

ECCT Modulates the Therapeutic Landscape for Advanced Lung Adenocarcinoma: A Case Series Demonstrating Efficacy Across EGFR-Mutant and Wild-Type Subtypes

Shinichiro Akiyama^{1,2,3*} | Warsito P. Taruno³ | Edi Sukur³ | Ahmad Novian Rahman Hakim³ | Wamid Antaboga³ | Dessy Arianty³

***Correspondence:** Shinichiro Akiyama

Address: ¹Natural Medica Japan., Co. Ltd, Japan Address: R3aoyama 3F, 1-3-1 Kitayama, Minato, Tokyo 107-0061 Japan;

²Department of Clinical Oncology, McGill University, Canada; ³Centre for Medical Physics and Cancer Research of CTECH Laboratories Edwar Technology, Tangerang, Indonesia

E-mail ✉: anc18271@nifty.com

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ABSTRACT

Background: Lung cancer remains a leading cause of cancer-related mortality worldwide. Electro-Capacitive Cancer Therapy (ECCT), a non-invasive treatment utilizing alternating electric fields, has emerged as a promising adjunctive modality by selectively disrupting cancer cell mitosis. This study presents a case series evaluating the clinical outcomes of ECCT as an additive therapy for patients with advanced lung adenocarcinoma. **Methods:** Six patients with clinical stage IIIA to IVA pulmonary adenocarcinoma (two EGFR wild-type, four EGFR-mutant) were treated with ECCT concurrently with standard therapeutic modalities, including chemotherapy, radiotherapy, immunotherapy, or tyrosine kinase inhibitors (TKIs). ECCT was administered at a frequency of 100–150 kHz and a voltage of 20–30 Vpp, typically for 30 minutes to 4 hours daily, five days a week. Treatment response was evaluated through serial imaging (CT and PET-CT). Safety Monitoring and Adverse Event Assessment: All patients were monitored closely for adverse events (AEs) throughout the treatment period, following a prespecified safety monitoring protocol. All adverse events were prospectively documented and graded according to the Common Terminology Criteria for Adverse Events (CTCAE), version 5.0. Special attention was paid to local skin reactions (dermatitis, erythema, or pain) occurring at the ECCT electrode application sites. **Results:** Five patients achieved a partial response (PR), and one maintained stable disease (SD), with significant tumor regression observed across all cases. The treatment was exceptionally well-tolerated, with no severe adverse events reported. Clinical improvements were observed irrespective of the patients' EGFR mutation status. **Conclusion:** This case series suggests that ECCT is a safe and effective adjunctive therapy for stage IV lung adenocarcinoma, significantly enhancing the efficacy of standard treatments across different molecular subtypes. These promising real-world results align with emerging evidence from larger trials and preclinical studies on electric field-based therapies. These findings warrant further investigation in larger, prospective clinical trials to establish ECCT's definitive role in precision oncology frameworks.

Keywords: Lung Adenocarcinoma, Electro-Capacitive Cancer Therapy, ECCT, EGFR

Introduction

Lung cancer remains one of the most prevalent and lethal malignancies worldwide, ranking as the leading cause of cancer-related deaths (Sung *et al.*, 2021; Bray *et al.*, 2024). Despite significant progress in diagnostic and therapeutic strategies, including targeted therapies and immunotherapies, the prognosis for advanced-stage lung cancer remains challenging, with 5-year survival rates below 10% (Patt *et al.*, 2020; Gurney *et al.*, 2021). Tumor recurrence, metastasis, and resistance to therapy continue to impede successful management. These challenges underscore the importance of targeting not only genetic alterations but also the metabolic and microenvironmental hallmarks of lung cancer (Clara *et al.*, 2020; Medema and Vermeulen, 2011; Boedtkjer and Pedersen, 2020; Zhang *et al.*, 2019; Bhutia *et al.*, 2016).

Recent advances in cancer biology have introduced electrical field (EF)-based therapies as promising non-invasive approaches. Electro-Capacitive Cancer Therapy (ECCT) utilizes alternating electric fields to disrupt mitotic spindle assembly by targeting microtubule dynamics during cell division, leading to apoptosis and inhibition of tumor growth (Kirson *et al.*, 2004). In fact, this therapeutic modality, known as Tumor Treating Fields (TTFields), has been established as a standard of care for patients with glioblastoma, where it has been shown to significantly improve survival in a large-scale randomized clinical trial (Stupp *et al.*, 2017).

ECCT has demonstrated antiproliferative effects in various preclinical models, including glioblastoma and breast cancer, with findings suggesting modulation of immune responses and anti-proliferative signaling pathways (Alamsyah *et al.*, 2025; Alamsyah *et al.*, 2021). Moreover, early clinical applications have shown tumor regression and improved patient outcomes (Pratiwi *et al.*, 2019). Initial toxicological studies also suggest a favorable safety profile on vital organs (Alamsyah *et al.*, 2015). Importantly, the electrical and metabolic vulnerabilities of the tumor microenvironment (TME) suggest that ECCT may provide a unique therapeutic advantage by selectively targeting cancer cell physiology while sparing normal tissues (Kirson *et al.*, 2004).

Given the molecular heterogeneity of lung cancer and the limitations of current treatment modalities, ECCT represents a potential paradigm shift in lung cancer therapy. Notably, the efficacy of this approach in advanced non-small cell lung cancer was recently demonstrated in the pivotal LUNAR phase 3 clinical trial, which reported a significant improvement in overall survival for patients receiving TTFields with standard therapies. In light of these promising findings, this study aims to report our clinical experience on the outcomes of ECCT in patients with lung adenocarcinoma and to explore its potential integration into precision oncology strategies.

