Journal of Chemical Health Risks

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JCHR (2025) 15(6), 2755-2763 | ISSN:2251-6727

Ascertaining the Role of Acetyl-L-Carnitine in Carpal Tunnel Syndrome – A Randomised Controlled Trial

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(Received: 25 October 2025 Revised: 27 November 2025 Accepted: 16 December 2025)

KEYWORDS

Carpal tunnel syndrome, Acetyl-L-carnitine, Randomized controlled trial, Neuropathic pain, Nerve conduction, Boston Carpal Tunnel Questionnaire

ABSTRACT:

Background: Carpal tunnel syndrome is the most common entrapment neuropathy with variable responses to conservative therapy, and Acetyl-L-Carnitine's putative neuroprotective and analgesic actions warrant evaluation as an adjunctive treatment.

Objectives: To determine the efficacy of Acetyl-L-Carnitine on neuroprotection, neuropathic symptoms, and hand function in carpal tunnel syndrome; and to study recurrence of symptoms in CTS patients on standard treatment.

Methods: We conducted a single-centre, prospective, open-label randomized trial at a neurology outpatient clinic, enrolling adults (18–65 years) with idiopathic CTS (N=80) and allocating them to standard care alone or Acetyl-L-Carnitine 500 mg three times daily for 12 weeks plus standard care. Outcomes (BCTQ-SSS/FSS, DN4) were assessed at baseline, 4, 12, and 20 weeks; median-nerve NCS were performed at baseline and 12 weeks.

Results: Both groups were similar at baseline (age 45.5 vs 47.3 years; p=0.210; female-predominant). Over 12 weeks, electrophysiology improved in both arms but more with ALCAR, notably in sensory metrics: sensory nerve conduction velocity rose 41.12→44.49 m/s (Δ+3.37) vs 40.12→41.78 m/s (Δ+1.66), and sensory distal latency fell 3.77→3.26 ms vs 3.68→3.29 ms; distal motor latency decreased more with ALCAR (−5.84% vs −4.04%; p=0.012). The best discriminator was % increase in sensory conduction velocity (AUC 0.775, 95% CI 0.675–0.875). Patient-reported outcomes favored ALCAR from 4 weeks onward: BCTQ-SSS 20.02 vs 26.00 ($p<0.001$) at 4 weeks and 16.20 vs 23.63 ($p<0.001$) at 12 weeks; BCTQ-FSS and DN4 showed parallel advantages, persisting to 20 weeks. Adverse events were mostly nausea (40%) and vomiting (40%); weight gain and hair fall occurred in 10% each.

Conclusion: Adjunctive Acetyl-L-Carnitine (500 mg three times daily for 12 weeks) significantly improved neuropathic pain, hand function, and sensory nerve conduction in mild–moderate carpal tunnel syndrome versus standard care, with partial symptom return after discontinuation.

Introduction

Carpal tunnel syndrome (CTS) is the most common entrapment neuropathy, imposing substantial functional limitations and productivity loss worldwide. Population studies estimate annual incidence between 0.5 and 5.1 per 1,000 person-years, with community cohorts reporting 3.5 per 1,000 and higher rates in women, peaking in the fourth to sixth decades.(1, 2)

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Occupational exposures (repetitive wrist flexion/extension, forceful grip, and hand-arm vibration) and systemic conditions—including diabetes, obesity, hypothyroidism, pregnancy, menopause, rheumatoid arthritis, chronic kidney disease, and heart failure—are consistently associated with increased risk.(3)

Clinically, CTS manifests with paresthesia, pain, and numbness in the median-nerve distribution, often nocturnally or with provocation, and may progress to weakness, thenar eminence atrophy, and impaired hand dexterity; bilateral involvement is frequent, with dominant-hand predominance.(4) Although electrodiagnostic testing, ultrasonography, and magnetic resonance imaging can corroborate the diagnosis or reveal alternative pathology, contemporary reviews emphasize that a careful history and examination remain central, particularly in typical presentations.(5) High-quality imaging studies additionally support the utility of median-nerve cross-sectional area on ultrasonography as an adjunctive marker.(6, 7)

