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Case Report

Oncology Section

Aromatase Inhibitor induced Carpal Tunnel Syndrome: A Rare Case of Under-reported Side Effect in Indian Population

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ABSTRACT

Carpal Tunnel Syndrome (CTS) has been increasingly identified in hormone receptor-positive Breast Cancer (BC) patients treated with Aromatase Inhibitors (AIs). While CTS is common in the general population, its association with AIs is underexplored. Recognition is important as such adverse events may compromise adherence to life-prolonging therapy. We report a case of a hormone receptor-positive BC 30-year-old married female patient who developed symptomatic CTS during AI therapy. The patient presented with classic symptoms of numbness, tingling, and pain in the hand, confirmed through clinical evaluation and diagnostic testing. The temporal relationship between AI initiation and symptom onset suggested a drug-related cause. Although AI-induced CTS has been described globally, reports remain rare in India. This case highlights the need for heightened clinical awareness, timely recognition, and management of CTS in patients receiving AIs to prevent treatment discontinuation and ensure optimal therapeutic outcomes.

Keywords: Breast neoplasms, Gonadotropin-releasing hormone analogues, Letrozole, Musculoskeletal diseases, Tamoxifen

CASE REPORT

A 30-year-old married woman with no comorbidities, mother of two (last childbirth 6 years prior), was diagnosed with left BC approximately 14 months before the onset of her current wrist and finger symptoms. Clinico-radiological staging, including Fluorodeoxyglucose Positron Emission Tomography–Computed Tomography (FDG PET-CT), showed cT4bN2M0 disease. Trucut biopsy revealed Invasive Carcinoma, Nothing Other Specified, with strong Oestrogen Receptor (ER) and Progesterone Receptor (PR) positivity, Her2neu 3+, and Ki-67 of 30%. The patient received dose-dense neoadjuvant chemotherapy (TCHP regimen), which was completed approximately six months after diagnosis, with a complete clinical response. One month later, she underwent Modified Radical Mastectomy (MRM); histopathology revealed a pathological complete response with all lymph nodes negative. Two months post-surgery, adjuvant radiotherapy (45 Gy in 20 fractions) was completed. Hormonal therapy with a monthly injection. Leuprolide 3.75 mg (continued for ~11 months) and Tab Letrozole 2.5 mg were initiated the same month. Bone health was optimised with calcium and zoledronate therapy; DEXA revealed mild osteopenia (T-score −1.1 at the right femur).

The patient remained asymptomatic for approximately 14 months after initiation of AI therapy, after which she developed wrist and finger pain with tingling for one month. The presentation was atypical for classical CTS, with no muscle wasting, objective neurological deficit, or weakness on examination. Although motor weakness and thenar wasting are recognised features of advanced or long-standing CTS, sensory symptoms such as pain, tingling, and numbness in the median nerve distribution are often the earliest and most prominent complaints in typical CTS, even in the absence of motor signs, and clinical tests alone may not reliably confirm the diagnosis. Standardised pain scales and grip strength measurements were not formally recorded; functional impact was assessed clinically, with pain-related limitation of wrist and finger movements, but no impairment of daily activities. There was no history of repetitive hand use, prior wrist trauma, occupational strain, or other recognised CTS risk factors. Given the overlap with AI-associated musculoskeletal symptoms, further evaluation was undertaken. Baseline laboratory investigations were within normal limits. Additional metabolic and endocrine evaluation included: Thyroid function {Thyroid Stimulating Hormone (TSH) 6.218 µIU/mL}, Vitamin B12 (within normal limits), diabetes screening {estimated average glucose 93.93 mg/dL, Gylcosylated Haemoglobin (HbA1c) 4.90%}, and lipid profile {triglycerides 157 mg/dL, total cholesterol 105 mg/dL, High Density Lipoprotein (HDL) 39 mg/dL, Low Density Lipoprotein (LDL) 58 mg/dL, Very Low Density Lipoprotein (VLDL) 31.40 mg/dL}. A provisional diagnosis of Letrozole-induced CTS was made, and the patient was referred to a neurologist for definitive evaluation. Nerve Conduction Velocity (NCV) showed bilateral median nerve prolonged distal latency with normal Compound Muscle Action Potential (CMAP) and conduction velocity, suggestive of bilateral CTS (R>L) [Table/Fig-1,2a-d].

Key findings from the nerve conduction study demonstrate prolonged distal latency of the bilateral median nerves with preserved CMAP amplitudes and normal conduction velocities, consistent with focal compression at the carpal tunnel. Sensory responses in the median nerves were reduced or absent, whereas ulnar, peroneal, and tibial nerve parameters were within normal limits, supporting a diagnosis of bilateral CTS (Right > Left) and excluding generalised peripheral neuropathy.

