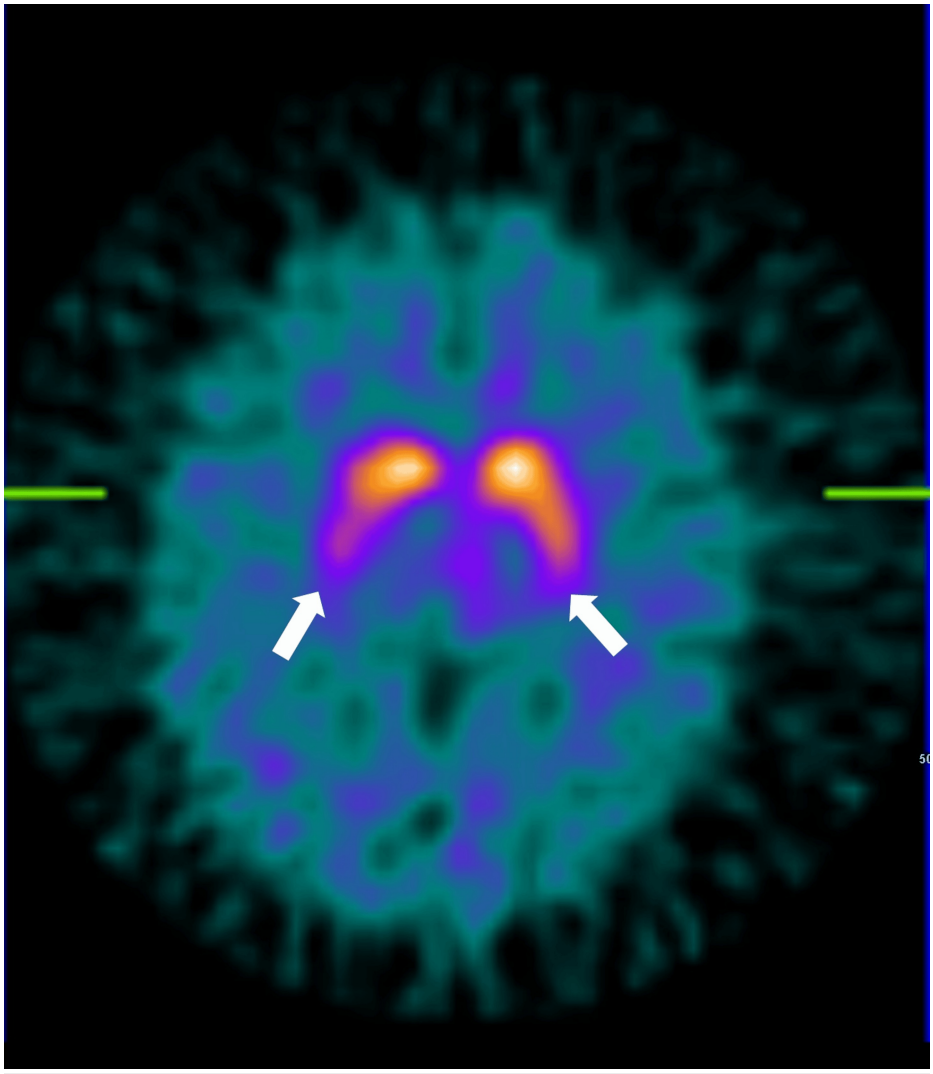


FIGURE 3: Essential Tremor DaTscan Imaging

DaTscan in essential tremor showing symmetric, normal tracer uptake (comma-shaped) in the striatum (white arrows).

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Diagnostic considerations: The differential diagnosis includes early Parkinson's disease (DaTscan abnormal), enhanced physiological tremor, dystonic tremor, and functional (psychogenic) tremor. False-positive diagnoses of Parkinson's can occur if clinical assessment overemphasizes tremor without evaluating for bradykinesia or rigidity. Conversely, essential tremor with significant asymmetry or a rest component may lead to unnecessary imaging if interpreted without clinical context. DaTscan is not routinely indicated for isolated action tremor but may assist in diagnostically uncertain cases. A normal scan does not completely exclude Parkinson's disease. Imaging is also useful in atypical cases or when considering alternative diagnoses [18-20].

PSP

Clinical overview: PSP is a neurodegenerative parkinsonian syndrome characterized by early postural instability, frequent falls, and vertical gaze palsy. Resting tremor is often absent or minimal, but axial rigidity and bradykinesia are prominent. PSP is often misdiagnosed as Parkinson's disease in its early stages [21-23].

Imaging findings: MRI (sagittal T1) demonstrates the classic "hummingbird sign" or "penguin sign" caused by selective midbrain atrophy with relative pontine preservation (Figure *4*). DaTscan demonstrates bilateral symmetric reduction in striatal uptake, often involving both the caudate and putamen. Unlike Parkinson's, this reduction tends to be more symmetric and diffuse [21-23].



FIGURE 4: Progressive Supranuclear Palsy Imaging Findings

Midbrain atrophy on sagittal T1 MRI showing the classic hummingbird sign (white arrow).