Management is tailored to clinical severity, electrophysiologic status, and patient preference. Nonpharmacologic options include neutral-position wrist splinting and structured exercises (nerve/tendon gliding), which have demonstrated short-term symptom relief and modest electrophysiologic gains in mild disease, though effect sizes vary across trials.(8, 9) Pharmacologic therapies most commonly involve corticosteroids; randomized and systematic evidence shows that local steroid injection provides greater short-term improvement than placebo and, at up to three months, greater benefit than oral steroids, while oral prednisone regimens also yield symptomatic improvement versus placebo.(10, 11) Longer-term comparisons suggest injections may delay—but not necessarily obviate—surgery, and the overall durability of nonsurgical effects remains uncertain.(12) NSAIDs are frequently used for pain, yet high-certainty evidence for disease-modifying effects is limited.(13)

Acetyl-L-carnitine (ALCAR), a mitochondrial cofactor with putative neurotrophic and analgesic actions, has shown pain reduction and electrophysiologic improvements in peripheral neuropathies in meta-analyses, supporting biological plausibility for benefit in entrapment neuropathies.(14, 15) Specific to CTS, a randomized, placebo-controlled protocol has proposed ALCAR as an adjunct to enhance nerve regeneration, reflecting ongoing interest but also highlighting limited definitive trial data to date.(16) Moreover, conflicting findings in other neuropathic contexts (e.g., chemotherapy-induced peripheral neuropathy) underscore the need for rigorous evaluation of efficacy and safety in CTS before routine adoption.(17)

Against this background, the objective of the present study was to determine the efficacy of Acetyl-L-Carnitine on neuroprotection, neuropathic symptoms, and hand function in carpal tunnel syndrome; and to study recurrence of symptoms in CTS patients on standard treatment.

Materials and Methods

This prospective, open-label, randomized interventional study was conducted in the Out-patient Department of Neurology, Kilpauk Medical College, Chennai, from May 2018 to April 2019 after prior approval from the Institutional Ethics Committee. Adults aged 18–65 years with a clinical diagnosis of idiopathic carpal tunnel syndrome corroborated by nerve conduction studies (NCS) were screened. Exclusion criteria comprised severe CTS warranting surgery at presentation, co-existing peripheral neuropathies, orthopaedic disorders affecting the wrist, uncontrolled diabetes mellitus, hypothyroidism, chronic kidney disease, pregnancy, and seizure disorder. Potentially eligible patients received a brief study explanation, and those willing to participate provided written informed consent.

A priori sample-size estimation indicated 74 participants (37 per group); to accommodate potential attrition, a total sample of 80 was targeted with 40 participants allocated to each arm. After recording detailed history, general and systemic examinations, vital signs, and routine laboratory investigations (including serum thyroid-stimulating hormone), participants were randomized by a computer-generated sequence to either standard treatment alone (control group) or Acetyl-L-Carnitine (ALCAR) plus standard treatment (intervention group). Standard treatment followed routine conservative care for CTS and included neutral-position wrist splinting, physiotherapy with tendon-gliding exercises, and non-steroidal anti-inflammatory drugs as needed. The intervention group

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JCHR (2025) 15(6), 2755-2763 | ISSN:2251-6727

received ALCAR 500 mg orally three times daily for 12 weeks in addition to standard care. Adverse events were actively solicited and documented throughout follow-up, and routine blood investigations were repeated at 3 months to monitor safety.

Outcome assessments were performed at baseline and at weeks 4, 12, and 20 (the latter corresponding to 8 weeks after stopping ALCAR). Symptom severity and functional status were measured using the Boston Carpal Tunnel Questionnaire (BCTQ, Symptom Severity Scale and Functional Status Scale), which is a validated disease-specific instrument for CTS. Neuropathic pain features were evaluated using the Douleur Neuropathique en 4 questions (DN4), a validated screening tool for neuropathic pain. Nerve conduction studies of the median nerve were performed at screening and repeated at week 12 in accordance with recommendations of the International Federation of Clinical Neurophysiology, recording distal latency, amplitude, and conduction velocity for sensory nerve action potentials (SNAP) and compound muscle action potentials (CMAP).

Statistical analysis was carried out using SPSS version 20.0. Efficacy analyses followed a per-protocol approach. Continuous variables from NCS, BCTQ (symptom severity and functional status), and DN4 were summarized as mean ± standard deviation. Within-group changes were analyzed with paired t-tests, and between-group comparisons with unpaired t-tests. Repeated-measures analysis of variance (ANOVA) was additionally applied to evaluate trajectories over time and group–time interactions. A two-sided p value <0.05 was considered statistically significant.