Method

Study Design and Patient Cohort: This study was conducted as a case series involving six patients diagnosed with advanced pulmonary adenocarcinoma. The patient characteristics are summarized in [Table 1](#). The cohort included two males and four females, with ages ranging from 37 to 76 years. The median age of the cohort was 53 years. Two patients had EGFR wild-type tumors, while four had EGFR-positive mutations. All patients were classified as clinical stage IIIA to IVA.

Treatment Protocol: All patients received ECCT as an adjunctive treatment to their primary standard-of-care therapy. The concurrent treatments included chemotherapy and radiotherapy (Case 1), chemotherapy and immunotherapy (Case 2), gefitinib (Cases 3 and 6), and osimertinib (Cases 4 and 5).

ECCT was administered in two sessions per day, five days per week, with two rest days allocated for patient recovery and condition monitoring. Each patient was provided with a comprehensive ECCT device set: a Helmet ECCT (for the head area), a Vest ECCT (for the localized thoracic area), and a Blanket ECCT (for whole-body application). The wide variation in session duration (from 30 minutes up to 2–4 hours per day) was due to the schedule being highly individualized. This was determined based on individual patient tolerance and concurrent medical therapies (e.g., chemotherapy or radiotherapy). The duration was gradually increased according to tolerance. When ECCT was combined with concurrent medical therapies, the duration per session generally followed the standard ECCT schedule without reduction, provided there were no patient complaints during use.

The average treatment compliance among all six patients was 87.5%, corresponding to a Good Compliance level. This high adherence indicates that the individualized protocol was both well tolerated and feasible for long-term application. No major therapy interruptions or deviations were observed during the treatment period. The detailed session durations and frequency for each patient are summarized in the accompanying [Table 1](#).

Table 1: Patient Characteristics and Treatment Overview.

Case No.	age	sex	pathology	TNM	clinical stage	EGFR	previous tx	concurrent tx	ORR
1	44	M	Adeno	T4N1M0	IIIA	Wild type	none	Chemo + RT	PR
2	52	M	Adeno	T4N2M1a	IVA	Wild type	none	Chemo + ICI	PR
3	37	F	Adeno	T4N2M0	IIIB	Positive	none	Gefitinib	SD
4	76	M	Adeno	T3N1M0	IIIA	Positive	none	Osimertinib	PR
5	54	F	Adeno	T3N2M1b	IVA	Positive	none	Osimertinib	PR
6	64	F	Adeno	T3N2M0	IIIB	Positive	none	Gefitinib	PR

ECCT Device Specification: The Electro-Capacitive Cancer Therapy (ECCT) device was manufactured by PT CTech Lab Edwar Technologi. The system utilized two types of oscillators: C1 (100-150 kHz frequency) and MVS (100 kHz frequency). The non-invasive ECCT applicators consisted of a Helmet ECCT, a Vest ECCT (targeting the localized thoracic area), and a Blanket ECCT (for whole-body application). ECCT was delivered using a non-invasive device, applying alternating electric fields at a frequency of 100-150 kHz and a peak-to-peak voltage of 20-30 Vpp. The treatment was typically administered for 30 minutes to 4 hours daily, five days per week.

Rationale for Frequency Selection (100-150 kHz)

The selection of the 100-150 kHz frequency range was based on multidisciplinary considerations, combining biomedical physics principles, preclinical evidence, and engineering feasibility.

1. **Tissue Penetration and Therapeutic Depth:** Frequencies in the hundreds of kilohertz range achieve deeper tissue penetration compared with megahertz ultrasound or higher-frequency electromagnetic fields, which are rapidly attenuated by biological tissues. This characteristic is critical for reaching both superficial and deeper target regions with less energy loss when applying ECCT via helmet, vest, and blanket modules.
2. **Non-Thermal Mechanobiological Effects:** The 100-150 kHz band modulates biological structures via mechanical vibration, membrane polarization, and ionic displacement, rather than direct heating. This low-intensity, non-thermal effect enhances cellular mechanotransduction (e.g., affecting membrane potential and calcium channel behavior) without exceeding safe thermal thresholds, which is desirable for the long-term safety of chronic or repeated ECCT use.
3. **Safety and Preclinical Validation:** Preclinical studies demonstrated inhibitory effects on tumor growth in mammary tumor models at 100 and 150 kHz, while preserving normal-tissue integrity. This work provides strong biological validation for this frequency band and confirms therapeutic feasibility within low-power, capacitive-coupled limits.
4. **Engineering and Practical Design Rationale:** From an instrumentation perspective, the 100-150 kHz range offers an optimal balance between signal stability, efficient coupling, and manageable heating in wearable systems. It allows stable voltage amplitudes (20-40 Vpp) while ensuring low battery load, efficient energy transfer, and safe exposure levels for repeated daily sessions.

Basis for Individualized Voltage Parameters (20-30 Vpp)

The individualized voltage parameter of 20-30 Vpp was determined through a balance between biological effectiveness, patient safety, and technical feasibility of the ECCT system.