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Diagnostic considerations: The differential diagnosis includes Parkinson's disease (asymmetric uptake, slower axial symptoms), MSA (hot cross bun sign, autonomic dysfunction), and corticobasal degeneration. Early PSP may have subtle or no imaging findings, and DaTscan alone cannot reliably distinguish PSP from other parkinsonian syndromes, requiring clinical correlation. MRI provides key structural clues (midbrain atrophy), while DaTscan helps confirm presynaptic dopaminergic deficit but lacks specificity [21-23].

MSA

Clinical overview: MSA is a rapidly progressive, adult-onset parkinsonian syndrome with autonomic dysfunction. It presents in two major subtypes: MSA-P (Parkinsonian type), which presents with bradykinesia and rigidity, mimicking PD, and MSA-C (cerebellar type), which is dominated by cerebellar ataxia and gait instability [17-19,24].

Imaging findings: MRI (axial T2 and FLAIR) may show putaminal atrophy with lateral margin hyperintensity (putaminal rim sign) in MSA-P, while MSA-C demonstrates the classic "hot cross bun sign" in the pons due to degeneration of pontocerebellar fibers (Figure 5). DaTscan shows bilateral reduced uptake, similar to PSP, but without clear caudate-sparing, and may be slightly more asymmetric than PSP [17-19,24]. Under the 2022 MDS diagnostic criteria, characteristic MRI markers are required for clinically established MSA but not for clinically probable MSA. The putaminal rim sign has limited specificity, particularly on 3T MRI, where it may also be observed in healthy individuals.



FIGURE 5: Multiple System Atrophy – Cerebellar Type (MSA-C)

(A) Axial T2-weighted image demonstrating the classic hot cross bun sign (white arrows) - T2 hyperintensities in the transverse pontocerebellar tracts within a flattened pons. (B) Sagittal T1-weighted image showing disproportionate pontine (white arrow) and cerebellar atrophy, including the middle cerebellar peduncles, consistent with MSA-C.

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Diagnostic considerations: The differential diagnosis includes Parkinson's disease, PSP, cerebellar ataxias, and corticobasal degeneration. The "hot cross bun" sign is not pathognomonic and can be seen in other cerebellar degenerations, while early MSA may have unremarkable imaging. MRI provides distinguishing signs; DaTscan confirms dopaminergic deficit but requires clinical synthesis for subtype differentiation [24,25]. The key clinical and imaging features of PD, PSP, and MSA are compared in Table *3*.

Feature	Parkinson's Disease (PD)	Progressive Supranuclear Palsy (PSP)	Multiple System Atrophy (MSA)
Typical Onset & Progression	Gradual onset after age 60; slow progression	Onset in 60s-70s; rapid progression	Onset in the 50s-60s; rapid progression
Tremor Type	Resting tremors, often asymmetric	Minimal tremor; axial rigidity	Postural/action tremor; cerebellar or parkinsonian features
Key Clinical Features	Bradykinesia, rigidity, postural instability; tremor improves with action	Early falls, vertical gaze palsy, frontal disinhibition	Autonomic dysfunction (e.g., orthostasis), cerebellar ataxia (MSA-C), or rigidity (MSA-P)
DaTscan Findings	Asymmetric decreased uptake in the putamen; caudate relatively spared	Symmetric reduction in striatal uptake (caudate and putamen)	Bilateral symmetric striatal reduction
MRI Findings	Loss of 'swallow tail sign' on SWI (may be absent in early disease)	Hummingbird sign due to midbrain atrophy	Hot cross bun sign (MSA-C); putaminal atrophy/rim hyperintensity (MSA-P)
Diagnostic Pitfalls	Early PD may show normal MRI; DaTscan false positives with certain drugs	DaTscan does not distinguish from PD; early MRI findings may be subtle	Overlap with PD and PSP; hot cross bun not pathognomonic

TABLE 3: Comparison of PD, PSP, and MSA

Comparison of clinical and imaging features of Parkinson's disease (PD), progressive supranuclear palsy (PSP), and multiple system atrophy (MSA) [21-25].

Spinocerebellar Ataxia (SCA)

Clinical overview: SCAs are a series of genetically heterogeneous, autosomal dominant neurodegenerative

disorders. Tremor in SCAs generally presents as a kinetic or postural tremor, commonly accompanied by gait instability, dysarthria, and oculomotor abnormalities. Ataxia may precede tremor in some conditions [17,26-28].

Imaging findings: MRI (axial and coronal T2) demonstrates atrophy of the cerebellum and middle cerebellar peduncles (MCPs) (Figure 6). Findings are often symmetrical and progressive, with involvement of the spinal cord in advanced disease. Imaging patterns vary among SCA subtypes, and MCP atrophy is not universally present. Advanced imaging with MR spectroscopy may show decreased NAA and increased myo-inositol in cerebellar white matter [26-28].

