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Review

Emrelis: A New Approach in Treating MET-high Locally Advanced or Metastatic Non-squamous NSCLC; A Mini Review

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Abstract

Non-small cell lung cancer (NSCLC) accounts for 85% of all lung cancers; however, a subset of patients experiences disease progression associated with alterations in the Mesenchymal-Epithelial Transition Factor (c-MET) pathway. These include MET exon 14–skipping mutations, which promote tumor growth, metastasis, and resistance to epidermal growth factor receptor (EGFR)-targeted therapies. Telisotuzumab vedotin (TV-t, Emrelis), a novel first-in-class c-MET inhibitor, is under investigation as a targeted therapy for c-MET-high NSCLC. This article highlights the therapeutic potential of TV-t as a targeted therapy for advanced NSCLC patients with limited post-treatment options. A targeted literature review was conducted using PubMed and ClinicalTrials.gov (2018 and 2025) with terms including "Telisotuzumab Vedotin," "c-MET," "c-MET–high," "c-MET overexpression," "MET exon 14," and "NSCLC." Both clinical and preclinical studies of the efficacy, safety, and pharmacological properties of TV-t were included. Also, those studies reported clinical findings relevant to TV-t in c-MET-altered NSCLC. In the pivotal LUMINOSITY phase II trial, TV-t showed a 28.6% overall response rate (ORR) in EGFR-wildtype, c-MET-overexpressing NSCLC, with 34.6% ORR in c-MET-high patients. Median duration of response was 9.0 months, overall survival 14.5 months, and progression-free survival (PFS) 5.7 months for the c-MET-high subgroup. In phase Ib studies, TV-t combined with osimertinib achieved a 50% ORR and 7.4-month PFS, while TV-t with nivolumab, the median PFS was 7.2 months, but ORR was low (7.4%). Common grade 3 or higher toxicities occurred in 27.9% of patients and included neuropathy, anemia, and pulmonary embolism, with no hepatotoxicity. Remarkable cardiac safety was observed. TV-t demonstrated promising efficacy and tolerability in patients, highlighting its clinical potential. However, further studies are needed to confirm its survival advantage, the durability of response, and safety profile, and to establish its long-term value and support its integration into routine clinical practice.

KEYWORDS: Non-squamous NSCLC, Emrelis, telisotuzumab vedotin, MET-high NSCLC, c-MET overexpression

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INTRODUCTION

Non-small cell lung cancer (NSCLC) is the most common type of lung cancer in adults. Approximately 85% of lung cancers are classified as NSCLC.[1] The three main histological subtypes of NSCLC according to the World Health Organization/International Association for the Study of Lung Cancer classification are squamous cell carcinoma, adenocarcinoma, and large-cell carcinoma. Among these subtypes, non-squamous subtypes (adenocarcinoma and large cell carcinoma) are the most prevalent, making these subtypes the leading cause of preventable death from lung cancer.[2] Mesenchymal-Epithelial Transition Factor (c-MET) is a receptor tyrosine kinase that, upon activation by hepatocyte growth factor (HGF), triggers downstream signaling pathways (e.g., PI3K/AKT), promoting cellular growth, tissue infiltration, and motility. Its abnormal activation causes persistent endosomal signalling, which leads to oncogenesis and is implicated in NSCLC.

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More recently, evidence indicates that c-MET is also involved in resistance to conventional cytotoxic chemotherapy through downstream activation of several endosomal pathways, particularly the PI3K/AKT pathway. It raises the clinical need for targeted therapies in previously treated, c-MET-high NSCLC patients, for whom the HGF-MET pathway has a confirmed vital role.[3] Food and Drug Administration (FDA) has recently granted accelerated approval to telisotuzumab vedotin (TV-t), a first-in-class c-MET-directed antibody-drug conjugate (ADC), for the treatment of NSCLC in adult patients.[4] This article aims to highlight the therapeutic potential of TV-t as a promising targeted therapy for adult patients with advanced NSCLC who have limited treatment options following prior therapies.

