Prevalence of Epidermal Growth Factors Receptors in Iraqi Patients with Non-Small Cell Lung Cancer

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Abstract

Background: Cancers, including lung cancer, are often targets for tyrosine kinase inhibitors. The prevalence of EGFR mutations is known to vary by ethnicity, with higher rates in East Asian populations. Limited data exist on EGFR mutations in non-small cell lung cancer patients from Iraq.

Objective: To assess the prevalence of EGFR mutations in non-small cell lung cancer patients in Iraq and compare the mutation rates across age groups, sex, and cancer subtypes.

Methods: A cohort of 430 confirmed lung carcinoma cases from southern Iraq (2016-2023) was analyzed. DNA was extracted from paraffin-embedded samples, and EGFR mutations in exons 18, 19, 20, and 21 were detected using real-time PCR. The prevalence of EGFR mutations was stratified by age, sex, and cancer subtype.

Results: EGFR mutations were detected in 35.3% of the total cohort (152/430). The most frequent mutations were found in exon 19 (53.9%), followed by exon 21 (19.7%) and exon 18 (11.8%). Combined mutations in occurred at 9.2%. The prevalence of EGFR mutations was significantly higher in the 60-69 age group (42.1%) at p= 0.037*, followed by the 70-79 group (20.4%) and 50-59 group (18.4%). There was no significant sex difference p=0.2*, with mutation rates in males at 51.97% and females at 48.03%. Adenocarcinoma was the predominant cancer subtype (95.3%) associated with EGFR mutations.

Conclusion: EGFR mutations are prevalent in 35.3% of Iraqi lung cancer patients, a rate comparable to East Asian populations and higher than in other Middle Eastern countries. This highlights the need for further research into genetic predispositions and targeted therapies in Iraq. Understanding the high prevalence could guide treatment decisions and improve survival rates among Iraqi lung cancer patients.

Keywords: EGFR, Exon 18, 19, 20, 21, Adenocarcinoma, Basra, Lung Cancer

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Introduction

Epidermal Growth Factor Receptor (EGFR), also known as ErbB1/Her1, is a receptor with a dual role: the extracellular part binds to epidermal growth factor, while the intracellular part possesses tyrosine kinase activity [(1)]. From the outset, EGFR was linked to cell proliferation and DNA synthesis in human fibroblasts due to its binding and phosphorylation activity [(2)]. The protein was later described as a tyrosine kinase complex because of its ability to phosphorylate tyrosine residues in proteins [(3)]. EGFR, along with HER2, HER3, and HER4, belongs to the ErbB family of tyrosine kinase receptors [(4)]. In addition to mutations or aberrations in EGFR, overexpression of this receptor has been shown to promote cell proliferation in various cancers. In carcinomas and sarcomas, EGFR expression is upregulated up to 50 times in malignant and virally transformed cells, with receptor numbers

comparable to those in tumor cells [5]. Active mutations in EGFR are implicated in aggressive and fatal lung cancers, making EGFR an ideal target for therapy [6]. Furthermore, EGFR is overexpressed in the early stages of breast cancer, positioning it as a target for personalized medicine [7]. Mutations in the kinase domain of EGFR are associated with non-small cell lung cancer, maintaining its transformation effect while reducing its expression. Consequently, EGFR copy number and protein overexpression can serve as both a prognostic tool and a predictive factor for response to tyrosine kinase inhibition therapy [8]. Similarly, upregulated EGFR expression and mutations are linked to glioblastoma multiforme, a deadly brain cancer [9], and EGFR gene copy number and protein expression have been observed in breast carcinomas [10] and colorectal carcinomas, where gene amplification is found in about 60% of EGFR-overexpressed tumors [11]. A strong correlation exists between EGFR mutations, copy number alterations, and adenocarcinoma, with most mutations occurring in exons 18 to 21 on chromosome 7 [12]. EGFR mutations are present in approximately 23% of adenocarcinomas, with 71% involving deletions in exon 19 and 29% involving L858R mutations in exon 21. Additionally, increased EGFR copy numbers are found in 52% of mutated tumors, and this overexpression is independent of smoking [13]. EGFR mutations are also associated with squamous cell carcinoma (SCC), one of the most common cancers linked to tobacco use, with mutations identified in about 7% of head and neck SCC cases [14]. Although EGFR and its related proteins are well known for promoting cell proliferation and counteracting apoptosis, new signaling pathways are continually being discovered, indicating that much remains to be understood [15]. Globally, EGFR mutations are becoming more prevalent and vary significantly across different ethnic populations, ranging from 5% to 11% in Western Europe to over 30% in Asian populations [16, 17, 18, 19]. In the Middle East, EGFR mutation prevalence ranges from 11.3% in Lebanon to 29.7% in the Gulf region, with an overall prevalence of 21% across the Middle East and North Africa [20;21; 22]. Recent data suggest an EGFR mutation rate of about 15% in the Arab world, predominantly among women and non-smokers [23].

