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ONCOLOGY

Advanced non small cell lung cancer: response to microwave ablation and EGFR Status

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Abstract
Objectives To verify the association between *EGFR* status and clinical response to microwave ablation (MWA) and survival.
Methods NSCLC patients with known *EGFR* status and treated with MWA in combination with chemotherapy were retrospectively enrolled in the study.
Results A total of 61 patients were recruited. *EGFR* mutations were found in 28 patients (39.4 %), and were more common in women (67.7 %) and nonsmokers (74.1 %). Complete ablation was achieved in 69.7 % of patients with *EGFR* mutant tumours and in 82.1 % of patients with *EGFR* wild-type tumours ($p = 0.216$). The median progression-free survival (PFS) and overall survival (OS) were 8.3 months and 27.2 months in patients with an *EGFR* mutant tumour. The corresponding values were 5.4 months ($p = 0.162$) and 17.8 months ($p = 0.209$) in patients with an *EGFR* wild-type tumour. Patients with complete ablation had longer PFS (7.8 months vs. 4.2 months, $p = 0.024$) and OS (28.1 months vs. 12.6 months, $p = 0.001$) than those with incomplete ablation. Multivariate analyses also showed that response to MWA was an independent prognostic factor for OS, but *EGFR* status was not, and that neither response to MWA nor *EGFR* status was a prognostic factor for PFS.
Conclusions The *EGFR* status was not related to response to MWA, and response to MWA was a predictor of survival.

Key Points
- *EGFR mutations were commonly seen in women and in nonsmokers*
- *EGFR status had no correlation with the response to MWA, PFS and OS.*
- *The response to MWA could predict PFS and OS.*

Keywords *EGFR* mutation · Microwave ablation · Non-small-cell lung cancer · Progression-free survival · Overall survival

Introduction

Lung cancer remains the leading cause of cancer-related mortality in China [1]. It is estimated that 486,555 patients per year died due to lung cancer and less than 20 % of patients survived for 5 years up to the year 2010 [1]. For patients with advanced non-small-cell lung cancer (NSCLC), platinum-based combination chemotherapy is the first-line standard treatment [2, 3], except in patients with tumours carrying an *EGFR* mutation or an *EML4-ALK* gene fusion, who benefit from epidermal growth factor receptor tyrosine kinase inhibitors (EGFR-TKIs) and ALK inhibitors, respectively [4–10].

Microwave ablation (MWA) has been used as an alternative treatment for patients who are unfit for surgery with severe cardiovascular and pulmonary diseases [11–13]. Furthermore, our previous studies have shown that MWA combined with chemotherapy can extend progression-free survival (PFS) of patients with advanced NSCLC when compared to chemotherapy alone [14, 15].

EGFR 19 del and 21 point mutations are the most common sensitive mutations of *EGFR*, and are most commonly found in tumours in women, patients with adenocarcinoma, never or

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light former smokers, and patients of east-Asian origin [16]. Several studies have demonstrated that patients with *EGFR* mutant tumours also benefit more from radiotherapy than those with *EGFR* wild-type tumours [17–21]. However, the association between *EGFR* mutation status and MWA outcomes in patients treated with MWA combined with chemotherapy or EGFR-TKIs has not yet been clarified. We assumed that patients with *EGFR* mutations respond better to MWA than patients with *EGFR* wild-type tumours and conducted this study to evaluate the relationship between *EGFR* mutation status and MWA outcomes.

Materials and methods

Patient selection

Between 12 April 2013 and 1 October 2015 patients who met the following criteria were retrospectively enrolled in the study: (1) pathologically verified peripheral NSCLC, (2) stage IIIB or IV, (3) adequate formalin-fixed paraffin-embedded (FFPE) tumour slides available for *EGFR* mutation testing, (4) chemotherapy-naive (except patients with recurrence treated with adjuvant chemotherapy or adjuvant radiation), (5) an Eastern Cooperation Oncology Group performance status (ECOG PS) of 0 to 2, and (6) adequate pulmonary, cardiac, hepatic, renal and haematological functions to allow anticancer treatment. The study was approved by the Ethics Committee of Shandong Provincial affiliated to Shandong University. All patients provided written informed consent.