Results

The two groups were demographically comparable; mean age was 45.5 years in controls and 47.3 years in the ALCAR arm (p=0.210), with female predominance in both (sex ratio male:female 1:3 vs 1:2). Across 3 months, nerve-conduction trends favoured improvement in both groups, with numerically larger gains in several sensory parameters with ALCAR. Median distal motor latency fell from 3.85→3.64 ms with ALCAR versus 4.22→4.06 ms in controls, while sensory distal latency declined 3.77→3.26 ms vs 3.68→3.29 ms. Sensory nerve conduction velocity increased more with ALCAR (41.12→44.49 m/s; Δ+3.37) than controls (40.12→41.78 m/s; Δ+1.66). Motor conduction velocity rose similarly (ALCAR 50.18→52.10 m/s; controls 49.76→51.76 m/s). Amplitudes improved modestly in both groups (e.g., SAMP 24.48→27.29 μV with ALCAR; 25.97→28.18 μV in controls; MAMP 10.7→11.18 mV vs 10.18→10.78 mV), indicating overall electrophysiologic improvement, particularly in sensory velocity and latencies in the ALCAR group.

At 12 weeks, ALCAR produced a significantly greater reduction in distal motor latency than control (mean % decrease 5.84±3.96 vs 4.04±2.40; p=0.012) and a markedly larger gain in sensory conduction velocity (mean % increase 8.34±4.25 vs 4.28±3.22; p<0.001). Other changes were numerically similar between groups: motor amplitude rose 5.23±4.19% with ALCAR vs 6.64±4.36% in controls (p=0.125), motor conduction velocity 3.89±2.67% vs 4.15±2.43% (p=0.636), sensory distal latency fell 13.93±7.28% vs 12.11±11.41% (p=0.375), and sensory amplitude increased 13.04±10.49% vs 11.38±9.68% (p=0.446). ROC analysis identified % increase in sensory nerve conduction velocity as the best discriminator (AUC 0.775, SE 0.051, 95% CI 0.675–0.875), while other parameters showed modest to poor discrimination: % decrease MDL AUC 0.629, % decrease SDL 0.603, % increase MAMP 0.599, % increase SAMP 0.554, and % increase MNCV 0.537.

Across patient-reported outcomes, ALCAR outperformed control from 4 weeks onward with benefits sustained to 20 weeks. BCTQ-SSS fell more with ALCAR than controls at 4 weeks (20.02±2.68 vs 26.00±1.89; p<0.001), 12 weeks (16.20±2.16 vs 23.63±1.90; p<0.001), and 20 weeks (17.09±1.81 vs 21.40±3.05; p<0.001), after similar baselines (26.88±2.38 vs 28.63±1.80; p=0.410). Functional scores (BCTQ-FSS) showed parallel advantages at 4 weeks (15.36±2.01 vs 16.63±1.52; p=0.001), 12 weeks (13.65±2.14 vs 15.68±1.52; p<0.001), and 20 weeks (13.95±2.03 vs 15.14±2.04; p=0.040). Neuropathic pain (DN4) was comparable at baseline (5.47±0.84 vs 5.34±0.80; p=0.240) and 4 weeks (4.04±0.96 vs 4.25±1.08; p=0.148), but favored ALCAR at 12 weeks (2.09±0.67 vs 3.02±0.92; p<0.001) and 20 weeks (2.50±0.69 vs 3.02±0.73; p=0.001), indicating a sustained—though slightly attenuated—analgesic effect

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after treatment cessation. Adverse events in the ALCAR group were predominantly nausea (40%) and vomiting (40%), with weight gain and hair fall reported in 10% each.

Discussion

In this randomized study, Acetyl-L-Carnitine (ALCAR) demonstrated clinically meaningful benefits in patients with mild to moderate carpal tunnel syndrome, complementing a body of literature that has explored its role across neuropathic conditions. Prior animal and human investigations have evaluated ALCAR in non-compressive neuropathies—most prominently chemotherapy-induced peripheral neuropathy (CIPN) and diabetic neuropathy—with several reports of analgesic and putative neurotrophic effects, while findings in compressive neuropathies have been mixed.(18, 19) Against this backdrop, our results support ALCAR as a useful adjunct in earlier CTS, in whom reversible pathophysiology—particularly demyelination—predominates.