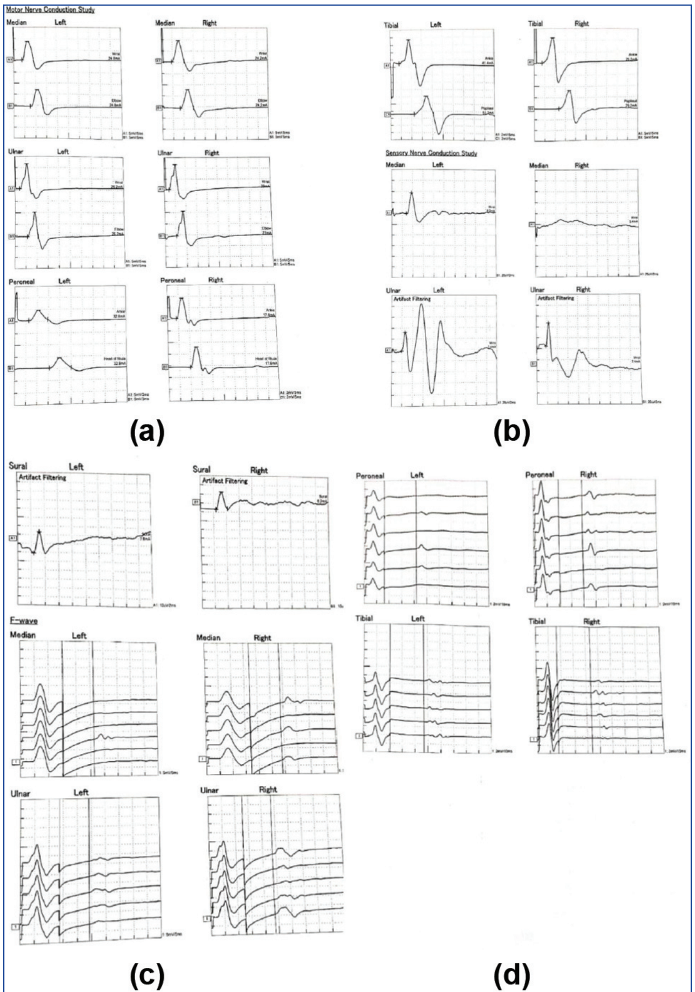
Letrozole was discontinued, and she was initiated on tapering steroids (tablet prednisolone 10 mg), along with tablet oxcarbazepine 150 mg, tablet gabapentin 100 mg, and tablet tolperisone hydrochloride 150 mg for 12 weeks. A multi-modal pharmacologic approach was employed, with corticosteroids addressing inflammation, anticonvulsants targeting neuropathic pain, and a muscle relaxant. This strategy aimed to optimise symptom control while minimising the need for surgical intervention, which is reserved for refractory or severe cases, ensuring both clinical efficacy and judicious resource utilisation. The patient improved clinically. Letrozole was replaced