EGFR tests

DNA was extracted from 4-μm FFPE tumour slides using the QIAamp DNA FFPE tissue kit (Qiagen, Germany) according to the manufacturer's instructions. Extracted DNA was dissolved in Tris-HCl (10 mmol/L, pH 8.0) to measure the DNA concentration using ultraviolet spectrophotometry. The recommended DNA concentration was 10 ng/μL. The BPSP-qPCR detection kit (Amoy Diagnostics Ltd., Fujian, China) was used for fluorescence PCR determination (StrataGene MX3000P) of four types of *EGFR* gene mutations in exons 18 to 21. A positive control to ensure adequate DNA quality and a no template control were included according to the protocol. A classic S-curve and a C_t value of >30 were considered a positive result, indicating the presence of a mutation.

Anticancer treatments

Patients were treated with MWA and chemotherapy or EGFR-TKIs. MWA of the primary tumour sites was first performed, followed by chemotherapy or EGFR-TKI treatment after an interval of 7 days. A GE LightSpeed 64 V spiral CT machine was used for the procedure. An MTC-3C MWA instrument was used (YZB 1408-2003, no. SFDA (III) 20073251059; Nanjing Qiya Medical Equipment Co., Jiangsu, China). The emission frequency of the microwave antenna was 2,450 ± 50 MHz, and the output energy ranged from 0 W to 100 W. The microwave antenna had an effective length of 100 – 180 mm and an outside diameter of 14 – 20 G, with a long tapered pointed end. A water circulation cooling system was used to reduce the surface temperature of the antenna. MWA with an output of 60 – 70 W has an ablative zone of nearly 3.5 × 3 cm. For tumours of 3.5 cm or more, the ablation procedure was performed with two ablative antennas.

Local anaesthesia and preemptive analgesia were used before the procedure, after which patients were moved into the appropriate position. A CT scan was performed to enable the channel to be planned preoperatively, and was followed by a skin incision at the puncture point and passing the ablation microwave antenna through the deeper layers of tissue to the target lesion. Once the cold circulating pipes and circulating pumps had been connected to the MWA antenna and MWA machine with a cable, MWA was performed. After the procedure, the MWA antenna was removed, and the puncture site disinfected and bandaged. A CT scan was performed immediately to evaluate the ablation margin, size and shape, and to ensure there were no complications. The proposed ablation margin was 0.5 cm.

The procedures used for MWA and the chemotherapy regimens have been previously described [14, 15]. The chemotherapy regimen consisted of pemetrexed at 500 mg/m^2 on day 1, docetaxel at 75 mg/m^2 on day 1 or gemcitabine at 1,250 mg/m^2 on days 1 and 8, in combination with cisplatin at 75 mg/m^2 on day 1 or carboplatin with an area under the curve of 5 mg/ml · min administered every 3 weeks for up to six cycles. In patients treated with EGFR-TKIs, oral gefitinib (250 mg daily) or erlotinib (150 mg daily) was administered until disease progression or intolerable toxicity.

Response assessment

The response to MWA was assessed according to expert consensus guidelines for thermal ablation of primary and metastatic lung tumours [22], and the response to chemotherapy was assessed according to the RECIST 1.1 guidelines. One month after ablation, a contrast-enhanced CT scan was performed to assess the response to MWA. Complete ablation was indicated by lesion disappearance, complete cavernous formation, fibrotic progression or scarring, or an involuted or unchanged solid nodule without signs on the contrast-enhanced CT scan, and/or atelectasis. Incomplete ablation was indicated by incomplete cavernous formation with some solid or liquid components remaining and irregular peripheral or internal enhancement signs on the CT scan, partial fibrosis with solid residues in the fibrotic lesion seen as irregular

peripheral or internal enhancement signs on the CT scan, and/or solid nodules with unchanged or increased size also seen as irregular peripheral or internal enhancement signs on the CT scan [22].

In patients treated with chemotherapy, a CT scan was performed every two cycles. In those receiving targeted therapies, the first CT scan was performed after 1 month, and then every 2 months. PFS was defined from the time of MWA to the time of disease progression at the site of the ablated lesion or other metastatic sites. Overall survival (OS) was defined as the time from the start of MWA to death from any cause.

Statistical analysis

SPSS v.17.0 was used for statistical analysis. The chi-squared test was used to evaluate the associations between the *EGFR* mutation status and clinicopathological characteristics or MWA response. PFS and OS were assessed using the Kaplan-Meier method, along with univariate and Cox regression multivariate analyses (including the *EGFR* status and those factors with $p < 0.1$ in univariate analysis). All tests were two-sided, and a p values <0.05 were considered significant.