1. **Biological Rationale - Effective Electric-Field Intensity:** The biological effect depends on the electric-field strength (E-field, V/m) reaching the tissue. The 20-30 Vpp output typically generates an E-field of 1-3V/cm at the target tissue surface. This field magnitude is sufficient to alter membrane potential and polarization dynamics of cancer cells without causing ionization or heating, aligning with ranges used in preclinical studies.
2. **Safety and Regulatory Considerations:** The selected voltage ensures that current density and Specific Absorption Rate (SAR) remain far below internationally accepted safety limits for intermediate-frequency electric-field exposure, operating well within established IEEE/ICNIRP guidelines for long-term occupational exposure.
3. **Engineering and Practical Design Basis:** From a hardware standpoint, 20-30 Vpp is the optimal operational window for the oscillator–electrode system. It provides stable output, minimizes power consumption and heat buildup in wearable components, and allows the use of standard lithium-ion batteries (8.4 V, 1500 mAh) for safe, portable daily operation. This range was also validated in preclinical studies using similar configurations.

Assessment and Follow-up: Tumor response was monitored using computed tomography (CT) and positron emission tomography-computed tomography (PET-CT) scans at baseline and regular intervals during the follow-up period. Radiologic changes in tumor size and metabolic activity (SUVmax) were documented to assess treatment efficacy. The clinical course and any adverse events were also monitored throughout the treatment period.

Ethics Statement

1. Institutional Review Board (IRB) Approval

This study was conducted under a protocol titled "Studi Kasus: Terapi Pasien Kanker Dengan Electro Capacitive Treatment (ECCT)" that received ethical approval (Approval No.: KE.01.07/EC/555/2012) from the Institutional Review Board (IRB) of the National Institute of Health Research and Development (KEPK-BPPK, Kementerian Kesehatan RI), Ministry of Health, Republic of Indonesia, on July 13, 2012.

2. Informed Consent and Patient Confidentiality

Written informed consent was obtained from all patients prior to initiating ECCT therapy. Each participant was thoroughly informed about the nature of the treatment, its potential benefits, and the possible risks associated with exposure to low-intensity alternating electric fields. The consent process ensured that all patients fully understood that ECCT is a non-invasive, adjunctive therapeutic approach and that participation was entirely voluntary.

All procedures were conducted in accordance with the ethical standards for clinical research. Patient data were anonymized, and personal identifiers were removed to maintain confidentiality. In addition, continuous clinical monitoring was performed throughout the treatment period to ensure patient safety, document any adverse reactions, and objectively evaluate therapeutic responses.

Results

We report six cases of stage IV pulmonary adenocarcinoma treated with Electro-Capacitive Cancer Therapy (ECCT) in combination with standard modalities. Two patients had EGFR wild-type tumors, while four exhibited EGFR mutations. All patients received ECCT at a frequency of 100–150 kHz and voltage of 20–30 Vpp, typically administered five days per week for 30 minutes to 4 hours daily. Clinical outcomes and imaging findings are summarized below. All six patients demonstrated sustained clinical benefit. Throughout the treatment period, no adverse events of CTCAE Grade 1 or higher were observed, confirming the treatment's favorable safety profile. Five patients achieved a partial response (PR), and one maintained stable disease (SD). The objective response rate (ORR) was 83.33% (5/6), with a corresponding 95% confidence interval (CI) ranging from 35.88% to 99.58% (Clopper-Pearson exact method).

Case 1: EGFR Wild-Type Adenocarcinoma with Chemotherapy and Radiotherapy

A 44-year-old male presented with a right lung mass (7.0 × 5.9 × 7.4 cm) confirmed as EGFR wild-type adenocarcinoma with PD-L1 TPS 80%. He underwent chemotherapy (6 cycles), radiotherapy (48 sessions), and ECCT before and after radiation. CT scans showed progressive tumor reduction: 5.8 × 5.6 × 1.8 cm at 6 months, 5.7 × 5.5 × 1.8 cm at 9 months, and 4.4 × 2.5 × 1.1 cm at 12 months ([Fig. 1A–D](#)). No new lesions were detected. The patient discontinued chemotherapy due to side effects and continued ECCT as maintenance.