Site	Lat (ms)	N.D.	Amp.	N.D.	Stim. (mA)	Segment	Dist (mm)	Intvl (ms)	NCV (m/s)	CCV (m/s)	N.D.	Temp
Median	Left					Temperature						
Wrist	4.0		8.1 mV		24.8	*Wrist		4.0				
Elbow	7.6		7.5 mV		24.8	Wrist-Elbow	180	3.6	50.0			
						Elbow-Axilla						
Median	Right					Temperature						
Wrist	4.7		9.2 mV		24.2	*Wrist		4.7				
Elbow	8.4		8.3 mV		24.2	Wrist-Elbow	185	3.7	50.0			
						Elbow-Axilla						
Ulnar	Left					Temperature						
Wrist	2.2		11.1 mV		26.2	*Wrist		2.2				
Elbow	5.6		10.8 mV		26.2	Wrist-Elbow	210	3.5	60.9			
						Elbow-Axilla						
Ulnar	Right					Temperature						
Wrist	2.2		11.5 mV		23.0	*Wrist		2.2				
Elbow	5.4		11.1 mV		23.0	Wrist-Elbow	200	3.2	63.5			
						Elbow-Axilla						
Peroneal	Left		4.3 mV		32.8	Temperature						
Ankle	3.8		4.1 mV		32.8	*Ankle		3.8				
Head of Fibula	9.3					Ankle-Head of fibula	300	5.6	53.8			
						Head of fibula-Popliteal						
Peroneal	Right					Temperature						
Ankle	4.2		3.5 mV		17.6	*Ankle		4.2				
Head of Fibula	10.0		3.4 mV		17.6	Ankle-Head of fibula	300	5.8	51.7			
						Head of fibula-Popliteal						
Tibial	Left					Temperature						
Ankle	4.2		6.5 mV		41.4	*Ankle		4.2				
Popliteal	11.4		4.9 mV		51.2	Ankle-Popliteal	380	7.2	53.1			
Tibial	Left					Temperature						
Ankle	3.8		10.8 mV		25.2	*Ankle		3.8				
Popliteal	12.0		8.2 mV		25.2	Ankle-Popliteal	355	8.2	43.6			
Site	**Lat.1 (ms)**	**N.D.**	**Amp.**	**N.D.**	**Stim. (mA)**	**Segment**	**Dist (mm)**	**Intvl (ms)**	**NCV (m/s)**	**CCV (m/s)**	**N.D.**	**Temp**
Median	Left					Temperature						
Wrist	2.8		37.4 uV		8.2	Wrist	130	2.8	46.1			
						Wrist-Elbow						
Median	Right											
Wrist	NR		NR		5.4	Temperature						
						Wrist			NR			
						Wrist-Elbow						
Ulnar	Left					Temperature						
Wrist	1.9		33.7 uV		7.4	Wrist	110	1.9	56.7			
						Wrist-Elbow						
Ulnar	Right					Temperature						
Wrist	1.8		43.2 uV		7.4	Wrist	110	1.8	61.1			
						Wrist-Elbow						
Sural	Left					Temperature						
Sural	2.4		24.3 uV		7.8	Sural	98	2.4	41.5			
Sural	Right					Temperature						
Sural	2.4		19.5 uV		8.2	Sural	130	2.4	53.7			

F-Wave							
Nerve	**Side**	**Stim Site**	**F-Lat**	**F-Lat N.D.**	**M.Lat**	**F-M Lat.**	**N.D.**
Median	Left	Wrist	26.3 ms		0.0 ms	26.3 ms	
Median	Right	Wrist	29.0 ms		0.0 ms	29.0 ms	
Ulnar	Left	Wrist	24.2 ms		0.0 ms	24.2 ms	

Ulnar	Right	Wrist	23.8 ms		0.0 ms	23.8 ms	
Peroneal	Left	Ankle	42.0 ms		0.0 ms	42.0 ms	
Peroneal	Right	Ankle	40.2 ms		0.0 ms	40.2 ms	
Tibial	Left	Ankle	47.1 ms		0.0 ms	47.1 ms	
Tibial	Right	Ankle	43.8 ms		0.0 ms	43.8 ms	

[Table/Fig-1]: Electrophysiologic parameters from Nerve Conduction Velocity (NCV) studies.
NCS: NCS of the bilateral median nerve shows prolonged distal latency, normal CMAP amplitude and conduction velocity. The right median sensory shows non-recordable SNAP amplitude. Rest of motor and sensory studies are normal; Impression:- Bilateral Moderate Carpal Tunnel Syndrome (CTS) (R>L)



[Table/Fig-2]: Nerve Conduction Study (NCV) findings. (a) Motor conduction studies showing prolonged distal latency of bilateral median nerves with preserved CMAP amplitudes and normal conduction velocities, consistent with focal median nerve compression; (b) Sensory conduction showing reduced median sensory responses relative to ulnar nerves; (c) F-wave recordings demonstrating normal proximal motor pathways; (d) Peroneal and tibial lower limb studies showing normal conduction, excluding generalised neuropathy.

with Tamoxifen, and GnRH analogue therapy was continued as part of standard adjuvant endocrine therapy, planned for a total duration of approximately five years. The patient has been on regular follow-up visits and, as of 2025, remains asymptomatic with no recurrence of CTS or other musculoskeletal complaints following the switch to Tamoxifen. As the patient is clinically well, repeat nerve conduction studies were not performed, consistent with current literature, which suggests that NCV evaluation is generally reserved for patients with persistent or progressive symptoms, functional impairment, or atypical presentation [1-3].

DISCUSSION

In 1854, Sir James Paget was the first to describe the clinical features of CTS [4,5]. Today, CTS is recognised as a common mononeuropathy in medical practice. Despite early identification, it took nearly a century to confirm the primary cause: compression of the median nerve at the wrist [4].