Results

Patient characteristics

Between 12 April 2013 and 1 October 2015 a total of 61 patients with NSCLC were enrolled. Of these 61 patients, 31 were women, 31 were 60 years of age or older, 31 were non-smokers, 58 had adenocarcinoma, 58 had an ECOG PS of 1, and 57 had stage IV disease. Local regional lymph nodes and pulmonary metastases were the most common metastatic sites (82.0 % and 50.8 %, respectively), and 48 patients (78.7 %) had one or two metastatic sites. The baseline characteristics of the patients and the primary tumours treated with MWA were summarized in Table 1.

Relationship between the *EGFR* status and baseline characteristics

All 61 patients were screened for *EGFR* mutations. In 28 patients (45.9 %), the tumour was found to carry an *EGFR* mutation, 13 of which were 19 del, 13 were L858R point mutation, and 2 were T790M mutation.

EGFR mutations were significantly more common in women (21 of 31 women, 67.7 %, vs. 7 of 30 men, 23.3 %; $p = 0.001$), and nonsmokers (23 of 31 non-smokers, 74.1 %, vs. 5 of 30 smokers, 16.7 %; $p = 0.000$). The baseline characteristics and treatment of the patients with and without *EGFR* mutation are shown in Table 2.

Table 1 Baseline characteristics of the 61 enrolled patients

Characteristic	Value
Gender, *n* (%)	
Male	30 (49.2)
Female	31 (50.8)
Age (years), *n* (%)	
≥60	31 (50.8)
<60	30 (49.2)
Smoking history, *n* (%)	
Nonsmoker	31 (50.8)
Smoker	30 (49.2)
ECOG performance status, *n* (%)	
0	3 (4.9)
1	58 (95.1)
Pathology, *n* (%)	
Adenocarcinoma	58 (95.1)
Other[a]	3 (4.9)
Stage, *n* (%)	
IIIB	14 (23.0)
IV	47 (77.0)
EGFR status, *n* (%)	
Positive	28 (45.9)
Negative	33 (54.1)
EGFR mutation, *n* (%)	
19 del	13 (21.3)
L858R	13 (21.3)
T790M	2 (3.3)
Tumour location, *n* (%)	
Peripheral/central	
Peripheral	61 (100.0)
Central	0 (0.0)
Right/left	
Right	31 (50.8)
Left	30 (49.2)
Lobe	
Upper and middle	39 (63.9)
Lower	22 (36.1)
Tumour size (cm), mean (range)	3.7 (0.8 – 10.0)
Tumour size (cm), *n* (%)	
≥3.5	34 (55.7)
<3.5	27 (44.3)
MWA power (W), *n* (%)	
70	54 (88.5)
60	7 (11.5)
MWA time (min), mean (range)	13.0 (3.0 – 56.0)
Number of antennas, *n* (%)	
One	26 (42.6)
Two	35 (57.4)
Metastatic sites, *n* (%)	
Local regional lymph node	50 (82.0)
Lung	31 (50.8)
Liver	2 (3.3)
Bone	9 (14.8)
Brain	8 (13.1)
Adrenal gland	10 (16.4)
Pleura and pericardium	12 (19.7)
Other[b]	2 (3.3)
Number of metastatic sites, *n* (%)	
One	17 (27.9)
Two	31 (50.8)
Three or more	13 (21.3)

MWA Microwave ablation

[a] Two patients had squamous cell carcinoma patients; one patient had unclassified non-small-cell lung cancer

[b] One patient had renal metastasis; one patient had retroperitoneal lymph node metastasis