Biological Role of c-MET in NSCLC

c-MET is a transmembrane receptor tyrosine kinase that is activated by its ligand, HGF, which belongs to the family of plasminogen-related growth factors. HGF/c-MET signalling leads to tissue repair, wound healing, tissue regeneration, angiogenesis, and epithelial-mesenchymal transition (EMT).[5] The HGF/c-MET axis works together with other tyrosine kinases and activates several key downstream signaling networks within tumor cells. These pathways include the PI3K/AKT, JAK/STAT, Ras/MAPK, SRC, and Wnt/β-catenin, all of which play crucial roles in cancer growth, metastasis, and survival.[5]

The c-MET tyrosine kinase receptor is overexpressed and overrepresented in NSCLC.[6] Activation of c-MET triggers phosphorylation at key tyrosine residues (Y1003, Y1313, Y1230/1234/1235, Y1349, Y1365), initiating downstream signaling cascades. Moreover, c-MET-mediated SRC activation promotes EMT, which contributes to the initiation of malignant transformation.[6] MET exon 14 skipping mutation (METΔex14) is present in three of every hundred patients with NSCLC.[7] When exon 14 is skipped, the CBL-mediated degradation of the c-MET protein is disrupted, preventing normal receptor turnover. This results in accumulation of c-MET receptors on the cell surface and sustained activation of oncogenic c-MET signaling, driving tumor growth and progression.[7] Both c-MET overexpression and exon 14 skipping contribute to an increased incidence of NSCLC.

Main Points

- Non-small cell lung cancer (NSCLC) constitutes approximately 85% of all lung cancer cases, making it the most prevalent form in adults.
- The three main histological subtypes of NSCLC are squamous cell carcinoma, adenocarcinoma, and large cell carcinoma. Non-squamous types (adenocarcinoma and large cell carcinoma) are more common.
- Non-squamous subtypes of NSCLC are a leading cause of preventable lung cancer deaths, highlighting their clinical significance.
- The Mesenchymal-Epithelial Transition Factor (c-MET) receptor tyrosine kinase, activated by hepatocyte growth factor (HGF), plays a key role in regulating cell proliferation, motility, and invasion.
- Aberrant activation of the c-MET/HGF pathway contributes to oncogenesis in NSCLC due to sustained endosomal signaling and uncontrolled cellular behavior.

The c-MET receptor promotes resistance to standard chemotherapeutic agents by activating the PI3K/AKT signaling axis. The epidermal growth factor receptor (EGFR) T790M mutation primarily mediates acquired resistance to EGFR-tyrosine kinase inhibitor (TKI) in NSCLC, whereas KRAS mutations are mainly associated with primary resistance. Chronic co-activation of the ERK and AKT signaling pathways drives EGFR-TKI resistance in NSCLC cells. Oncogenic SRC and RAS proteins can trigger the EMT, leading to the breakdown of E-cadherin complexes at cell junctions. This disruption enhances cellular invasiveness and metastatic potential.[6]

Mechanism of Action

Receptor-mediated Internalization

TV-t is a novel ADC that advances targeted therapy for NSCLC with MET overexpression. The anti-c-MET monoclonal antibody moiety exhibits remarkable selectivity for its targets. It is about tenfold stronger than the binding of natural HGF. Due to its potent binding to c-MET, the drug selectively targets c-MET-overexpressing tumor cells while having minimal effect on normal tissues. The binding initiates the drug's endocytosis. The complex is rapidly internalized via clathrin-mediated endocytosis, with 80% of surface-bound drug internalized within 30 minutes.

MMAE Payload and Microtubule Disruption

Another noteworthy feature of the drug is the cleavable valine-citrulline linker, which remains intact during circulation but is cleaved by lysosomal proteases.[8] This potent microtubule-disrupting payload, monomethyl auristatin E (MMAE), is directly delivered to tumor cells via a controlled-release approach, resulting in intracellular concentrations that are 50- to 100-fold higher than levels in the normal surrounding tissues. The binding of MMAE causes severe disruption of the microtubule network, leading to cell death within 24-48 hours. The bystander effect, in which hydrophobic MMAE molecules permeate cell membranes and affect neighboring tumor cells, is particularly useful.[9]

Comparison with MET Inhibitors

Unlike small-molecule inhibitors such as capmatinib and tepotinib, which target the MET exon 14-skipping mutation, TV-t acts on all c-MET-overexpressing tumors, regardless of mutation status. The mechanism of action, showing TV-t delivery via c-MET, is illustrated in Figure 1.