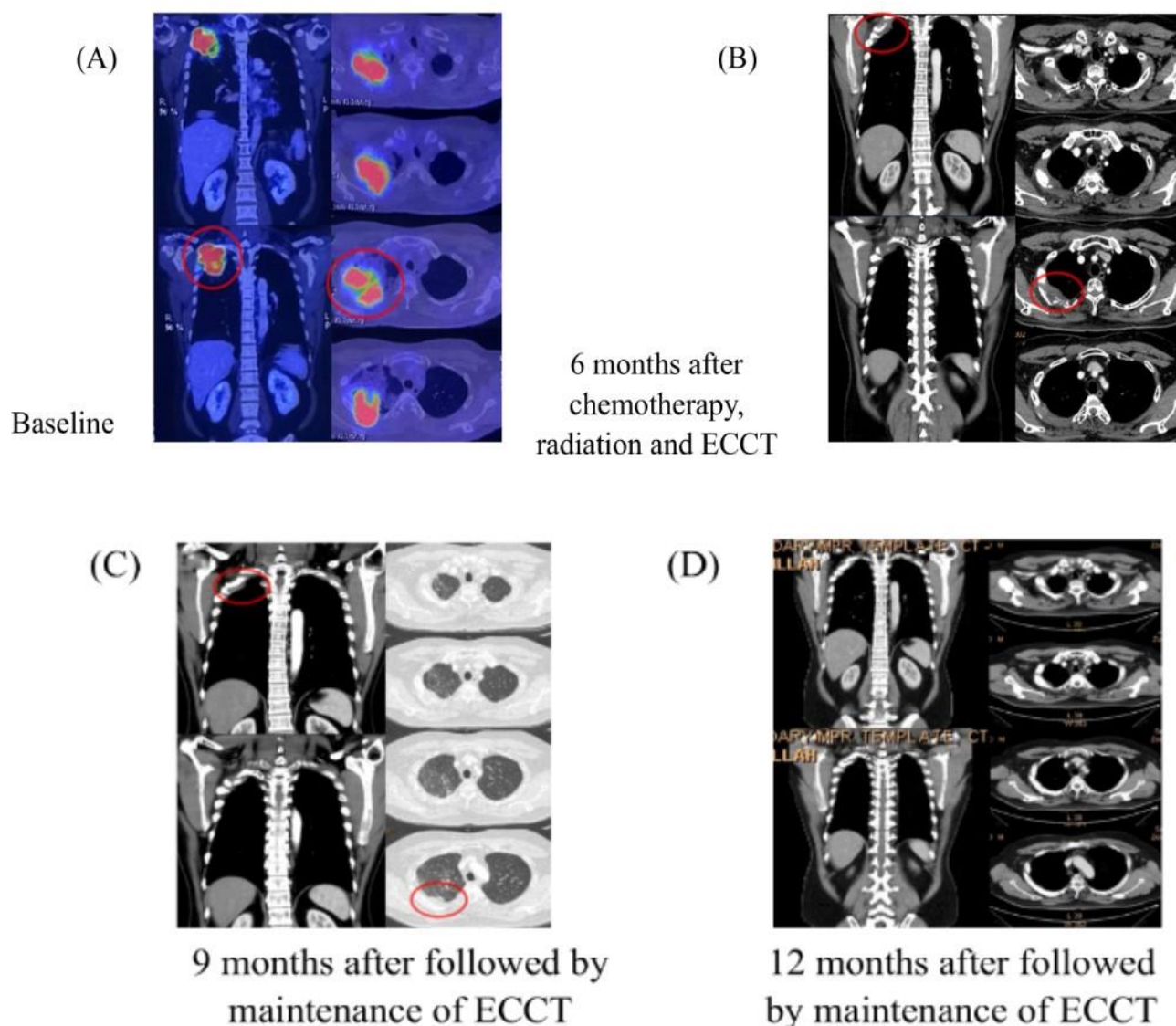


Figure 1: Case 1: Serial CT and PET-CT scans showing baseline tumor (A), reduction at 6 months (B), 9 months (C), and 12 months (D) post-treatment.

Case 2: EGFR Wild-Type Adenocarcinoma with Chemotherapy and Immunotherapy

A 52-year-old male with bilateral pulmonary masses was diagnosed with stage IV EGFR wild-type adenocarcinoma. He received chemotherapy (6 cycles), immunotherapy (discontinued at 3 months), and ECCT. PET-CT at 6 months showed tumor shrinkage from $6.0 \times 5.8 \times 7.5$ cm (SUVmax 30.8) to $4.5 \times 3.9 \times 5.4$ cm (SUVmax 14.2), with reduced nodules and metabolic activity (Fig. 2A–B). ECCT was well tolerated, with no severe adverse events.

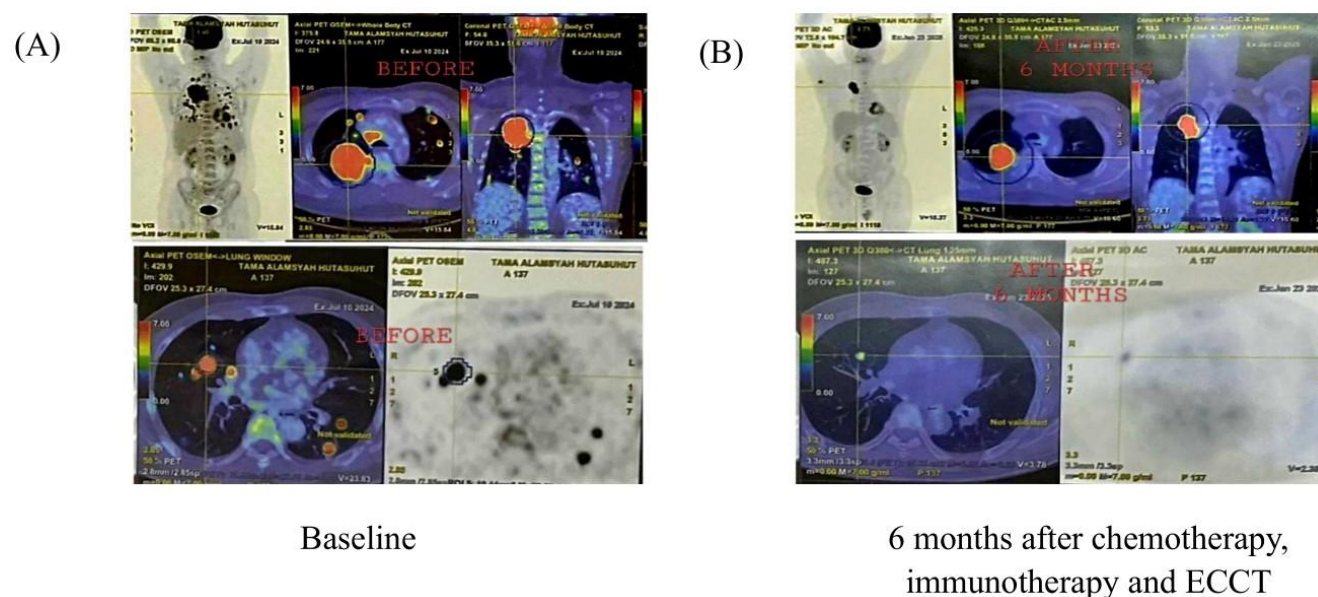


Figure 2: Case 2: Baseline PET-CT (A) and 6-month follow-up (B) showing significant tumor regression.