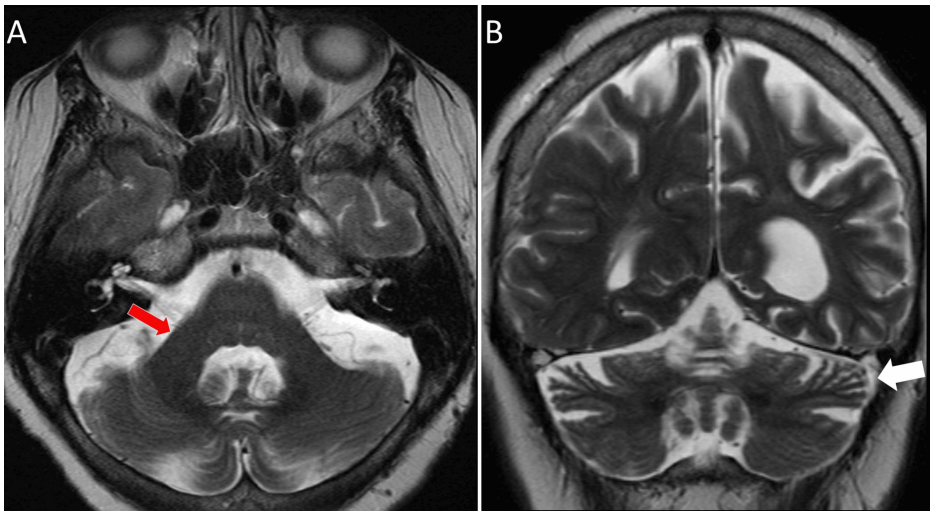


FIGURE 6: Spinocerebellar Ataxia Imaging Findings

(A) Axial T2-weighted MRI showing marked atrophy of the middle cerebellar peduncles (red arrow). (B) Coronal T2-weighted MRI demonstrating cerebellar folia atrophy (white arrow).

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Diagnostic considerations: The differential diagnosis includes MSA-C (which may also affect MCPs but with faster progression), Fragile X-associated tremor/ataxia syndrome (FXTAS), and acquired cerebellar degeneration (e.g., alcohol-induced). MRI findings may overlap with other ataxias, and genetic testing is often required for definitive diagnosis. Imaging confirms neurodegenerative ataxia and helps narrow the differential, especially when distinguishing hereditary from acquired causes [17,26-28].

Dystonic Tremor Due to Prior Thalamic ADEM (Acute Disseminated Encephalomyelitis) Lesion

Clinical overview: Dystonic tremor is defined as a tremor occurring in a body part that is also affected by dystonia. It may be focal or segmental and often presents with irregular amplitude and variable frequency. In some cases, tremor may be secondary to prior inflammatory or structural lesions of the motor pathways, particularly the thalamus or basal ganglia [29-31].

Imaging findings: MRI (axial T2-weighted) shows focal T2 hyperintensity in the right thalamus, a nonspecific finding that may reflect a chronic demyelinating lesion but cannot independently establish prior ADEM. This location is critical, as the thalamus plays a key role in tremor modulation via cerebello-thalamo-cortical pathways (Figure 7) [29-31].