Aside from a few studies with small sample sizes, this study is the first to assess EGFR prevalence in a large cohort of Iraqi patients. Therefore, this study aims to determine the prevalence of EGFR mutations, the distribution of mutations across different age groups, and their association with sex in the Iraqi population.

Methods

Samples were collected between 2016 to 2023 which were diagnosed with primary non-small cell lung carcinoma by conventional histopathological examination. IRB Approval was obtained from the Institutional Review Board (IRB) of Basrah University. Hospitals Involved: Data were collected from Al-Sader Teaching Hospital, Basrah Teaching Hospital, and Private laboratory. Written informed consent was obtained from all patients or their legal guardians before inclusion in the study. Demographic Data Collected: Age, sex, and cancer subtype (adenocarcinoma, squamous cell carcinoma, poorly differentiated carcinoma, adenosquamous carcinoma) were recorded for each patient. All 430 samples were sent as Formalin Fixed Paraffin Embedded tissue blocks to a molecular pathology laboratory, for EGFR mutation analysis by preparing 3-4 sections taken with 10-micron thickness collected in a sterile microfuge tube, in aseptic conditions. DNA was extracted by Qiagen Kit Ref. No. 51306. DNA concentration was measured by quantiflor from Promega. The DNA was used as a template to detect mutations and/ or deletions in exons 18, 19, 20 and 21 by real-time PCR of QuantStudio 5 from Applied biosystem, using AmoyDx EGFR29 mutation detection kit (Cat. No 8.01.0134). Then the results were reported and the threshold was set and read according to the instructions of the manufacturer.

Results:

The results showed that out of 430 examined samples, 152 samples were positive for EGFR mutation with 35.3% (Figure 1).

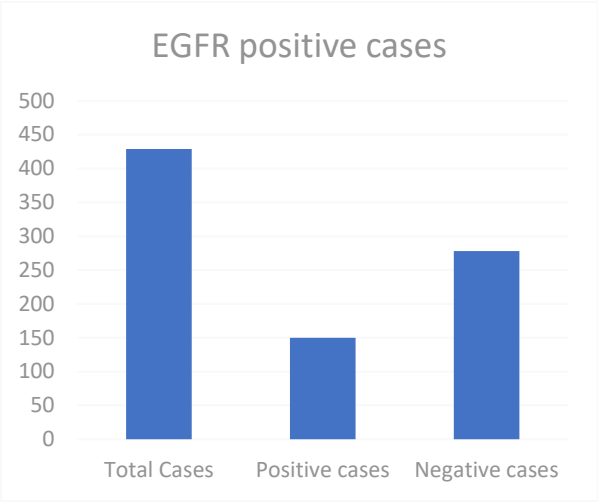

Figure 1: Prevalence of EGFR in the studies cohort

Mutation location in the gene sequence

In order to detect the mutation in which exon, the analysis showed that the top three mutations/deletion were found in 19, 21 and 18 with 53.9%, 19.7% and 11.8, respectively. Exon 19 was involved in the highest combined mutations, 19 and 21, 19 and 18 at a percentage of 2.65 (Table 1)

Table 1: Mutation / Deletion distribution in different exons of EGFR gene

Type of Mutation/Deletion	Number of Cases	Percentage
Exon 18	18	11.8%
Exon 19	82	53.9%
Exon 20	8	5.3%
Exon 21	30	19.7%
More Than One Mutation and /or Deletion	14	9.2%
Total	152	100%

Age groups and prevalence of EGFR mutations

To identify the risk factors for developing EGFR mutation, the age was categorized and percentages of EGFR according to the age were assessed. The results displayed that EGFR mutation was prevalent in the following order: 41.9%, 20.7%, 17.7%, 7.4%, 7.2% or 5.1% in age groups 60-69, 70-79, 50-59, 40-49, >=80 or 30-39 years old respectively (Table 2).