Table 2 Baseline characteristics of patients with and without EGFR mutation

Characteristic	EGFR-positive	EGFR-negative	*p* value
Gender, *n* (%)			
Male	7 (25.0)	23 (69.7)	0.001
Female	21 (75.0)	10 (30.3)	
Age (years), *n* (%)			
≥ 60	10 (35.7)	21 (63.6)	0.055
< 60	18 (64.3)	12 (36.4)	
Smoking history, *n* (%)			
Nonsmoker	23 (82.1)	8 (24.2)	0.000
Smoker	5 (17.9)	25 (75.8)	
ECOG performance status, *n* (%)			
0	2 (7.1)	1 (3.0)	0.589
1	26 (92.9)	32 (97.0)	
Pathology, *n* (%)			
Adenocarcinoma	27 (96.4)	31 (93.9)	1.000
Other	1 (3.6)	2 (6.1)	
Stage, *n* (%)			
IIIB	4 (14.3)	10 (30.3)	0.138
IV	24 (85.7)	23 (69.7)	
TKI treatment, *n* (%)			
First-line	13 (46.4)	1 (3.0)	0.272
Subsequent line	7 (25.0)	3 (9.1)	
First-line treatment, *n* (%)			
TKI	13 (3.0)	1 (3.0)	0.001
Chemotherapy	15 (97.0)	32 (97.0)	
First-line chemotherapy regimen, *n* (%)			
Pemetrexed	12 (80.0)	24 (75.0)	1.000
Other	3 (20.0)[a]	8 (25.0)[b]	
Second-line and subsequent line treatment, *n* (%)			
Second-line	11 (39.3)	13 (39.4)	1.000
Subsequent line	3 (10.7)	9 (27.3)	
Tumour location			
Peripheral/central			
Peripheral	28 (100)	33 (100)	1.000
Central	0 (0)	0 (0)	
Right/left			
Right	17 (60.7)	14 (42.4)	0.154
Left	11 (39.3)	19 (57.6)	
Lobe			
Upper and middle	17 (60.7)	22 (66.7)	0.613
Lower	11 (39.3)	11 (33.3)	
Tumour size (cm), mean (range)	3.5 (0.8 – 6.5)	3.9 (1.4 – 10.0)	
Tumour size (cm), *n* (%)			
≥ 3.5	13 (46.4)	21 (63.6)	0.178
< 3.5	15 (53.6)	12 (36.4)	
MWA power (W), *n* (%)			
70	25 (89.3)	29 (87.9)	1.000
60	3 (10.7)	4 (12.1)	
MWA time (min), mean (range)	12.7 (3.0 – 32.0)	13.2 (4.5 – 56.0)	
Number of antennas, *n* (%)			
One	14 (50)	12 (36.4)	0.283
Two	14 (50)	21 (63.6)	

MWA Microwave ablation, *TKI* Tyrosine kinase inhibitor

[a] Three patients received docetaxel

[b] Six, one and one patients received docetaxel, gemcitabine and paclitaxel, respectively

EGFR status and response to MWA in relation to survival

Progression-free survival

All patients underwent MWA at the primary tumour site. Among the 28 patients with *EGFR* mutations, 20 received EGFR-TKIs, as first-line treatment in 13 and as second or subsequent lines of treatment in 7. Another 15 patients were treated with platinum-based combination chemotherapy as first-line treatment, pemetrexed in 12 and docetaxel in 3. Four of the *EGFR* wild-type patients received EGFR-TKIs, as first-line treatment in one and as second or subsequent lines

of treatment in three. In 32 patients, platinum-based combination chemotherapy was used as first-line treatment (pemetrexed in 24, docetaxel in 6, gemcitabine in 1 and paclitaxel in 1).

Complete ablation was achieved in 46 patients (75.4 %). No correlation between *EGFR* status and complete ablation was observed (23 of 33 patients, 69.7 %, with *EGFR* mutant tumours vs. 23 of 28 patients, 82.1 %, with *EGFR* wild-type tumours; $p = 0.261$).

At the time of the last follow up on 14 November 2015 (median follow-up 16.9 months, range 2.5 – 36.5 months) 38 patients had progressed including 24 patients with an *EGFR* wild-type tumour and 14 with an *EGFR* mutant tumour. The median PFS was 6.2 months (95 % confidence interval, CI, 4.9 – 7.4 months) in the whole group of patients. No difference was observed between the two groups. The median PFS was 8.3 months (95 % CI 3.8 – 12.8 months) in patients with an *EGFR* mutant tumour, and 5.4 months (95 % CI 3.2 – 7.6 months; $p = 0.162$) in patients with an *EGFR* wild-type tumour (Fig. 1). Patients with complete ablation had longer PFS (7.8 months, 95 % CI 5.0 – 10.6 months, vs. 4.2 months, 95 % CI 2.2 – 6.2 months; $p = 0.024$, Fig. 2). PFS was also longer in female patients (8.5 months, 95 % CI 2.3 – 17.6 months, vs. 4.2 months, 95 % CI 1.6 – 6.8 months; $p = 0.011$) and those with adenocarcinoma (6.3 months, 95 % CI 4.7 – 7.9 months, vs. 2.3 months, 95 % CI 1.0 – 4.3 months; $p = 0.010$, Table 3). In the multivariate analysis including *EGFR* status, gender, pathology and response to MWA, gender was the only significant prognostic factor for PFS ($p = 0.032$); *EGFR* status, pathology and response to MWA (patients with complete ablation) were not significant prognostic factors for PFS ($p = 0.579$, $p = 0.216$ and $p = 0.092$, respectively; Table 4).