Case 3: EGFR-Mutant Adenocarcinoma with Gefitinib

A 37-year-old female with EGFR-positive adenocarcinoma presented with dyspnea and chest pain. Initial CT showed a mass of $3.6 \times 3.6 \times 7.3$ cm. Gefitinib and ECCT were initiated concurrently. At 4 months, CT showed partial regression ($2.6 \times 3.6 \times 6.2$ cm), and at 8 months, tumor volume had decreased by $\sim 30\%$ (Fig. 3A–C). The patient resumed normal activities with minimal symptoms.

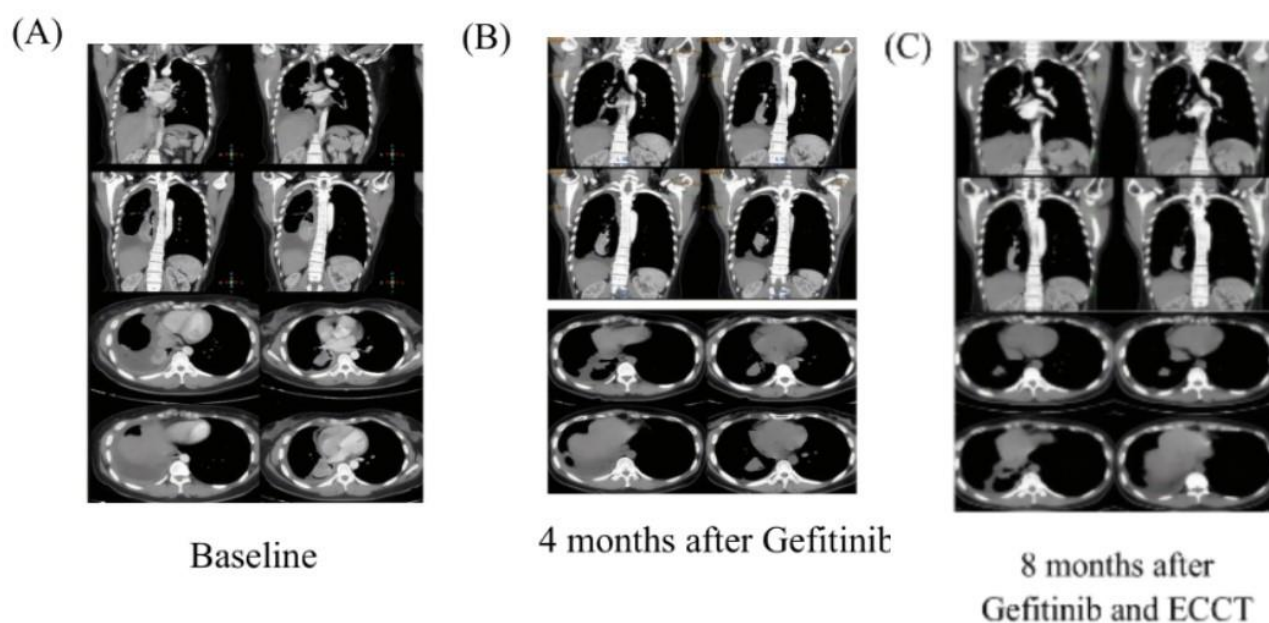


Figure 3: Case 3: Baseline CT (A), 4-month (B), and 8-month (C) scans showing progressive tumor reduction.

Case 4: EGFR-Mutant Adenocarcinoma with Osimertinib

A 76-year-old male with EGFR-positive adenocarcinoma received Osimertinib and ECCT. Initial CT showed a mass of $4.3 \times 6.8 \times 4.0$ cm. Tumor size reduced to $1.3 \times 2.0 \times 1.5$ cm at 3 months and $0.8 \times 0.7 \times 0.7$ cm at 5 months (Fig. 4A–C). The patient maintained stable clinical status and resumed light physical activity.