Table 2: EGFR mutations in different age groups (p= 0.037*)

Age	EGFR				Total	
	Positive		Negative			
	No.	%	No.	%	No.	%
30-39	8	5.3	14	5	22	5.1
40-49	13	8.6	19	6.8	32	7.4
50-59	28	18.4	48	17.3	76	17.7
60-69	64	42.1	116	41.7	180	41.9
70-79	31	20.4	58	20.9	89	20.7
>80	8	5.3	23	8.3	31	7.2
Total	152	35.2	278	64.7	430	100

`Prevalence of EGFR in both sexes

To identify the risk factors for developing EGFR mutation, percentages of EGFR according to sex were detected. The results showed that percentages of EGFR in males and females were comparable at 51.97% and 48.03% respectively (Table 3).

Table 3: EGFR distribution in male and female

Sex	EGFR			
	Positive		Negative	
	No.	%	No.	%
Male	79	51.97	169	60.79
Female	73	48.03	109	39.21
Total	152	100	278	100

EGFR mutation in both sexes according to the age group

In order to correlate the relationship between the age group and sex in the study, the ratios were separately calculated in each age group. The results showed that female percentages were slightly higher in age groups 60-69 and 40-49 (figure 2).

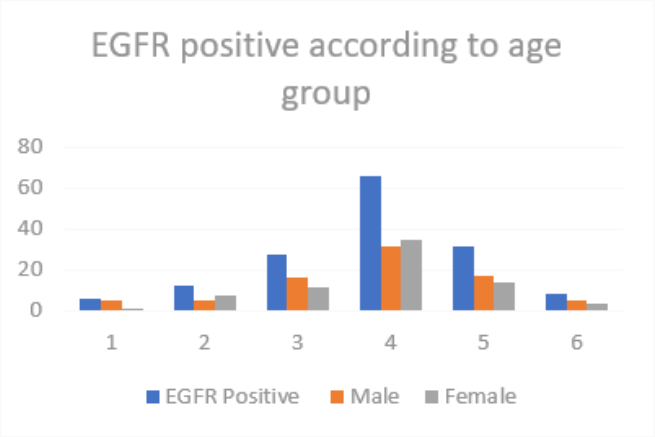

Figure 2: EGFR distribution in both sexes according to the age group

Discussion:

The results showed that the prevalence of EGFR mutation is about 35.3% in the study cohort. The results are consistent with a study conducted to estimate the prevalence of EGFR which revealed the latter prevalence of 33% of Non-Small Cell Lung Cancer (NSCLC) patients globally [(24)]. The same study found that in countries in Asia including India, China, Japan and Taiwan where EGFR prevalence increased to 42% with higher for women, non-smokers and adenocarcinoma. In fact, the results are consistent with the pooled prevalence overall in the world but it is slightly less compared with countries like China but two-fold higher compared to Europe [(25)]. However, the results are much lower with a study conducted in Europe where the EGFR mutation prevalence didn't exceed 12% with women sex as a risk factor [(26)]. Compared to the Middle East percentages, Iraq showed the highest where it was more than two-fold compared to Lebanon and slightly higher than the Gulf Area [(21)]. Compared to Iraqi studies, the results are higher than those reported by [(27)] who showed the presence of EGFR in 27% of NSCLC patients. The difference could be attributed to the fact that the cohort included in this study is about four times.

The top three mutations/deletions were found in exons 19, 21 and 18 with 53.9%, 19.7% and 11.8% respectively. This agrees with [(28)], as a deletion in exon 19 was the most abundant followed by mutations in exon 21 at 54.5% or 36.36% respectively. In agreement with [(27)], an advantage for exon 19 mutations followed by L858R in exon 21 or multiple mutations in 20 and 19 together at percentages of 65.8%, 26.3% or 5.3% was revealed.

The results displayed that EGFR mutation was prevalent at 41.9%, 20.7%, 17.7%, 7.4%, 7.2% or 5.1% in age groups 60-69, 70-79, 50-59, 40-49, >=80 or 30-39 years old respectively. The results are in line with the known ageing risk factor. Risk factors for EGFR mutation in lung cancer include ageing as it was higher in their 50s, 60s, 70s and older than 80 years compared to the patients in their 40s.