Clinical Trial Evidence

First-in-Human Phase I Trials

The earliest multicenter phase I study (NCT02099058) established the safety and dosing of TV-t and provided early signals of efficacy in adults with advanced solid tumors, including c-MET-overexpressing NSCLC. The overall response rate (ORR) was 18.8% (3 PRs) among 16 c-MET-positive NSCLC patients at doses of 2.4-3.0 mg/kg, with a median progression-free survival (MPFS) of 5.7 months and a median duration of response (DOR) of 4.8 months. Non-NSCLC tumors showed no reaction. TV-t drug levels were dose-proportional, with a terminal elimination half-life of two to four days.[10] A Japanese phase I trial confirmed

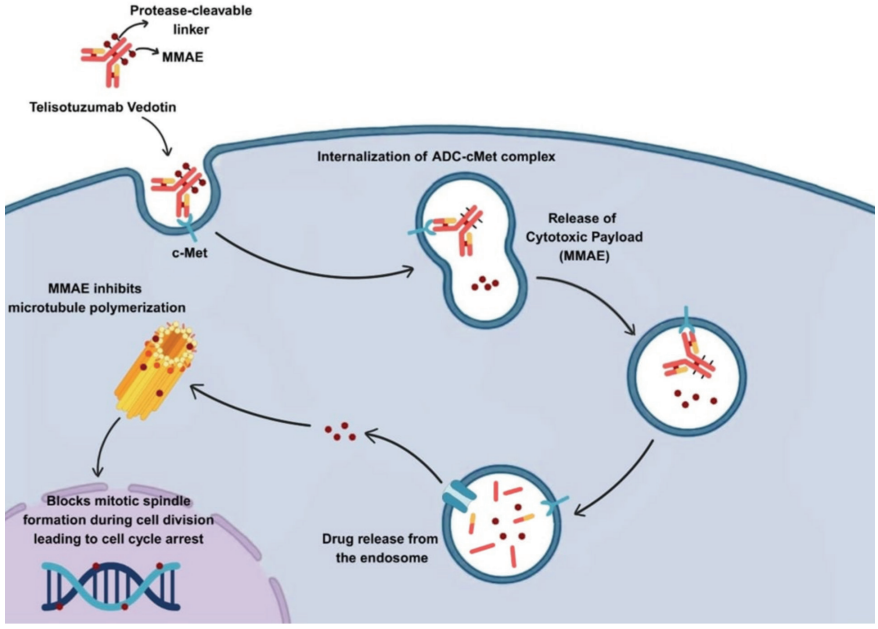

Figure 1. The mechanism of action of telisotuzumab vedotin

MMAE: monomethyl auristatin E, ADC: antibody–drug conjugate, c-MET: Mesenchymal-Epithelial Transition Factor

TV-t's tolerability and effectiveness in an Asian population. With a disease control rate of 89% (8/9 patients), the observed ORR was 22% and the MFS was 7.1 months.[11]

LUMINOSITY Phase II Trial

In the pivotal LUMINOSITY phase II trial (NCT03539536), TV-t monotherapy was administered to 172 patients with treated EGFR-wildtype non-squamous NSCLC and c-Met overexpression. It was a phase II, open-label, single-arm, multicenter trial evaluating the effectiveness of TV-t. An ORR of 28.6% was achieved. Patients with c-MET overexpression had an ORR of 34.6%. For the intermediate group, ORR was 22.9%. Response duration was 9.0 months in the MET-high subgroup and 7.2 months in the intermediate subgroup. Although survival outcomes were favorable across the entire cohort, the median overall survival and progression-PFS were 14.5 months and 5.7 months, respectively.[12]

Phase Ib Combination Studies

The combination potential of TV-t has been evaluated in two significant phase Ib studies.

TV-t + osimertinib: Among patients with EGFR mutations who progressed on osimertinib, TV-t with continued EGFR blockade demonstrated notable activity, achieving a 50% response rate and an MPFS of 7.4 months.[13]

TV-t + nivolumab: The phase Ib nivolumab combination study (NCT02099058) offered insights into TV-t's possible synergy with immunotherapy, albeit with relatively subtle efficacy signals. Among the 27 c-MET-positive patients, the combination showed acceptable safety. However, very limited antitumor activity was demonstrated, with an ORR of 7.4%. Interestingly, an MPFS of 7.2 months was achieved overall and was similar between programmed death-ligand 1-positive and -negative subgroups. This trial showed that TV-t's pharmacokinetic profile did not change when nivolumab was administered, which is an important consideration for combination therapies.[14]

TV-t + erlotinib: An earlier phase Ib trial of erlotinib plus TV-t in patients pretreated with EGFR TKIs showed similarly optimistic outcomes and a notable response rate of 52.6% in the c-MET-high group.[15]

Comparative Clinical Interpretation

Divergence in response rates between combination regimens: The difference in ORR for osimertinib (50%) versus nivolumab (7.4%) combinations appears multifactorial: First, mechanistic distinctions exist: by co-inhibiting EGFR and c-MET, osimertinib is more effective than nivolumab in targeting acquired c-Met amplification, a known EGFR-evasive pathway, because immune-modulatory mechanisms do not synergize with telisotuzumab vedotin's cytotoxic activity. Second, patient enrichment: osimertinib studies limited inclusion to EGFR-mutant patients with evidence of acquired resistance, whereas nivolumab enrolled an unselected, heterogeneous patient population.