Overall survival

At the time of data cut-off, 16 patients had died, including 12 patients with an *EGFR* wild-type tumour and 4 with an *EGFR* mutant tumour. The median OS was 21.7 months (95 % CI 18.6 – 24.7 months). Kaplan-Meier univariate analysis showed that response to MWA was associated with OS: patients with complete ablation had a median OS of 28.1 months (95 % CI 22.8 – 33.4 months) and those with incomplete ablation a median OS of 12.6 months (95 % CI 8.7 – 16.5 months; $p = 0.001$, Fig. 3). However, *EGFR* mutation status was not associated with OS: patients with an *EGFR* mutant tumour had a median OS of 17.8 months (95 % CI 14.1 – 21.5 months) and those with an *EGFR* wild-type

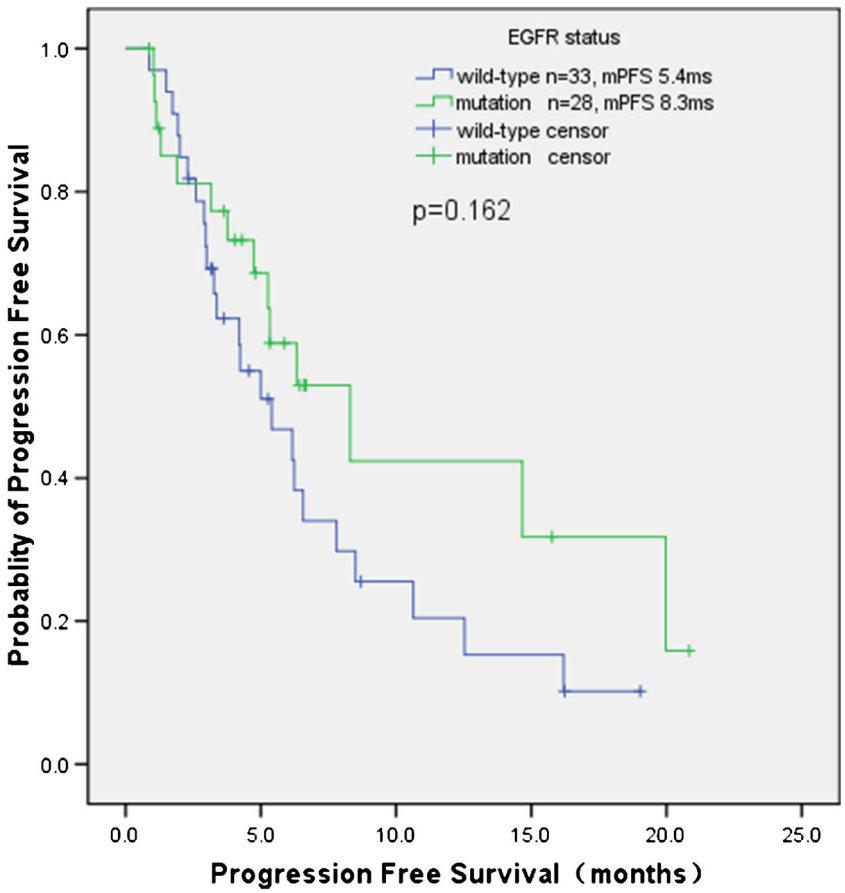

Fig. 1 Kaplan-Meier curves of PFS in relation to EGFR status

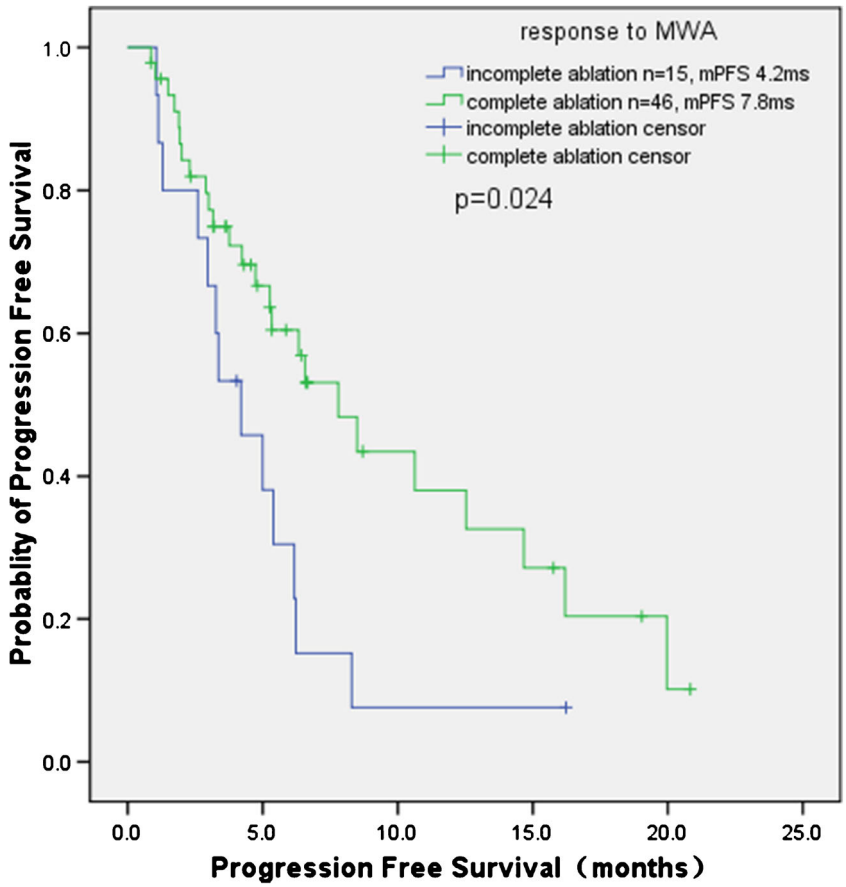

Fig. 2 Kaplan-Meier curves of PFS in relation to response to MWA

Table 3 Univariate analysis of progression-free survival

Variable	Median PFS (months)	95 % CI	*p* value
Gender			
Male	4.2	1.6 – 6.8	0.011
Female	8.5	2.3 – 17.6	
Age			
≥ 60 years	5.3	2.9 – 7.7	0.242
< 60 years	7.8	5.0 – 10.6	
Smoking history			
Nonsmoker	6.3	4.8 – 11.8	0.379