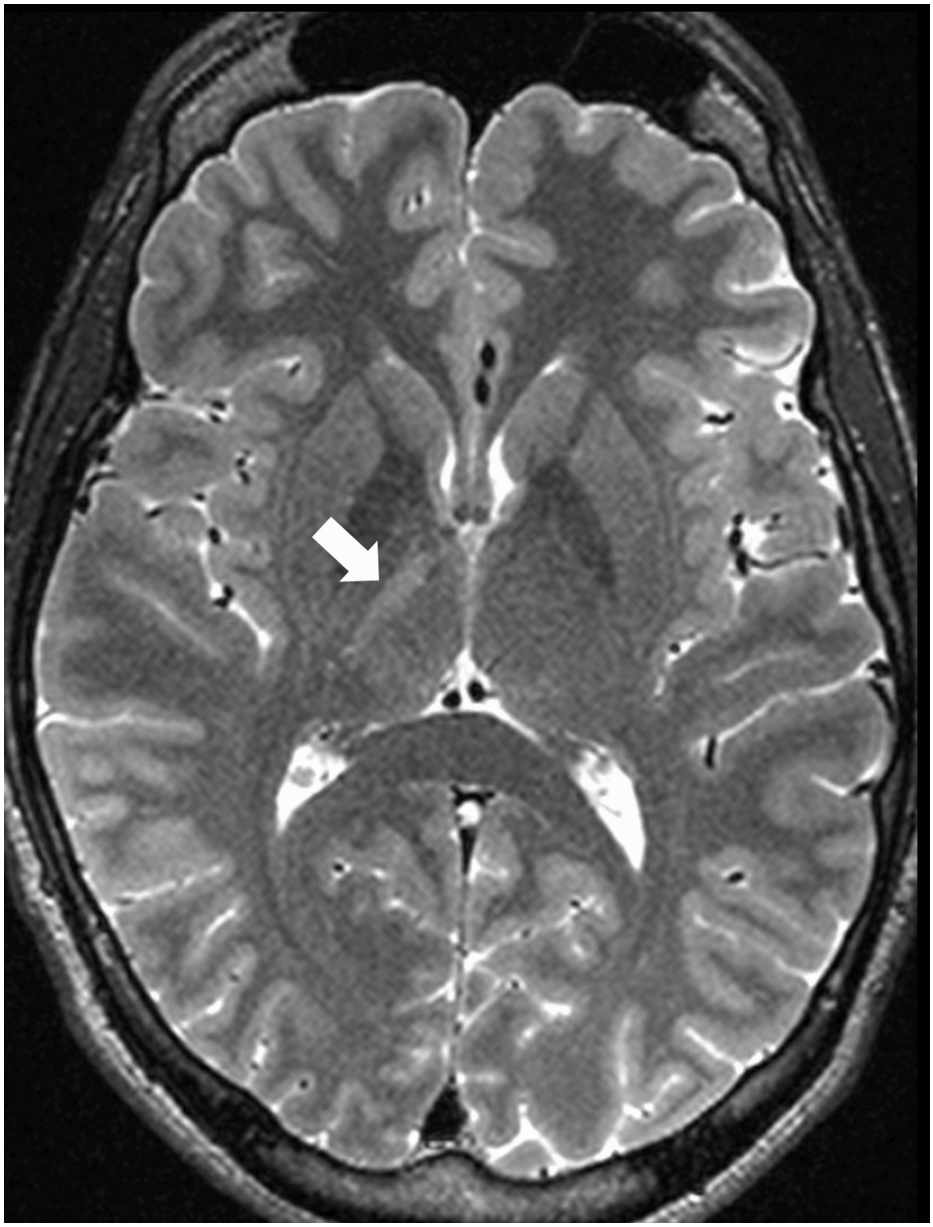


FIGURE 7: Dystonic Tremor Associated With Prior Thalamic ADEM

Axial T2-weighted MRI demonstrating T2 hyperintensity in the right thalamus (white arrow), consistent with a prior ADEM lesion contributing to dystonic tremor.

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Abbreviation: ADEM, acute disseminated encephalomyelitis.

Diagnostic considerations: The differential diagnosis includes Holmes tremor (which may also involve the thalamus but presents with a mixture of rest, postural, and intention tremor), essential tremor, and other secondary causes (e.g., infarct, tumor). Chronic inflammatory lesions may be overlooked if subtle or misinterpreted as nonspecific gliosis, and clinical history is essential. Identifying structural lesions in motor relay centers such as the thalamus can establish a causal link to movement disorders and guide neurologic referral or further etiologic workup [29-31].

Kinetic Tremor Due to Left Cerebellar Infarct

Clinical overview: Kinetic tremor refers to tremor that occurs during voluntary movement and is generally related to cerebellar impairment. Vascular lesions, particularly cerebellar hemispheric infarcts, are a well-recognized cause. Patients may present with unilateral, intention-predominant tremor accompanied by

dysmetria, gait imbalance, or limb ataxia. The tremor generally arises days to weeks after the infarct, reflecting impairment of cerebellar outflow channels, especially the dentate nucleus and superior cerebellar peduncle, which govern smooth motor execution [29-31].

Imaging findings: Axial T2-weighted MRI reveals T2 hyperintensity and parenchymal volume loss in the left cerebellar hemisphere, indicative of chronic infarct. The lesion location correlates with disruption of cerebellar output tracts involved in motor control (Figure *8*) [29-31].



FIGURE 8: Kinetic Tremor Associated With Chronic Cerebellar Infarct

Axial T2-weighted MRI showing chronic infarct in the left cerebellar hemisphere with associated gliosis and atrophy (white arrow), corresponding with contralateral kinetic tremor.

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Diagnostic considerations: The differential diagnosis includes multiple sclerosis plaques (if periventricular), cerebellar degeneration (alcohol, paraneoplastic), and structural malformations. Old infarcts may be misread as nonspecific gliosis without clinical correlation, while bilateral cerebellar lesions may produce truncal ataxia without classic tremor. Imaging confirms lesion localization and etiology, which is essential for distinguishing vascular from degenerative tremor causes and for guiding management and rehabilitation strategies [29-31].

FXTAS

Clinical overview: FXTAS is a late-onset neurodegenerative illness that develops in male carriers of the fragile X premutation (usually 55-200 CGG repeats in the FMR1 gene). It appears with intention tremor, gait ataxia, cognitive deterioration, and autonomic dysfunction. FXTAS is an important differential diagnosis in older adult men presenting with mixed tremor and ataxia, especially with a family history of fragile X syndrome or intellectual disability [28-30].

Imaging findings: Axial T2 or FLAIR MRI demonstrates T2 hyperintensity in the MCPs (MCP sign), considered a hallmark feature of FXTAS. White matter changes may also be seen in the splenium of the corpus callosum and periventricular areas in advanced cases (Figure *9*). The MCP sign reflects degeneration of frontocerebellar tracts and is closely linked to clinical severity [28-30].

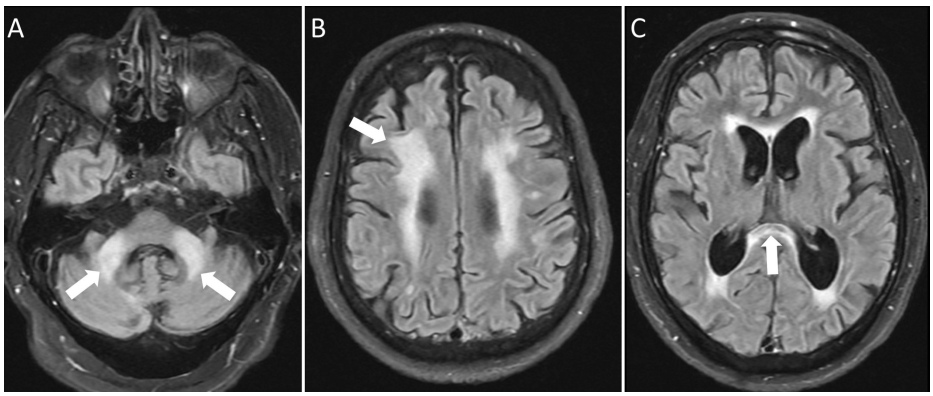


FIGURE 9: Fragile X-Associated Tremor/Ataxia Syndrome Imaging Findings

(A) Axial FLAIR images showing bilateral symmetric T2 hyperintensity of the middle cerebellar peduncles (MCP sign; white arrows), (B) splenium of the corpus callosum (white arrow), and (C) periventricular areas (white arrow), characteristic of FXTAS.