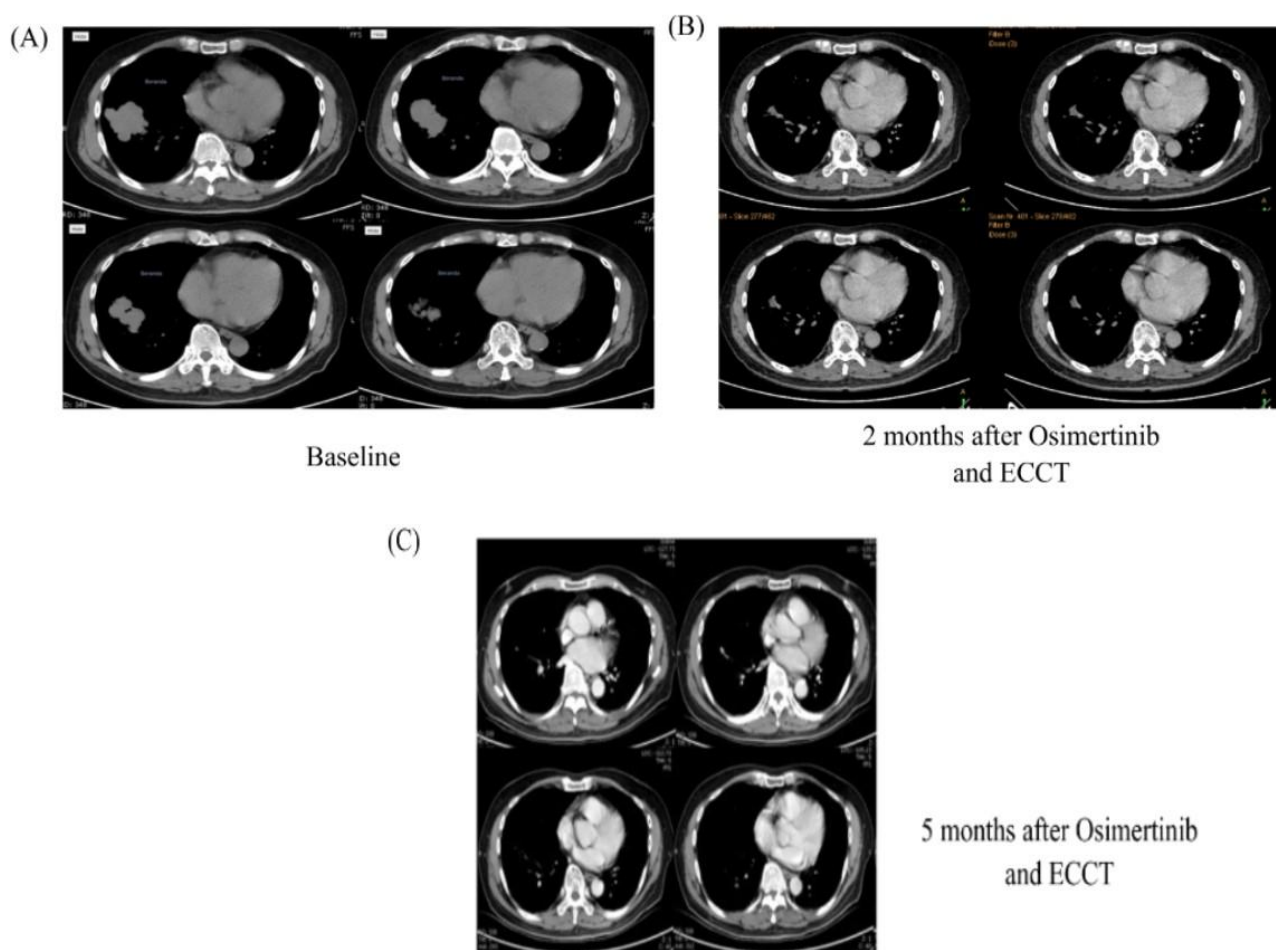


Figure 4: Case 4: Baseline CT (A), 2-month (B), and 5-month (C) scans showing tumor regression.

Case 5: EGFR-Mutant Adenocarcinoma with Osimertinib and Metastases

A 54-year-old female with EGFR-positive adenocarcinoma presented with pulmonary nodules, bone metastases, and brain lesions. She received Osimertinib and ECCT. At 5 months, PET-CT showed tumor shrinkage and resolution of bone metastases. At 9 months, lung lesions were significantly reduced (Fig. 5A–C). The patient regained mobility and daily function.

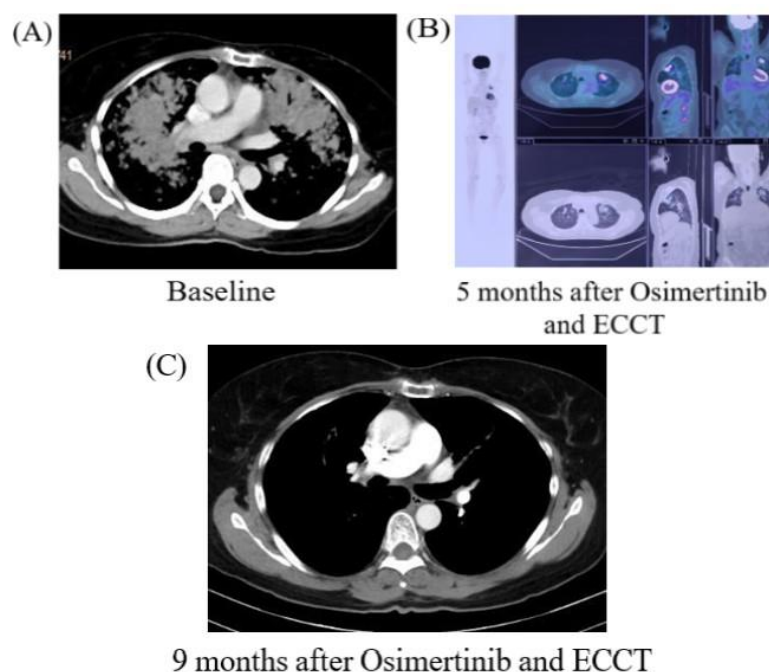


Figure 5: Case 5: Baseline CT (A), 5-month (B), and 9-month (C) scans showing systemic response.

Case 6: EGFR-Mutant Adenocarcinoma with Gefitinib

A 64-year-old female with EGFR-positive adenocarcinoma received Gefitinib and ECCT. Initial CT showed a mass of 5.2×4.8 cm. After one year, dyspnea resolved. Treatment was interrupted during the COVID-19 pandemic. Upon resumption, CT in 2025 showed tumor reduction compared to 2023 (Fig. 6A–C). The patient remained asymptomatic.