The carpal tunnel is a narrow passage in the palm formed by bones and ligaments. The median nerve travels through this passage from the forearm to the hand, controlling muscle movement and motor function. Compression of this nerve leads to CTS. CTS affects approximately 1-5% of the general population. Obesity doubles the risk. While rare in children, it commonly affects adults aged 40-60 years [1]. Women are three times more likely to develop CTS, especially around menopause. Additionally, conditions associated with reduced oestrogen levels-such as pregnancy, use of combined oral contraceptives, and bilateral oophorectomy- are linked to a higher risk of CTS [6]. India alone reports nearly 10 million CTS cases [7].

AIs have revolutionised BC treatment by improving survival and reducing recurrence [7]. AIs are now the standard in postmenopausal women with ER-positive BC following surgery. Recommendations include AI therapy for at least five years, either replacing tamoxifen, after 2-3 years of tamoxifen (sequential treatment), or after five years of tamoxifen (extended treatment) [8]. Tamoxifen blocks ERs while retaining some oestrogen-like activity. In contrast, AIs inhibit oestrogen production from peripheral tissues [9]. These mechanisms may partly explain differences in side-effect profiles, although the reasons are not yet fully understood. Oestrogen is thought to have analgesic effects via the brain and spinal cord and may influence tissue inflammation. The absence of oestrogen's protective effects may contribute to joint pain and stiffness in patients on AIs [2].

However, 20-60% of patients discontinue AI therapy prematurely due to side effects [10]. Adverse events mainly affect the musculoskeletal system and can be grouped into: 1) bone-related issues, including increased fracture risk; 2) AI-associated joint pain; and 3) increased incidence of autoimmune joint and connective tissue disorders [8]. The pathophysiology behind these effects likely relates to prolonged oestrogen suppression, though exact mechanisms remain unclear. One significant side effect is AI Musculoskeletal Syndrome (AIMSS), which includes joint pain, muscle stiffness, tendon disorders, and CTS. AIs also contribute to bone loss [11]. Studies show a higher incidence of CTS in AI users (1.3%) compared to those not on hormone therapy (0.4%). Women with prior hormone replacement therapy (p=0.007) or chemotherapy (p=0.01) are more prone to CTS. Notably, those aged 60 or older have a lower risk [4].

CTS results from median nerve compression, presenting as hand and finger pain, weakness, and numbness [11]. Symptoms may be vague and are often difficult for patients to articulate, making diagnosis challenging. Symptoms of CTS may overlap with chemotherapy-induced peripheral neuropathy, cervical radiculopathy, tendon disorders, and inflammatory arthropathies; therefore, electrodiagnostic testing is essential for accurate diagnosis [1,5]. Combining clinical findings with electrodiagnostic tests is the most reliable approach [3,12]. Imaging (ultrasound or MRI) is warranted only if a structural abnormality (e.g., tumour or ganglion cyst) is suspected [1]. Patients with mild CTS may not require further evaluation. However, when symptoms are atypical, impact sleep, or involve hand dysfunction, additional diagnostics such as NCV are advised [1]. There are no AI-specific pharmacologic guidelines for CTS, and management is extrapolated from standard CTS treatment recommendations, including nonsteroidal anti-inflammatory drugs, short courses of corticosteroids, and neuropathic pain medications, with surgical decompression reserved for refractory or severe cases [5,6].

We report a 30-year-old female, a treated case of left breast carcinoma, who developed symptoms consistent with moderate-to-severe CTS following AI therapy.

Study/Author	Age/ Sex	AI Used	Key Symptoms	Management/Outcome
Current case	30 F	Letrozole + GnRH analogue	Hand pain, tingling, numbness	Stop AI, medications, switch to Tamoxifen → asymptomatic
Sestak 2009 (ATAC)	63 F	Anastrozole	Hand pain, tingling	AI discontinuation/symptomatic management → most improved
Nishihori 2008	55-70 F	Anastrozole / Letrozole	CTS, functional limitation	Conservative±Surgery → improvement / refractory cases
Shin 2022	50-65 F	AI therapy	Hand pain, tenosynovitis	Nonsteroidal Anti-Inflammatory Drugs (NSAIDs), Physiotherapy → Partial to Full resolution
Chung 2022	45–68 F	AI therapy	CTS, tenosynovitis	Conservative±Surgery → 67% resolved, others surgery

[Table/Fig-3]: Comparison of key clinical features and management of Aromatase Inhibitor (AI)–associated Carpal Tunnel Syndrome (CTS) across published reports and the current case. This table summarises the clinical characteristics, AI agents used, presenting symptoms, and management outcomes of previously reported cases and series of AI-associated CTS and tenosynovitis. The current case is included for comparison. Age and sex of patients, type of AI therapy, key symptoms, and treatment approaches (including conservative, pharmacologic, and surgical strategies) are presented to illustrate variability and similarities in presentation and response to management.