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Diagnostic considerations: The differential diagnosis includes MSA-C (which may also affect MCPs but lacks genetic association), spinocerebellar ataxias, and chronic small vessel disease. The MCP sign is not exclusive to FXTAS; clinical correlation and genetic testing are crucial for diagnosis. Imaging serves as a red flag prompting genetic testing for FMR1 premutation, especially in the setting of characteristic clinical symptoms [28-30].

Kinetic Tremor in Lithium Intoxication

Clinical overview: Lithium, frequently used in the treatment of bipolar disorder, can elicit kinetic or postural tremor even at therapeutic levels. However, toxic buildup, particularly in the setting of decreased renal function, may lead to irreversible neurotoxicity, showing up as tremor, ataxia, confusion, and, in severe cases, permanent neurologic impairments. While most neurotoxic consequences of lithium are clinical, long-term lithium usage can also harm other organs - especially the kidneys - where structural imaging results may be observed [32-34].

Imaging findings: Coronal CT of the abdomen reveals numerous small cortical and medullary microcysts in both kidneys (red arrows). This "moth-eaten" appearance is characteristic of chronic lithium nephropathy (Figure *10*). While this is not a brain imaging instance, the existence of structural renal damage can serve as a proxy signal of prolonged lithium exposure and potential systemic toxicity, including neurologic side effects like tremor [32-34].

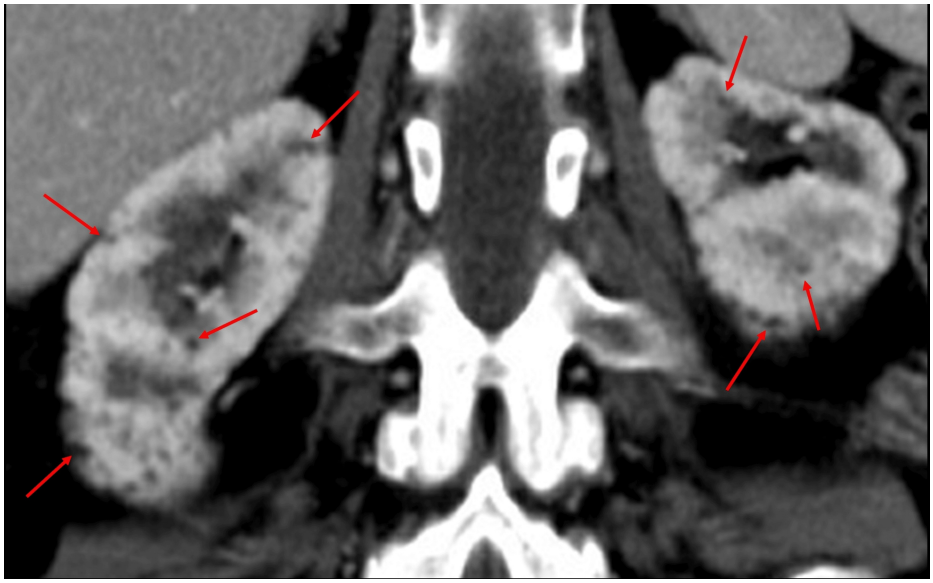


FIGURE 10: Lithium-Induced Tremor With Chronic Lithium Nephropathy

Coronal CT image showing numerous 1–2 mm microcysts diffusely distributed throughout the renal cortex and medulla (red arrows), consistent with chronic lithium nephropathy in a patient with lithium-induced tremor.

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Diagnostic considerations: The differential diagnosis includes polycystic kidney disease (usually larger cysts), acquired cystic kidney disease in dialysis patients, and other toxic nephropathies. Absence of brain imaging abnormalities does not exclude lithium-related neurologic dysfunction, and clinical context is essential. While brain MRI may appear normal, the recognition of systemic lithium toxicity (including renal cysts) should raise concern for lithium-induced tremor, particularly if temporal correlation exists [32,33].

Additional Representative Tremors

To complete the clinical and imaging spectrum, we briefly highlight several additional tremor syndromes of diagnostic importance that may not have specific imaging findings or are less commonly visualized in routine clinical practice.

Holmes tremor: A rare yet dramatic tremor that combines resting, postural, and purposeful components, generally induced by lesions affecting the dentatorubrothalamic tract - often involving the midbrain, thalamus, or cerebellum. MRI may reveal an infarct, demyelination, or trauma involving the red nucleus or adjacent structures. Onset is often delayed weeks to months after the causative insult [2,35].

Wilson's disease: A genetic disorder of copper metabolism leading to tremor, dystonia, and parkinsonism in younger patients (typically <40 years). T2 hyperintensities may be seen in the basal ganglia, thalamus, and midbrain, and the "face of the giant panda" sign may be seen in the midbrain on axial T2. Tremor may be mixed and disabling; Wilson's disease should be considered in all young patients with unexplained parkinsonism or hepatic dysfunction [18,36].