The results showed that percentages of EGFR in males and females were comparable at 51.97% and 48.03% respectively. In fact, these results contradict global and regional data since studies revealed that women not smoking is a risk factor for EGFR mutation NSCLC [(29)]. Many risk factors could be suggested to explain such differences. Familial history is considered a substantial risk factor for EGFR mutations [(30)]. Dietary habits are significant

factors in EGFR lung cancer, the higher consumption of vegetables correlated with lower EGFR+ Lung cancer. In contrast, higher EGFR+ was reported with higher consumption of meat [30]. In Iraq, men tend to consume higher quantities of meat. Finally, genome-wide association studies showed that there are loci in the patient genome that are sensitive to EGFR mutations [30]. In conclusion, the study highlights the importance of conducting more research to find the underlying reason for this surge in EGFR mutation and the different sex ratios and to draw the attention of policymakers to adopt targeted therapy to increase the survival rates of the diagnosed people. Also, genome sequencing data are required since the Iraq population showed a higher mutational EGFR rate comparable to the Asian population It has not been elucidated yet why Asian people show a higher EGFR mutational rate compared to other ethnic groups. It could be due to the presence of CA simple sequence repeat 1 (CA-SSR1) which contains repeats from 14 to 21 of CA nucleotides with high sensitivity to be mutated (Nomura et al., 2007).

Conclusion: Larger cohort showed the alarming prevalence of EGFR in the Iraqi population, 35% mutation rate makes Iraq the highest in the Arabic and Middle East region and comparable to East Asian countries. The study highlights the importance of conducting more research to find the underlying reason for this surge in EGFR mutation and to draw the attention of policymakers to adopt the targeted therapy to increase the survival rates of the diagnosed people.

Limitations: It was not possible to study the relationship between causes and results due to the limitations in facilities such as whole genome sequencing. The cohort under this study represents only the south of Iraq and a larger picture is needed for future studies.

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انتشار مستقبلات عوامل النمو البشري لدى المرضى العراقيين المصابين بسرطان الرئة غير صغير الخلايا

الخلفية: تُعتبر السرطانات، بما في ذلك سرطان الرئة، أهدافًا شائعة لمثبطات التيروزين كيناز. يُعرف أن انتشار طفرات مستقبلات عوامل النمو البشري (EGFR) يختلف حسب العرق، مع معدلات أعلى بين سكان شرق آسيا. هناك بيانات محدودة حول طفرات EGFR لدى مرضى سرطان الرئة غير صغير الخلايا في العراق.

الأهداف: تقييم انتشار طفرات EGFR بين مرضى سرطان الرئة غير صغير الخلايا في العراق ومقارنة معدلات الطفرات بين الفئات العمرية، الجنس، وأنواع السرطان.

الطرق: تم تحليل مجموعة مكونة من ٤٣٠ حالة مؤكدة لسرطان الرئة من جنوب العراق (٢٠١٦-٢٠٢٣). تم استخراج الحمض النووي من عينات مدمجة بالبارافين، وتم الكشف عن طفرات EGFR في الإكسونات ١٨، ١٩، ٢٠، و٢١ باستخدام تقنية تفاعل البوليميراز المتسلسل في الوقت الحقيقي (Real-time PCR). تم تصنيف انتشار الطفرات حسب العمر، الجنس، ونوع السرطان.

النتائج: تم اكتشاف طفرات EGFR في ٣٥,٣٪ من العينة الكلية (١٥٢/٤٣٠). كانت الطفرات الأكثر شيوعًا في الإكسون ١٩ (٥٣,٩٪)، تليها الإكسون ٢١ (١٩,٧٪) والإكسون ١٨ (١١,٨٪). ظهرت الطفرات المشتركة بنسبة ٩,٢٪. كان انتشار طفرات EGFR أعلى بشكل ملحوظ في الفئة العمرية ٦٠-٦٩ سنة (٤٢,١٪) عند مستوى دلالة p=0.037*، تليها الفئة ٧٠-٧٩ سنة (٢٠,٤٪) والفئة ٥٠-٥٩ سنة (١٨,٤٪). لم يكن هناك فرق دال إحصائيًا بين الجنسين (p=0.2*)، حيث كانت معدلات الطفرات في الذكور ٥١,٩٧٪ وفي الإناث ٤٨,٠٣٪. كان النوع الأكثر ارتباطًا بطفرات EGFR هو سرطان الغدة (٩٥,٣٪).

الخلاصة: تنتشر طفرات EGFR بنسبة ٣٥,٣٪ بين مرضى سرطان الرئة في العراق، وهي نسبة تقارن مع سكان شرق آسيا وأعلى من بعض دول الشرق الأوسط. تسلط هذه النتائج الضوء على الحاجة لمزيد من الأبحاث حول الاستعدادات الوراثية والعلاجات المستهدفة في العراق. فهم الانتشار العالي للطفرات يمكن أن يساعد في توجيه القرارات العلاجية وتحسين معدلات البقاء على قيد الحياة بين مرضى سرطان الرئة العراقيين.

الكلمات المفتاحية: EGFR، إكسون ١٨، ١٩، ٢٠، ٢١، سرطان الغدة، البصرة، سرطان الرئة.