Outcome domains in earlier ALCAR trials have typically clustered around two constructs; neuropathic pain and nerve regeneration. Symptom domains have been captured using validated patient-reported measures such as the Boston Carpal Tunnel Questionnaire (BCTQ) and DN4, whereas neuroregeneration has been inferred indirectly from electrodiagnostic parameters or, less commonly, assessed directly via tissue measures (e.g., nerve biopsy) or proxy markers (e.g., soleus weight, intraepidermal nerve fiber density on skin biopsy).(20-23) Our trial adhered to this paradigm, employing serial nerve conduction studies (NCS) together with BCTQ and DN4 to quantify both physiologic and symptomatic trajectories. With respect to nerve regeneration surrogates, we observed significant improvements in sensory nerve conduction velocity (SNCV) and a reduction in distal latency of the compound muscle action potential (CMAP) in the ALCAR arm relative to controls at 12 weeks. Because SNCV and CMAP distal latency are sensitive to demyelinating change, these shifts point toward amelioration of segmental demyelination in median-nerve fibers. Notably, sensory parameters improved more than motor parameters. This gradient is biologically plausible: sensory abnormalities typically manifest earlier in CTS, and demyelination is an early, potentially reversible lesion, whereas motor involvement and axonal loss tend to accrue later. These observations therefore suggest that ALCAR's benefits are most pronounced when administered during the earlier, demyelination-dominant phase of disease.

Our electrophysiological findings accord with a multicenter, randomized, examiner-blinded study in CTS by Cruccu and colleagues,(22) who reported significant gains in SNCV ($P<0.001$) and broad improvements in sensory neurophysiology with ALCAR 500 mg twice daily over four months, while CMAP indices improved less than sensory measures. By contrast, Curran et al.(16) evaluated high-dose ALCAR (3,000 mg/day) after carpal tunnel release in adults with severe CTS and found no enhancement in nerve regeneration or functional severity over two months—an outcome that likely reflects the advanced axonal pathology and postoperative milieu in that cohort, where the window for demyelination-focused recovery may have passed.(22) Together, these comparator data reinforce a stage-dependent therapeutic signal: ALCAR appears more effective in mild to moderate CTS than in severe disease.

The patient-reported outcomes in our study mirror the physiological improvements. Using repeated-measures ANOVA, ALCAR produced statistically significant and clinically relevant reductions in BCTQ Symptom Severity and Functional Status scores and in DN4 scores from baseline to one month, with further favourable changes through three months. Although partial attenuation was seen by five months—eight weeks after stopping ALCAR—the symptom and function scores in the ALCAR group remained superior to both their own earlier time points and to the control group, indicating durability beyond the active treatment period. These results are broadly consistent with the study by Cruccu et al.,(22) who observed a 39% reduction in BCTQ symptom severity (from 19.3 [95% CI 17.6–21.1] to 11.8 [95% CI 10.3–13.2]) and an 18% reduction in functional severity (from 15.2 [95% CI 13.9–16.5] to 12.5 [95% CI 11.5–13.5]) at four months ($P<0.0001$ for both), with no further decrement between months three and four on post-hoc testing. The

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JCHR (2025) 15(6), 2755-2763 | ISSN:2251-6727

convergence of symptomatic and electrophysiologic benefits in our trial strengthens the inference that ALCAR exerts both antinociceptive and myelin-supportive effects in early CTS.

Taken together, the present findings add granularity to a heterogeneous evidence base. They indicate that, in mild to moderate CTS, ALCAR can improve sensory conduction, reduce symptom severity, and enhance hand function, with sensory fibers showing the earliest and largest gains—an effect pattern aligned with the disease's natural history and with prior randomized data.(24) Conversely, null findings in severe CTS after surgery caution against extrapolation to advanced or post-decompression contexts. Given prior signals in CIPN and diabetic neuropathy and supportive surrogate and histological observations from experimental work, our results help position ALCAR as a rational adjunct to conservative care in appropriately selected CTS patients, while underscoring the importance of disease staging and timing of intervention in determining therapeutic yield.