Drug-induced tremor: Medications such as valproate, lithium, SSRIs, and stimulants can cause action or postural tremors. These are often symmetric, low-amplitude, and associated with dose or serum levels. Imaging is typically normal, while resolution with dose adjustment or discontinuation supports the diagnosis [34,37].

Functional (psychogenic) tremor: A non-organic tremor is typically characterized by inconsistency, variability, and entrainment. It often occurs in the setting of psychological distress or conversion disorder. Imaging is normal across all modalities. Diagnosis is clinical; imaging is used to exclude structural or neurodegenerative causes [38,39].

These additional tremor syndromes underscore the complexity of tremor evaluation and the relevance of imaging not only for identifying structural abnormalities but also for guiding diagnosis when imaging is normal. To unify the clinical and imaging patterns covered in this section, we provide a final composite summary that incorporates key tremor subtypes and their representative imaging findings (Figure *11*). This

picture duplicates the diagnostic classification schema presented earlier (Figure *1*) but now grounds the framework in real-world radiologic examples. It serves as a practical reference for evaluating tremor-related imaging and recognizing high-yield indications across modalities.

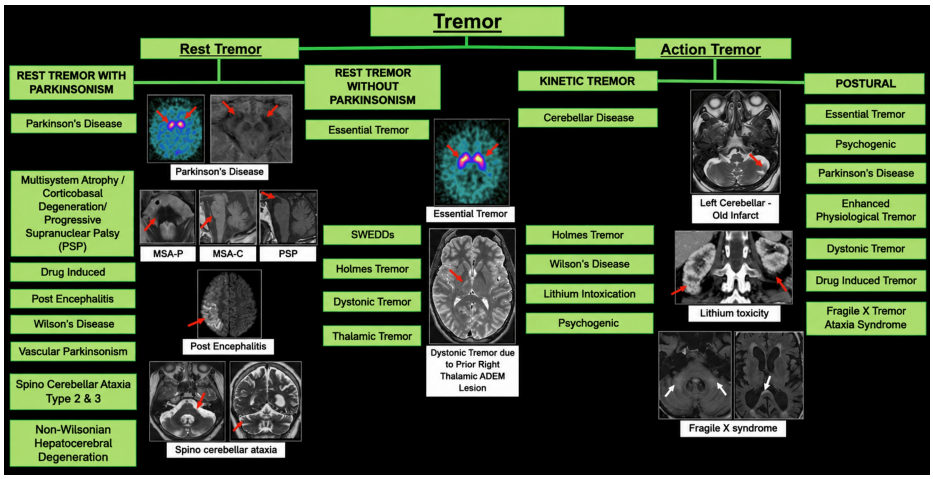


FIGURE 11: Composite Imaging Overview of Tremor Syndromes

Visual synthesis of major tremor subtypes paired with representative neuroimaging findings. The figure mirrors the classification schema in Figure 1, highlighting key structural or functional imaging signs associated with each tremor type. Examples include asymmetric putaminal tracer loss in Parkinson's disease (DaTscan), hot cross bun sign in MSA-C (T2 MRI), hummingbird sign in PSP (sagittal T1), middle cerebellar peduncle hyperintensity in FXTAS (FLAIR), and cerebellar infarct in kinetic tremor (T2 MRI).

This composite figure was created by the authors and is not reproduced or adapted from a previously published figure. The representative imaging examples are from the authors' institutional teaching collection and were used in the development of this educational figure.

To conclude this section, we present a summary table that consolidates the key clinical and imaging features of representative tremor syndromes discussed throughout this review. Although it does not cover all tremor causes, the table highlights the most diagnostically relevant entities for radiologists, emphasizing imaging modalities, high-yield findings, and diagnostic clues that assist in clinical differentiation. This serves as a practical reference for both trainee education and real-world diagnostic decisions (Table *4*).

Tremor Type	Etiology	Key Imaging Modality	Representative Finding	Diagnostic Clue
Parkinson's Disease (PD)	Neurodegenerative	DaTscan, SWI MRI	Asymmetric putaminal uptake loss; loss of swallow tail sign	Resting tremors, bradykinesia, response to levodopa
Essential Tremor (ET)	Idiopathic/Genetic	DaTscan (for exclusion)	Normal symmetric striatal uptake	Bilateral action tremor, improved with alcohol
Multiple System Atrophy-Cerebellar Type (MSA-C)	Neurodegenerative	MRI (T2/FLAIR)	Hot cross bun sign; MCP atrophy	Cerebellar ataxia with autonomic dysfunction
Progressive Supranuclear Palsy (PSP)	Neurodegenerative	MRI (Sagittal T1), DaTscan	Midbrain atrophy (hummingbird sign)	Early falls, vertical gaze palsy, axial rigidity
Fragile X-Associated Tremor/Ataxia Syndrome (FXTAS)	Genetic (FMR1 premutation)	MRI (FLAIR)	Middle cerebellar peduncle T2 hyperintensity	Older men with tremor, ataxia, cognitive decline
Dystonic Tremor	Secondary (lesional or idiopathic)	MRI (T2-weighted)	Thalamic lesion or basal ganglia injury	Focal or irregular tremors with dystonic posture
Cerebellar Infarct	Vascular	MRI (T2-weighted)	Cerebellar hemisphere atrophy or gliosis	Delayed tremor onset post-stroke
Wilson’s Disease	Metabolic/Genetic	MRI (T2-weighted)	Basal ganglia or midbrain T2 hyperintensity	Young onset with hepatic symptoms
Holmes Tremor	Structural (midbrain/thalamic lesions)	MRI (T2-weighted)	Lesion near red nucleus, thalamus, or midbrain	Mixed tremor with delayed onset after brainstem lesion
Drug-Induced Tremor	Pharmacologic	CT/MRI (usually normal)	Normal or subtle white matter changes	History of medication use; tremor resolves with withdrawal
Functional Tremor	Functional (non-organic)	MRI/CT (normal)	Normal imaging; variable tremor pattern	Inconsistency, entrainment, normal imaging
Spinocerebellar Ataxia (SCA)	Genetic (Autosomal Dominant)	MRI (T2-weighted)	Cerebellar and MCP atrophy	Family history, slowly progressive ataxia