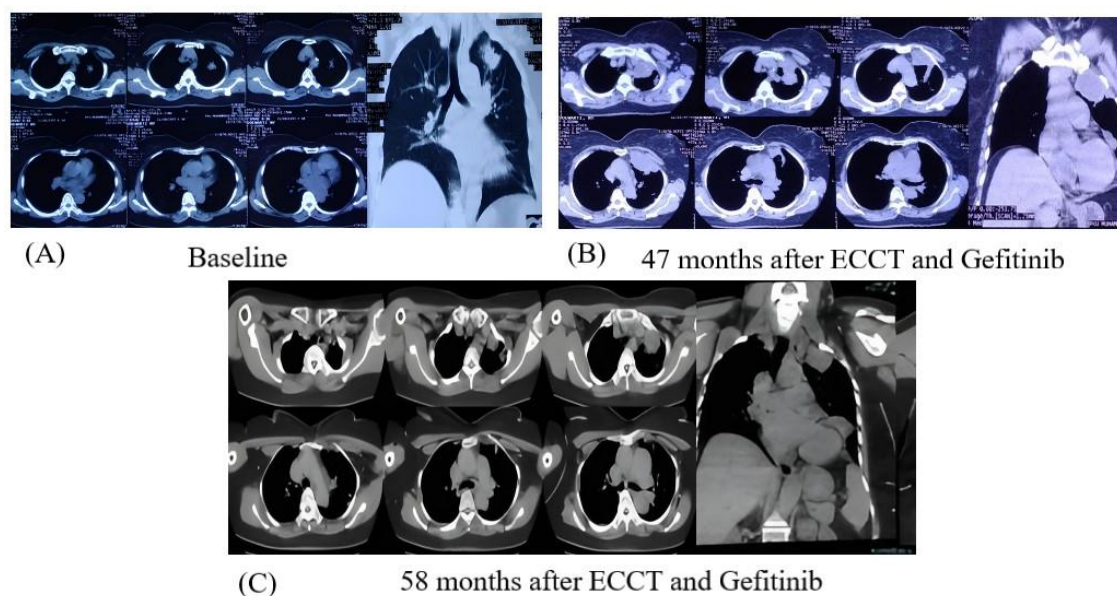


Figure 6: Case 6: Baseline CT (A), 47-month (B), and 58-month (C) scans showing long-term disease control.

Integration with Discussion

These results demonstrate consistent partial responses across diverse EGFR mutation statuses and treatment combinations. Notably, ECCT was well tolerated and contributed to tumor regression in both EGFR wild-type and EGFR-mutant cases. The imaging data support the hypothesis that ECCT may enhance the efficacy of conventional and targeted therapies by exploiting the electrical and metabolic vulnerabilities of the tumor microenvironment.

The observed outcomes align with prior preclinical findings on ECCT's antiproliferative effects, and suggest its potential utility in precision oncology, particularly for patients with limited options or intolerance to systemic therapies.

Discussion

This case series highlights the potential clinical utility of Electro-Capacitive Cancer Therapy (ECCT) as an adjunctive modality in the management of stage IV pulmonary adenocarcinoma. Our findings demonstrate that the addition of ECCT to standard treatments resulted in sustained clinical benefit across six patients, with five achieving partial remission and one maintaining stable disease. Despite the small sample size (N=6), the high observed ORR of 83.33% is noteworthy, though the 95% CI remains broad (35.88%-99.58%), suggesting the need for validation in larger cohorts. Particularly, Case 2, who received ECCT concurrently with an immune checkpoint inhibitor (ICI), achieved a deep and sustained partial remission. This outcome aligns with emerging evidence regarding the synergistic interaction between electric field therapies and modern immunotherapy. Mechanistically, ECCT shares the fundamental principle of Tumor Treating Fields (TTFields) by utilizing alternating electric fields to disrupt mitotic spindle formation. However, ECCT is distinct in its physical parameters and biological reach. Specifically, ECCT operates using a capacitive coupling method at a low voltage (20-30 Vpp) and an intermediate frequency (100-150 kHz). Furthermore, preclinical studies suggest that beyond direct cytostasis, ECCT may induce immunomodulatory effects, such as enhancing cytotoxic T cell responses and altering signaling pathways related to inflammation, which warrants further investigation into its comprehensive anti-tumor profile. While the therapeutic efficacy of Tumor Treating Fields (TTFields) has traditionally been attributed to the physical disruption of mitosis, recent investigations suggest that its anti-tumor activity is also potentiated by the induction of an immune response. The cell death induced by TTFields-specifically Immunogenic Cell Death (ICD)- leads to the release of Danger-Associated Molecular Patterns (DAMPs), which are proposed to facilitate the maturation and activation of dendritic cells (DCs) in preclinical models.

Furthermore, TTFields contribute to the modulation of the Tumor Microenvironment (TME).

Specifically, they have been shown to reduce the infiltration of immunosuppressive cell populations, such as Myeloid-Derived Suppressor Cells (MDSCs) and Regulatory T cells (Tregs), and to alter PD-L1 expression on tumor cells. This effect creates an environment that enhances the action of Immune Checkpoint Inhibitors (ICIs), thus providing a mechanistic basis for the observed synergistic effects in combination strategies.

Specifically, recent literature indicates that the application of Tumor Treating Fields (TTFields) can enhance the immune response by promoting immunogenic cell death (ICD) and subsequently increasing the presentation of tumor antigens. Furthermore, this physical stress may modulate the tumor microenvironment and increase T-cell infiltration, thus potentially overcoming resistance to ICI. Therefore, the exceptional response observed in Case 2 may be a direct result of this potent synergy, where ECCT acts not only as a cytostatic agent but also as an *in-situ* tumor vaccine, maximizing the efficacy of the accompanying ICI treatment. The clinical utility of combining TTFields with standard systemic therapy was definitively established by the pivotal Phase III LUNAR trial in patients with metastatic Non-Small Cell Lung Cancer (mNSCLC). In patients who had progressed on or after platinum-based chemotherapy, the addition of TTFields to standard-of-care (ICI or docetaxel) resulted in a statistically significant and clinically meaningful prolongation of Overall Survival (OS) (18). Notably, a subgroup analysis of the ICI-treated population (TTFields + ICI vs. ICI alone) demonstrated the most pronounced benefit, with a median OS of 18.5 months versus 10.8 months (HR 0.63; P=0.03). This outcome not only positions TTFields as a valuable new treatment option for this patient cohort but also provides strong clinical evidence supporting the hypothesis that an immunological mechanism is substantially involved in the therapeutic action of TTFields.