Comparison with other c-MET targeted therapies (tepotinib, capmatinib): Telisotuzumab vedotin, with an ORR of 28.6%, has a lower ORR than tepotinib, which has reported ORRs of 40-45% in cases with MET exon 14-skipping mutations, likely because telisotuzumab is indicated based on c-MET overexpression rather than specific genetic alterations. The DOR associated with TV-t (9.0 months in MET-high cohorts) is similar to that observed with capmatinib (8.3 to 9.7 months in METex14), although established differences in trial design prevent firm cross-trial inference.

Safety differentiation from other antibody-drug conjugates: The TV-t construct demonstrates certain hematologic advantages: the incidence of severe neutropenia (grade ≥3) is reported as a comparatively modest 4%, lower than the rates

observed with trastuzumab deruxtecan. When unique toxicity events are investigated, the profile of TV-t regarding interstitial lung disease warrants emphasis. c-MET receptor blockade is associated with a decreased incidence of this complication relative to conjugates that target HER2. Peripheral neuropathy attributable to the bridged MMAE cytotoxic payload is consistent with patterns observed in other ADCs that employ this payload class. Table 1 summarizes clinical trials.

Adverse Effects and Safety Profile

Common adverse effects observed in the phase II trial include peripheral sensory neuropathy, fatigue, and peripheral edema. Grade 3 or higher events occurred in 27.9% of patients; peripheral neuropathy was the most common (7%).[12] Anemia (11%) and pulmonary embolism (8%), the most frequent grade 3 treatment-related side effects, were observed in a phase Ib study of TV-t in combination with osimertinib.[13]

The absence of certain toxicities in the toxicity profile greatly enhances its benefit. Unlike most targeted therapies, telisotuzumab vedotin has not been associated with interstitial lung disease or pneumonitis, which represents a notable lack of pulmonary toxicity. No hepatotoxicity was observed. Cardiac safety is also notable.

Patients should be closely monitored for signs of peripheral neuropathy, such as burning sensations, neuropathic pain, or muscle weakness. Upfront management strategies have been designed to enhance tolerability. For osimertinib combination

Table 1. Summary of all the clinical trials

Study/phase	Study population and design	ORR (%)	Median DOR (months)	Median PFS (months)	Median OS (months)	Common any-grade AEs	Common grade ≥3 AEs
First-in-human phase I (10)	48 advanced solid tumors (incl. NSCLC)	c-MET + NSCLC: 18.8%	4.8	5.7	NR	Fatigue (42%), nausea (27%), constipation (27%), decreased appetite (23%), vomiting (21%), dyspnea (21%), diarrhea (19%), peripheral edema (19%), and neuropathy (17%)	Fatigue, anemia, neutropenia, and hypoalbuminemia (4% each)
Phase I study in Japanese (11)	Japanese patients with advanced solid tumors	22%	8.2 (overall DCR 89%)	7.1	NR	Peripheral sensory neuropathy (44%), and nausea, decreased appetite, and decreased WBC count (33% each)	Neutropenia and hypoalbuminemia in two patients (22%) each, and hypophosphatemia and fatigue in one patient (11%) each
Phase II LUMINOSITY trial (12)	172 patients with EGFR-wildtype NSCLC	Overall: 28.6%; c-MET high: 34.6%; c-MET intermediate: 22.9%	Met high: 9.0 Met intermediate: 7.2	5.7	14.5	Peripheral sensory neuropathy 30%, peripheral edema 16%, fatigue 14%	Peripheral sensory neuropathy 7%
Phase Ib (TV-t+ osimertinib) (13)	38 patients with EGFR-mutant NSCLC	50.0%	NR	7.4	NR	Peripheral sensory neuropathy 50%, peripheral edema 32%, nausea 24%	Anemia 11%, pulmonary embolism 8%
Phase Ib (Teliso-V + Nivolumab) (14)	37 patients; 27 c-Met IHC+ NSCLC (PD-L1 + n = 15; PD-L1– n = 9; PD-L1 unknown n = 3)	7.4	NR	Overall: 7.2; PD-L1+: 7.2; PD-L1–: 4.5	NR	Fatigue 27%, peripheral sensory neuropathy 19%	Not specified
Phase Ib (Teliso-V + Erlotinib) (15)	42 EGFR-mutant NSCLC	Overall: 32.1%; MET-high: 52.6%	NR	5.9 overall; 6.8 in non-T790M+ / unknown		Peripheral sensory neuropathy	