Smoker	5.0	3.1 – 6.9	
Pathology			
Adenocarcinoma	6.3	4.7 – 7.9	0.010
Other	2.3	0.3 – 4.3	
EGFR mutation			
Positive	8.3	3.8 – 12.8	0.162
Negative	5.4	3.2 – 7.4	
Response to MWA			
No	4.2	2.2 – 6.2	0.024
Yes	7.8	5.0 – 10.6	
TKI treatment			
Yes	6.2	4.9 – 7.6	0.782
No	6.2	3.1 – 9.2	

MWA microwave ablation, *TKI* Tyrosine kinase inhibitor

tumour a median OS of 27.2 months (95 % CI 19.6 – 24.8 months; $p = 0.209$, Fig. 4). Furthermore, female patients, patients with adenocarcinoma and patients younger than 60 years also had better OS (Table 5). In the Cox regression multivariate analysis including *EGFR* mutation status, gender, age, smoking history, histological type and response to MWA, pathology ($p = 0.002$) and response to MWA ($p = 0.005$) were independent prognostic factors for OS (Table 6).

Discussion

In this study, *EGFR* mutations in NSCLC were more common in women and nonsmokers. The *EGFR* mutation status was

Table 4 Multivariate analysis of progression-free survival

Variable	OR	95 % CI	*p* value
Gender	2.190	1.068 – 4.491	0.032
Pathology	2.249	0.622 – 8.125	0.216
EGFR mutation	0.943	0.444 – 2.001	0.879
Response to MWA	0.519	0.242 – 1.114	0.092