This study has several limitations. It was a single-centre, open-label trial with a modest sample (N=80), which limits external validity and introduces potential performance and reporting bias for patient-reported outcomes (BCTQ, DN4) in the absence of blinding or a placebo/sham control. Follow-up was relatively short (20 weeks, only 8 weeks post-cessation), precluding assessment of long-term durability, time to surgery, or relapse beyond two months off therapy. Electrophysiology was performed only at baseline and 12 weeks, so interim and longer-term neurophysiologic trajectories were not captured; imaging (e.g., median-nerve CSA on ultrasonography) and quantitative sensory testing were not incorporated. The intervention was given as an adjunct to heterogeneous "standard care" (splinting/physiotherapy/PRN NSAIDs), which may confound effect attribution; adherence to both ALCAR and exercises was not objectively monitored. Exclusion of severe CTS reduces generalizability to advanced disease, and the study was not powered for safety endpoints or subgroup analyses.

Conclusion

In conclusion, Acetyl-L-Carnitine (500 mg three times daily) significantly reduced neuropathic pain, improved hand function, and produced favorable electrophysiological changes consistent with nerve regeneration in patients with mild to moderate carpal tunnel syndrome. Partial symptom recurrence and minor functional decline were observed after discontinuation, suggesting benefits are greatest during active therapy and early disease. Larger, blinded trials with longer follow-up are warranted to define optimal dosing, durability of effect, and long-term safety and efficacy.

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JCHR (2025) 15(6), 2755-2763 | ISSN:2251-6727

Table 1: Demographic comparison of control group and ALCAR group

	Controls	**ALCAR**	**p value**
Age (years)	45.53	47.31	0.210
Sex (male, female ratio)	1:3	1:2	

Table 2: Nerve conduction analysis in the control group and ALCAR group at baseline and at the end of 3rd month

NCS Parameters	**NCS, baseline Mean±SD**		**NCS, end of 3rd month Mean±SD**	
	Controls	**ALCAR**	**Controls**	**ALCAR**
MDL	4.22±1.15	3.85±.92	4.06±1.15	3.64±0.94
MAMP	10.18±4.43	10.7±3.62	10.78±4.54	11.18±3.69
MNCV	49.76±7.93	50.18±6.18	51.76±7.87	52.10±6.28
SDL	3.68±0.86	3.77±0.58	3.29±0.94	3.26±0.62
SAMP	25.97±13.44	24.48±7.58	28.18±13.67	27.29±7.88
SNCV	40.12±7.31	41.12±6.46	41.78±7.41	44.49±6.71
MDL – Distal latency of compound muscle action potential, MAMP – Amplitude of compound muscle action potential, MNCV – Nerve conduction velocity of compound muscle action potential, SDL – Distal latency of sensory nerve action potential, SAMP – Amplitude of sensory nerve action potential, SNCV – Nerve conduction velocity of sensory nerve action potential				

Table 3: Comparison of change in NCS parameters at the end of 12 weeks compared to baseline in control and ALCAR groups

	% decrease MDL Mean±SD	**% increase MAMP Mean±SD**	**% increase MNCV Mean±SD**	**% decrease SDL Mean±SD**	**% increase SAMP Mean±SD**	**% increase SNCV Mean±SD**
Control	4.04 ± 2.4	6.64 ± 4.36	4.15 ± 2.43	12.11 ±11.41	11.38 ± 9.68	4.28 ± 3.22
ALCAR	5.84 ± 3.96	5.23 ± 4.19	3.89 ± 2.67	13.93 ± 7.28	13.04 ±10.49	8.34 ± 4.25
P value	0.012	0.125	0.636	0.375	0.446	<0.001
MDL – Distal latency of compound muscle action potential, MAMP – Amplitude of compound muscle action potential, MNCV – Nerve conduction velocity of compound muscle action potential, SDL – Distal latency of sensory nerve action potential, SAMP – Amplitude of sensory nerve action potential, SNCV – Nerve conduction velocity of sensory nerve						

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action potential

P<0.05 is considered statistically significant

Table 4: ROC analysis showing AUC of NCS parameters

	AUC	**SE**	**95% CI**
% decrease MDL	0.629	0.0603	0.511 to 0.747
% decrease SDL	0.603	0.0610	0.483 to 0.722
% increase MNCV	0.537	0.0624	0.414 to 0.659
% increase SNCV	0.775	0.0510	0.675 to 0.875
% increase MAMP	0.599	0.0608	0.480 to 0.718
% increase SAMP	0.554	0.0624	0.431 to 0.676