AIs are known to exacerbate joint and muscle-related issues [13]. These adverse effects can impair quality of life and hinder treatment adherence [14]. CTS may represent a localised manifestation of AI-induced musculoskeletal toxicity, possibly due to oestrogen withdrawal affecting wrist connective tissues. Anastrozole is more commonly associated with CTS, but Letrozole-induced CTS- though rare- is increasingly recognised [15]. In our case, after 14 months of AI therapy, the patient developed severe pain and tingling in the right wrist, index, and middle fingers. Clinical examination showed mildly restricted finger movements, likely due to pain, without muscle wasting, tenderness, or neurological deficit. Symptoms were evaluated with routine baseline labs, which were normal. NCV testing- considered the gold standard for CTS diagnosis- was performed [16]. It revealed bilateral median nerve distal latency prolongation with normal CMAP amplitude and conduction velocity, consistent with bilateral CTS (right > left) [Table/Fig-2a-d].

CTS in this patient was provisionally attributed to Letrozole based on clinical presentation, timing, and literature evidence [17]. AI-associated CTS is believed to arise from low oestrogen levels that affect cartilage integrity and immune response. Oestrogen replacement has been shown to improve joint symptoms [8].

Management of AI-induced CTS involves both conservative and pharmacologic approaches. In this case, Letrozole was stopped, and the patient was treated with tapering doses of Tab prednisolone 10 mg, along with Oxcarbazepine 150 mg, Gabapentin 100 mg tablet, and Tolperisone Hydrochloride 150 mg tablet for 12 weeks. The patient showed marked clinical improvement. Letrozole was replaced with tamoxifen, and Gonadotropin-Releasing Hormone (GnRH) analogues were continued.

Studies suggest that up to 25% of women discontinue AIs within the first year, with musculoskeletal symptoms being a major factor [6,18]. Literature supports that switching from AIs to tamoxifen relieves CTS symptoms in some patients [19]. Management of AI-induced CTS includes both nonsurgical and surgical strategies, depending on symptom severity and response to initial therapy. Conservative measures such as analgesics, corticosteroids, neuropathic pain medications, and modification or discontinuation of AI therapy have been shown to result in complete symptom resolution in up to 67% of cases. However, in patients with persistent or severe symptoms, surgical decompression remains the standard of care. A systematic review evaluating 19 studies identified a significantly higher incidence of CTS and stenosing tenosynovitis in patients receiving AIs compared to tamoxifen, with some studies reporting up to a tenfold increase in risk. Although the quality of evidence was variable, all studies evaluating surgical intervention reported symptom resolution following carpal tunnel release. Therefore, early recognition and appropriate escalation to surgical management are essential in refractory cases to prevent prolonged morbidity and treatment discontinuation [20].

To provide an overview of previously reported AI-associated CTS cases and compare them with our patient, we have summarised key clinical features, management strategies, and outcomes in [Table/Fig-3].

In line with European Society for Medical Oncology (ESMO) and American Society of Clinical Oncology (ASCO) guidance, routine screening for CTS is not recommended in asymptomatic patients receiving AIs [21]. Evaluation should be symptom-driven, with electrodiagnostic testing reserved for persistent or function-limiting complaints [3]. There is currently no evidence to support routine pharmacologic prophylaxis for AI-associated musculoskeletal toxicity; therefore, preventive strategies focus on patient education, ergonomic measures, and early symptom reporting. Patients should be counseled regarding potential musculoskeletal adverse effects of AIs to support adherence and shared decision-making. In patients with moderate-to-severe or refractory symptoms, switching endocrine therapy to tamoxifen may be considered after multidisciplinary assessment, balancing oncologic efficacy with quality-of-life considerations [22].

CONCLUSION(S)

This case highlights the importance of recognising CTS as a potential adverse effect of AI therapy in patients with BC. Despite an increasing incidence, the condition remains underdiagnosed, particularly in the Indian setting, where available literature is largely limited to population and occupation-based prevalence studies, with very few reports addressing AI-associated CTS. The pathogenesis of AI-induced CTS is not yet fully understood, and clinical presentation may overlap with other treatment-related neuromuscular conditions, necessitating careful evaluation. Further research from Indian populations is required to better define risk factors, underlying mechanisms, and evidence-based management strategies for CTS in BC patients receiving hormonal therapy.

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