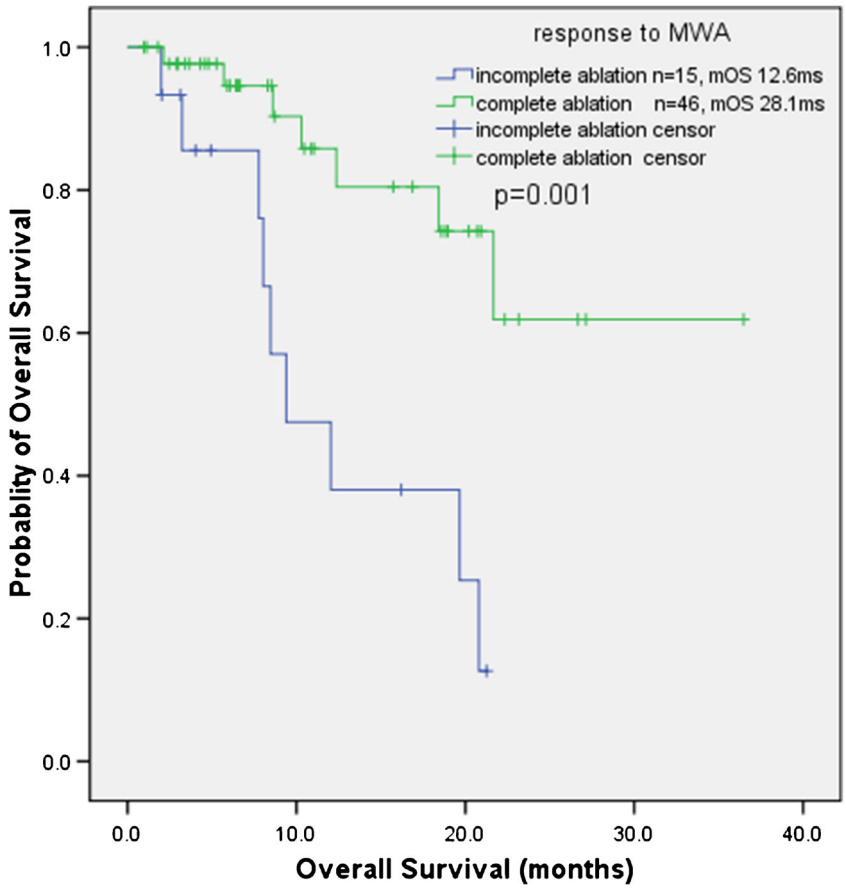

Fig. 3 Kaplan-Meier curves of OS in relation to response to MWA

not related to the outcome of MWA, including response, PFS and OS. Patients with complete ablation had longer PFS and OS. *EGFR* mutations are characteristic of a unique subtype of NSCLC. Patients with *EGFR*-sensitive mutations may derive more benefit from EGFR-TKIs than from conventional chemotherapy, leading to both a higher overall response rate and a longer PFS [4–7], and even extended OS [23].

In this study, *EGFR* mutation was found in the tumours of 45.9 % of the enrolled patients, and was more common in women (67.7 %) and nonsmokers (74.1 %). These findings are similar to those of previous studies [16]. Previous studies have also shown that *EGFR* mutations are more frequent in adenocarcinomas [16]. Our failure to confirm this finding could have been because of the imbalance in histological tumour types in this study.

Thermal ablation has been widely used for the treatment of several types of solid tumour [24–28]. In this study, we explored the relationship between the *EGFR* status and response to MWA, as well as survival after the procedure. In contrast to the findings of previous studies of the relationship between *EGFR* status and radiotherapy, *EGFR* tumour mutations were not associated with response to MWA, PFS or OS. Das et al. [29] found that *EGFR* mutant lung cancer cell lines are 500 – 1,000-fold more sensitive than *EGFR* wild-type cells to ionizing radiation, and Yagishita et al. [20] and Mak et al. [21] found that patients with *EGFR* mutant NSCLC derive greater benefit –from chemoradiotherapy than their counterparts with *EGFR* wild-type tumours. Several studies have also shown that patients with *EGFR* mutant lung cancers with brain metastases have longer PFS and local control time after undergoing whole-brain radiation therapy [17–19], which might be a result of different mechanisms of action during these treatments. Radiation could induce DNA double-strand breaks, for which homologous recombination repair and non-homologous end-joining repair are the common restorative pathways. DNA-dependent protein kinase (DNA-PK) is a key enzyme in nonhomologous end-joining repair, and the protein product from *EGFR* genes carrying a sensitive mutation is defective in radiation-induced translocation to the nucleus and fails to bind the catalytic and regulatory subunits of DNA-PK [30]. However, the electromagnetic heating in MWA that results from water molecule agitation produces friction and heat, and thus induces cell death via coagulation necrosis, which could not be prevented by cellular repair pathways [31].