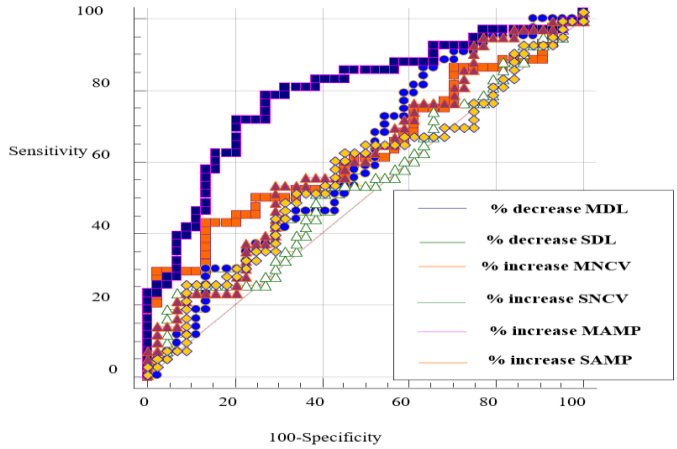

Figure 1: ROC analysis showing AUC of NCS parameters

Table 5: Comparison of BCTQ – Symptom severity scale (BCTQ-SSS) and functional severity scale (BCTQ-FSS) between ALCAR and controls

Time of analysis	**BCTQ-SSS**			**BCTQ-FSS**		
	Mean ± SD		**p value**	**Mean ± SD**		**p value**
	Controls	**ALCAR**		**Controls**	**ALCAR**	
Baseline	28.63±1.80	26.88±2.38	0.410	18±1.61	17.90±1.81	0.450
At the end of 4th week	26±1.89	20.02±2.68	<0.001	16.63±1.52	15.36±2.01	0.001
At the end of 12th week	23.63±1.90	16.20±2.16	<0.001	15.68±1.52	13.65±2.14	<0.001

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At the end of 20th week	21.40±3.05	17.09±1.81	<0.001	15.14±2.04	13.95±2.03	0.040

Table 6: Comparison of ALCAR and controls by DN4

	Mean ± SD		**p value**
	Controls	**ALCAR**	
Baseline	5.34 ± 0.80	5.47 ± 0.84	0.240
At the end of 4th week	4.25±1.08	4.04 ± 0.96	0.148
At the end of 12th week	3.02 ± 0.92	2.09 ± 0.67	<0.001
At the end of 20th week	3.02 ± 0.73	2.5 ± 0.69	0.001

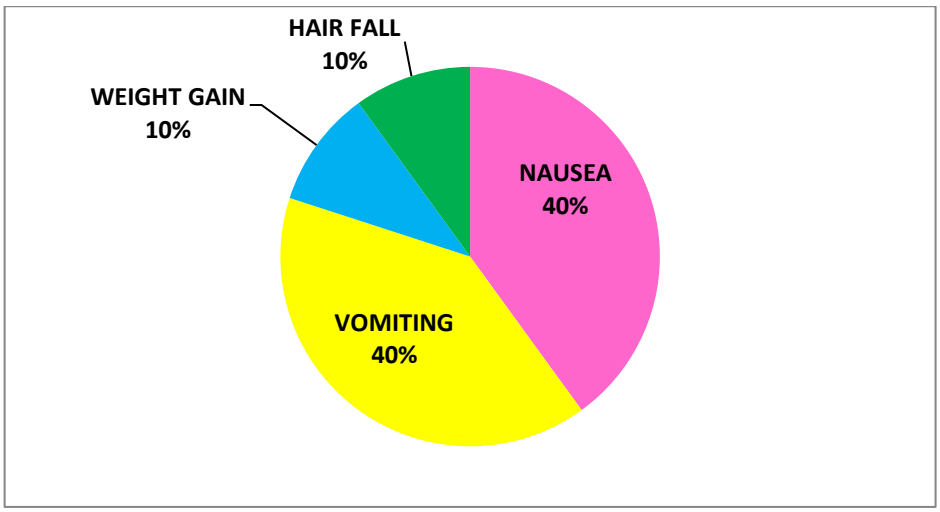

Figure 2: Adverse events in the ALCAR group