TABLE 4: Summary Table of Tremor Syndromes

Summary of representative tremor syndromes, etiologies, key imaging modalities, representative imaging findings, and diagnostic clues [2-38].

Abbreviations: DaTscan, dopamine transporter scan; MRI, magnetic resonance imaging; CT, computed tomography; PSP, progressive supranuclear palsy; MSA, multiple system atrophy; MCP, middle cerebellar peduncle.

Management and post-treatment imaging

Management of tremor syndromes requires a comprehensive understanding of their underlying causes, clinical severity, and how they respond to treatment. While medication is the primary treatment for most patients, a subgroup - especially those with medically refractory tremor - might benefit from interventional procedures such as DBS and MRgFUS. These advanced treatments have specific imaging features that radiologists need to recognize during both planning and follow-up.

DBS

DBS involves stereotactic implantation of electrodes into motor control nuclei and is used for tremor syndromes refractory to medical therapy. Common targets include the subthalamic nucleus (STN), typically for Parkinson’s disease; the ventral intermediate nucleus (VIM) of the thalamus, for essential tremor; and the globus pallidus internus (GPi), for dystonia or Parkinson’s disease [6,40-42].

Postoperative imaging (CT or MRI) confirms electrode position (Figure *12*), excludes hemorrhage, and helps plan neuromodulation settings. Imaging artifacts are common but manageable with proper protocols. Imaging also helps in localizing the target and guidance during the procedure [6,40-42].

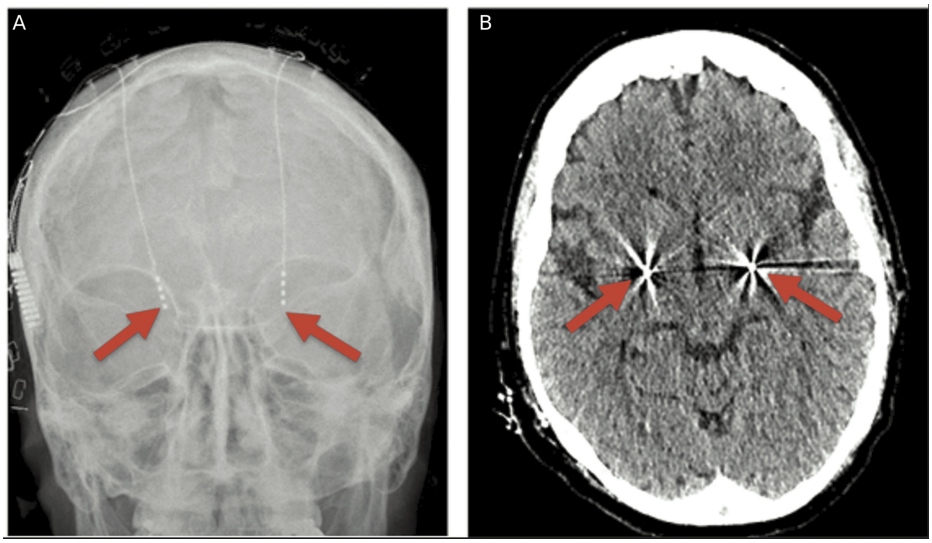


FIGURE 12: Deep Brain Stimulation Electrode Placement

(A) Frontal skull X-ray and (B) axial CT image demonstrating bilateral deep brain stimulation (DBS) electrode placement targeting the subthalamic nuclei (red arrows) in a patient with Parkinson's disease.

MRgFUS

MRgFUS is an incisionless ablative treatment for selected patients with essential tremor and Parkinson's disease, with indications varying by regulatory approval and clinical setting. It utilizes real-time MRI guidance and thermal feedback to ablate the ventral intermediate nucleus (VIM) in the thalamus, often unilaterally. Benefits include no surgical incision, shorter recovery time, and immediate tremor improvement. MRI confirms treatment location and assesses for perilesional edema or unexpected effects. Lesions appear T2 hyperintense acutely and T1 hypointense chronically (Figure *13*) [7,43,44].



FIGURE 13: MRgFUS Treatment for Essential Tremor

Axial T2-weighted MRI shows focal hyperintensity in the left thalamus (white arrow), consistent with MRgFUS-induced thermal ablation for essential tremor.