Many factors can influence PFS and OS. Although in this study *EGFR* status was not associated with survival, patients with complete ablation had longer PFS and OS. Perhaps the

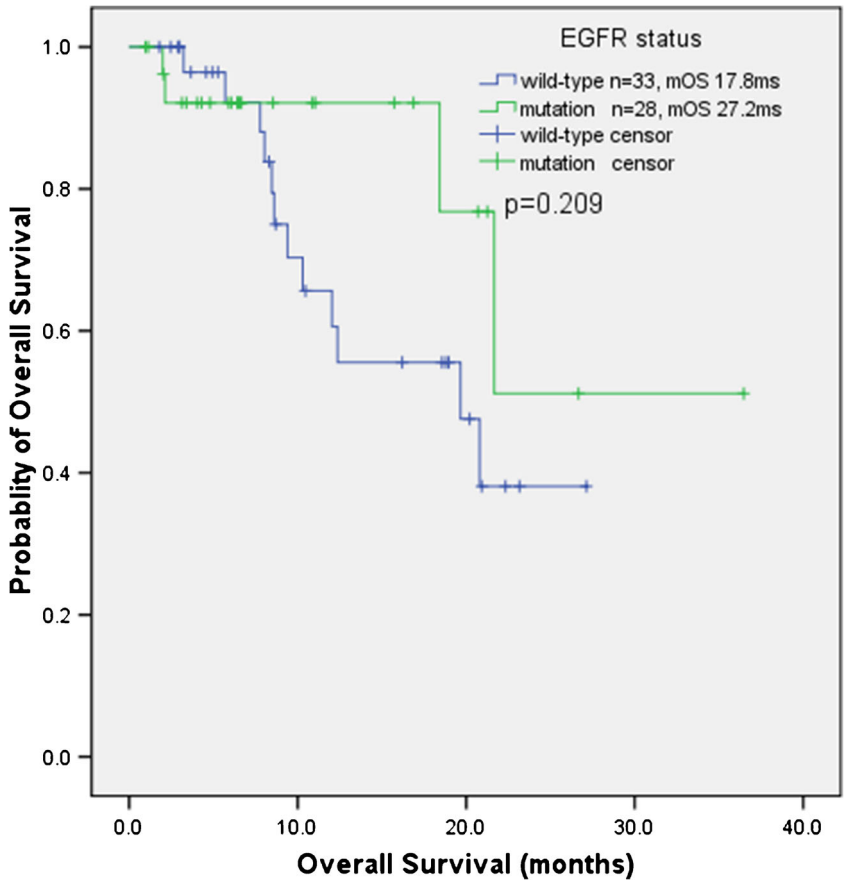

Fig. 4 Kaplan-Meier curves of OS in relation to EGFR status

Table 5 Univariate analyses of overall survival

	Median OS (months)	95 %CI	*P*
Gender			
Male	15.2	11.2 – 19.2	0.003
Female	29.6	23.5 – 35.8	
Age			
≥ 60 years	16.2	12.0 – 24.4	0.011
< 60 years	23.4	20.6 – 26.4	
Smoking history			
Nonsmoker	28.1	21.7 – 34.5	0.052
Smoker	15.3	12.1 – 18.4	
Pathology			
Adenocarcinoma	24.2	19.4 – 29.0	0.000
Other	4.5	1.7 – 7.3	
EGFR mutation			
Positive	27.2	19.6 – 24.8	0.209
Negative	17.8	14.1 – 21.5	
Response to MWA			
No	12.6	8.7 – 16.5	0.001
Yes	28.1	22.8 – 33.4	
TKI treatment			
No	16.2	13.0 – 19.5	0.279
Yes	25.3	18.9 – 31.8	

MWA microwave ablation, *TKI* Tyrosine kinase inhibitor

reduction in tumour burden led to the survival benefit. ^{18}F-Fluorodeoxyglucose positron emission tomography (FDG PET) can be used to assess the prognostic value of a reduction in tumour burden. It is has been demonstrated in nonsurgical NSCLC patients that total metabolic tumour volume, total lesion glycolysis and standardized uptake value can be used as prognostic factors for OS independent of clinical stage [32]. In a study of patients with NSCLC treated with MWA, PET/CT scans revealed no residual FDG activity in the ablated tumour sites when complete ablation was achieved [33]. Other previously verified prognostic factors such as gender, age and pathology have also been found to be associated with

Table 6 Multivariate analysis of overall survival

Variable	OR	95 %CI	*p* value
Gender	3.631	0.627 – 21.013	0.150
Age	6.987	1.288 – 37.914	0.024
Smoking history	0.631	0.076 – 5.230	0.670
Pathology	30.370	3.604 – 255.919	0.002
EGFR mutation	2.930	0.451 – 19.306	0.259
Response to MWA	0.201	0.065 – 0.623	0.005

MWA Microwave ablation

survival [17–21, 34, 35]. Patients aged less than 60 years had longer OS than patients aged 60 years or more.

In conclusion, the *EGFR* status was not related to the outcomes of MWA treatment in NSCLC, but the response to MWA was a predictor of survival. However, owing to the small sample size, this conclusion needs verification.

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