Conversely, our patient cohort also included four patients (Cases 3-6) receiving ECCT concurrently with Epidermal Growth Factor Receptor-Tyrosine Kinase Inhibitors (EGFR-TKIs), such as gefitinib and osimertinib. All four patients demonstrated sustained clinical benefit without significant additive toxicity, suggesting a feasible and effective combination regimen. The potential synergy between TTFields and targeted therapies, particularly in NSCLC, has been highlighted in recent academic reviews. KÖHLER M, *et al.* specifically reviewed the rationale for combining TTFields with EGFR-TKIs and other targeted agents. They propose that TTFields may sensitize cancer cells to these molecular drugs by disrupting the cell cycle and altering membrane permeability, thereby enhancing drug uptake. Our observation of prolonged stability and response in patients with EGFR-mutated disease strongly aligns with the mechanistic hypothesis described in this narrative review. This suggests that ECCT may serve as a critical component in overcoming or delaying resistance mechanisms often associated with TKI monotherapy.

Potential Mechanisms for Synergistic Efficacy

The promising clinical response observed with the concurrent administration of ECCT and systemic therapies (ICI in Case 2; TKI in Cases 3–6) suggests a potential synergistic relationship, mediated by ECCT's unique biophysical effects.

1. Synergy with Immunotherapy (Case 2: ICI):

ECCT's direct cytotoxic effect may induce Immunogenic Cell Death (ICD). As cancer cells undergo ECCT-mediated apoptosis and necrosis, they release Damage-Associated Molecular Patterns (DAMPs), such as ATP and HMGB1, into the tumor microenvironment. This release acts as an *in situ* vaccination, promoting the maturation and migration of Dendritic Cells (DCs) and enhancing antigen presentation to cytotoxic T lymphocytes. Consequently, ECCT could convert "cold" tumors into "hot" immunogenic ones, significantly overcoming the primary resistance mechanisms often encountered with ICI alone, leading to enhanced and sustained anti-tumor immunity.

2. Synergy with Targeted Therapy (Cases 3–6: TKI):

For EGFR-mutant cases treated with TKI, ECCT may enhance drug efficacy through two mechanisms. First, the application of alternating electric fields may temporarily increase cell membrane permeability (electroporation-like effect), facilitating the intracellular uptake of TKI agents into tumor cells, thereby increasing local drug concentration and maximizing targeted inhibition. Second, the ECCT-induced stress response and disruption of the tumor microenvironment may suppress acquired resistance pathways or re-sensitize TKI-resistant clones, leading to more durable responses observed in these cases. Nonetheless, several limitations must be acknowledged. First, the absence of a control group precludes definitive conclusions regarding ECCT's independent efficacy. Second, imaging-based assessments were not uniformly standardized, and metabolic activity was not consistently quantified across all cases. Third, the compassionate use setting introduces potential selection bias, as patients opting for ECCT may differ in clinical motivation or disease trajectory.

Economic and Global Accessibility Rationale

While Tumor Treating Fields (TTFields) has demonstrated compelling efficacy in randomized clinical trials, its high acquisition and running costs often pose significant challenges to patient access, particularly in resource-limited settings. Recent cost-effectiveness analyses for TTFields in glioblastoma suggest substantial economic burden, even when factoring in survival benefits.

In contrast, ECCT utilizes a low-cost, intermediate-frequency device and offers a potentially more accessible and economically feasible adjunctive treatment. This economic profile differentiates ECCT not only on a mechanistic level but also on a global public health level, making it a viable option for patients where TTFields access is currently constrained.

Future studies should aim to validate these findings in larger, controlled cohorts with standardized imaging protocols and biomarker analyses. Integration of ECCT into precision oncology frameworks—potentially guided by TME profiling or electrical impedance mapping—may further refine patient selection and optimize therapeutic outcomes. The high upfront cost of TTFields therapy necessitates rigorous health economic evaluation to justify its clinical value. Following the positive LUNAR trial results, recent Cost-Effectiveness Analyses (CEAs) have been conducted. For instance, model analysis from a US payer perspective suggested that the strategy of combining TTFields with ICI or docetaxel may be considered cost-effective, as the Incremental Cost-Effectiveness Ratio (ICER) falls within conventional willingness-to-pay thresholds and is accompanied by a gain in Quality-Adjusted Life Years (QALYs)¹⁷.

However, consistent with prior CEAs for Glioblastoma (GBM) treatment, the ICER for mNSCLC remains highly sensitive to the cost of the device. Therefore, while the clinical efficacy is clear, adjustments in pricing and reimbursement policies within healthcare systems will remain a critical factor in determining the widespread accessibility of TTFields therapy.

Conclusion

This case series demonstrates that Electro-Capacitive Cancer Therapy (ECCT) is a promising and well-tolerated adjunctive modality in the management of stage IV pulmonary adenocarcinoma. Given its favorable safety profile and synergistic potential, ECCT warrants rigorous, prospective clinical investigation to firmly establish its optimal role within precision oncology frameworks.

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