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Post-Treatment Imaging Surveillance

Radiologists play a key role in verifying DBS lead location and identifying complications (e.g., hemorrhage, pneumocephalus, peri-lead edema, electrode displacement, or infection), confirming MRgFUS lesion location and monitoring its evolution over time, and recognizing expected imaging artifacts from neuromodulation devices and adjusting protocols accordingly [42-44].

These improved treatment techniques underline the expanding significance of imaging in tremor management - not just in finding diagnostic patterns but also in validating therapy effects and directing longitudinal care. As therapies like DBS and MRgFUS become more commonly employed, radiologists must be conversant with both expected post-procedural appearances and potential problems. A good comprehension of these imaging data promotes more informed interdisciplinary conversations and supports better patient outcomes.

Artificial intelligence and ChatGPT in tremor imaging

Artificial intelligence (AI), particularly large language models (LLMs) such as ChatGPT, represents an emerging tool that may complement the clinical and imaging evaluation of tremor disorders. Sorrentino et al. specifically evaluated ChatGPT-3.5 in essential tremor by assessing its responses to 10 frequently asked questions among movement-disorder professionals and laypersons. Although the responses were generally considered clear, relevant, comprehensive, and useful, agreement among professional evaluators was only moderate, highlighting variability in the reliability of LLM-generated medical information [45]. In radiology, Suthar et al. evaluated GPT-4 using 140 neuroradiology cases from the American Journal of Neuroradiology "Case of the Month" and reported an overall diagnostic accuracy of 57.86%, demonstrating both the potential utility and limitations of LLMs in diagnostic reasoning [46]. More recent work by Lotfian et al., including Suthar, evaluated AI performance when clinical information was provided incrementally in challenging neuroradiology cases, highlighting the importance of the amount and sequence of information available to an AI model during diagnostic reasoning [47]. Similarly, Abdul Sami et al., including Suthar, compared GPT-4 Turbo with LLaMA 3.1 on 79 text-based pediatric radiology questions and found overall accuracies of 88.6% and 77.2%, respectively; however, the study excluded image-based questions, limiting direct extrapolation to radiologic image interpretation [48]. These findings are relevant to tremor imaging because diagnostic classification frequently depends on integration of clinical phenomenology with subtle and sometimes nonspecific imaging findings. Future multimodal AI systems may potentially integrate clinical features with structural MRI, functional imaging, nuclear medicine studies, and quantitative imaging biomarkers to improve tremor subtype classification and treatment-response prediction. Nevertheless, important limitations include hallucination, dependence on prompt formulation and the information provided, model-to-model variability, limited transparency, and potential biases related to training datasets and patient populations [49]. AI models may also demonstrate reduced performance when applied to populations, imaging protocols, or clinical presentations that are underrepresented in their training data. Accordingly, ChatGPT and other LLMs should currently be considered adjunctive tools rather than replacements for expert neurologic and radiologic assessment. Future tremor-specific studies should incorporate standardized multimodal datasets, external validation, subgroup-specific performance assessment, and prospective evaluation of clinical utility and patient safety.

Limitations and future directions

While this graphical analysis offers a practical, imaging-focused overview of tremor disorders, certain limitations should be noted. First, the case-based approach, although illustrative, does not represent an exhaustive or statistically validated sample of tremor presentations. Many diagnoses were hypothesized based on clinical context and imaging features but lack histopathologic confirmation. Additionally, the cases emphasize those with distinct imaging correlates and therefore underrepresent functional tremors and other disorders where imaging appears normal or is non-contributory.

Additionally, the value of nuclear medicine techniques (e.g., DaTscan, FDG PET) is discussed selectively, reflecting their restricted availability in routine practice and diverse institutional experience. The review also does not include pediatric tremor etiologies, which may present atypical imaging findings and necessitate a different diagnostic strategy.

Looking ahead, advancements in functional imaging, machine learning, and radiomic analysis hold promise for increasing the use of imaging in tremor characterization. Integration of quantifiable biomarkers, standardized imaging techniques, and clinical decision support technologies may enable earlier detection, improved subtype classification, and better prediction of therapy response.

Conclusions

Tremor syndromes encompass a wide and often overlapping spectrum of clinical entities, with imaging findings that may support diagnosis and management, although conventional neuroimaging may be normal or nonspecific in many cases. This case-based pictorial review provides radiologists and trainees with a structured, real-world framework to recognize key imaging findings across tremor subtypes - including degenerative, vascular, metabolic, and post-interventional causes. By integrating neuroimaging examples with clinical and diagnostic context, the review emphasizes high-yield pearls and pitfalls relevant to everyday practice.

Importantly, this resource functions not only as a diagnostic tool but also as an educational aid, strengthening pattern recognition and building confidence in evaluating complex movement disorders. As the use of advanced therapies like DBS and MRgFUS continues to grow, radiologists must be ready to accurately assess pre- and post-treatment imaging. Ultimately, radiologists play a key role in the multidisciplinary care of tremor patients, and a strong understanding of tremor-related imaging greatly improves that contribution. Nevertheless, imaging findings should always be interpreted alongside clinical assessment, as they may be normal or nonspecific, particularly in essential and functional tremor.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the

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