NSCLC: non-small cell lung cancer, c-MET: Mesenchymal-Epithelial Transition Factor, EGFR: epidermal growth factor receptor, PD-L1: programmed death-ligand 1, IHC: immunohistochemistry, TV-t: telisotuzumab vedotin, ADC: antibody–drug conjugate, MMAE: monomethyl auristatin E, ORR: overall response rate, DOR: duration of response, PFS: progression-free survival, OS: overall survival, DCR: disease control rate, NR: not reported, AE: adverse event, PR: partial response, WBC: white blood cell, EMT: epithelial–mesenchymal transition

therapy, hematologic monitoring is crucial. In patients with mild to moderate organ impairment, a dosage of 1.9 mg/kg every two weeks, up to a maximum of 190 mg, exhibits linear pharmacokinetics and does not require adjustment. Research continues into different dosing approaches that may broaden the therapeutic window.

Diagnostic Tool: VENTANA MET RxDx Assay

The VENTANA MET SP44 rxdx assay is approved by the FDA as a companion diagnostic test for the detection of c-MET protein overexpression in patients eligible for treatment with TV-t.[16] It is an immunohistochemistry based test that employs the rabbit monoclonal anti-MET clone SP44 expression in formalin-fixed paraffin-embedded non-squamous NSCLC specimens by light microscopy.[17] The assay has been used in clinical studies, including the phase II LUMINOSITY trial and phase Ib combination trials, where it was used as a pre-screening tool. It stratifies patients into MET-high and MET-intermediate groups based on staining intensities and the proportion of tumor cells that stain strongly. In LUMINOSITY, MET-high was defined as ≥50% of tumor cells exhibiting strong (3+) staining; this demonstrated higher response rates to telisotuzumab vedotin.[18] This standardized diagnostic approach ensures accurate patient selection and enables oncologists to target therapy to patients most likely to respond, thereby avoiding unnecessary treatment in others.

Clinical Relevance and Future Directions

TV-t represents an important advance in precision oncology by providing a targeted treatment option for patients with NSCLC who exhibit high c-MET protein expression but lack MET exon 14-skipping mutations, a group for whom MET TKIs such as capmatinib are ineffective. For this population with limited options for targeted therapies, TV-t offers a biomarker-driven alternative.[10] Due to its favorable safety profile, TV-t also has potential for combination with immune checkpoint inhibitors (anti-PD-1/PD-L1 agents) or conventional chemotherapy. Ongoing phase II and Ib trials are refining patient selection and optimizing treatment approaches. However, the knowledge gaps remain regarding the durability of the response, the mechanisms of resistance, and the long-term survival outcomes. Beyond NSCLC, the success of TV-t highlights the broader role of companion diagnostics and biomarker-driven therapies in guiding precision medicine across oncology.

CONCLUSION

The approval of Emrelis by the FDA represents a major step forward in the treatment of locally advanced or metastatic non-squamous NSCLC and is expected to reduce the number of deaths it causes globally. In its LUMINOSITY trial, it demonstrated a notable ORR and a favorable tolerability profile in patients, highlighting its clinical potential. However, its real-world data and long-term results remain limited. Further studies are needed to confirm its survival benefits, assess the durability of responses, and characterize its toxicity profile, thereby validating its long-term benefits; these data will be critical for defining its role in routine clinical practice.

Footnotes

Authorship Contributions

Surgical and Medical Practices: S.M.K., A.A., Concept: S.M.K., A.N., A.A., Design: S.M.K., A.N., A.A., Data Collection or Processing: S.M.K., A.N., A.A., Y.S., Analysis or Interpretation: S.M.K., A.A., Y.S., S.R.M.K., Literature Search: S.M.K., A.N., Y.S., S.R.M.K., Writing: S.M.K., A.N., Y.S., S.R.M.K.

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