RETROSPECTIVE MULTICENTER STUDY IN NON SMALL CELL LUNG CANCER (NSCLC) PATIENTS WITH EPIDERMAL GROWTH FACTOR RECEPTOR (EGFR) ACTIVATING MUTATION TREATED FIRST-LINE TYROSINE KINASE INHIBITOR (TKI): EVALUATION OF PROGRESSION ACCORDING TO RECIST, THERAPEUTIC APPROACH AND ITS EFFECT

J.B. Auliac1, J. C. Fournier2, C. Audigier Valette2, A. M. Perot3, E. Tsiek2, I. Monnet4, C. Decroisette Phan van Ho5, S. Bota Ouchlif6, R. Corre7, G. Le Garff8, F. Fournel9, N. Blaise10, R. Lamy11, A. Vergnenegre12, D. Arpin13, B. Marin14, L. Greillier15, R. Gervais16

1Pulmonology Department, hospital Quesnay, Mantes la Jolie, FRANCE
2Multidisciplinary Oncology and Therapeutic Innovations, Assistance publique-hôpitaux de Marseille, Marseille, FRANCE
3Oncology, C.H.T.S. Font-Péru Centre Hospitalier Intercommunal Toulon la Seyne sur Mer, Toulon, FRANCE
4Medical Oncology, Centre Léon Bérard, Lyon, FRANCE
5Pulmonology Department, CHU Saint-Jean, Mulhouse, FRANCE
6Pulmonology, Centre Hospitalier Intercommunal de Creteil, Creteil, FRANCE
7Centre Hospitalier de La Région d’annecy-chra (Pringy), Centre Hospitalier de la Région d’Annecy-CHRA (Pringy), Pringy, FRANCE
8Clinique Pneumologique, CHU Hôpitaux de Rouen-Charles Nicolle, Rouen, FRANCE
9Pulmonology Department, CHU Pontchaillou, Rennes, FRANCE
10Pulmonology Department, hospital Saint Brieux, Saint Brieux, FRANCE
11Department d’oncologie Médicale, Institut de Cancérologie Lucien Neuwirth, Saint-Priest en Jarez, FRANCE
12Pneumologie, C.H.U. Angers, Angers, FRANCE
13Pneumology Department, Centre Hospitalier de Bretagne Sud, Lorient, FRANCE
14Service de Pneumologie, Hospital du ChateauCHU Dupuytren, Limoges, FRANCE
15Pulmonology, Centre Hospitalier de Macon, Macon, FRANCE
16Unité Fonctionnelle de Recherche Clinique et Biostatistique, Facultés de Médecine et de Pharmacie, ch. lémages, Limoges, FRANCE
17Multidisciplinary Oncology & Therapeutic Innovations, Aix-Marseille Univ, Assistance Publique-Hôpitaux de Marseille, Marseille, FRANCE
18Oncology Department, Centre Francois Baclesse, Caen, FRANCE

NSCLC, metastatic

Aim: Background: EGFR-TKI are a standard treatment for patients (pts) with NSCLC harboring activating EGFR mutations. All pts develop acquired resistance. At progression, the standard treatment is chemotherapy. Retrospective studies suggest that continuous use of EGFR-TKI beyond progressive disease (PD) may benefit some pts.

Objective: The purpose of our retrospective multicentric study is to determine the frequency of continuation of EGFR-TKIs beyond RECIST-PD, and investigate the association of pts and disease characteristics with continuation of EGFR-TKIs at progression.

Methods: Main inclusion criteria were: pts with NSCLC and activating EGFR mutations, EGFR-TKIs as their initial systemic therapy received between January 2010 and July 2012. Measurable lesion according to RECIST 1.1, acquired resistance to EGFR-TKI according to Jackman’s criteria. Following data were collected: demographic and clinical data, Progression free survival (PFS), Overall Survival (OS), mutational status, mode of progression, therapeutic approach at PD. A comparison of clinical data and outcome of pts receiving EGFR-TKI beyond PD (group 1) versus discontinuing EGFR-TKI at PD (group 2) was made.

Results: 133 pts were recruited in 29 centers: age 69 ± 12.7 years, female 67.6%, EGFR mutation exon 19/21/other: 65.4% / 30.8% / 3.8%, never smokers 68.5%, PS 0/1: 80.5%. First line treatment: gefitinib 77.4%, erlotinib 21.8%, 46.6% pts continued EGFR-TKI beyond RECIST-PD (25.6% EGFR-TKI alone, 15% EGFR-TKI combined with local treatment). 59.3% pts changed treatment (39.8% chemotherapy, 7.5% combination chemotherapy + EGFR-TKI, 12% BSC). Median PFS was 9.4 (CI95% :8-10.9) months and median OS was 21.6 (CI95%18.7-25.8) months in the entire population. In group 1 and 2, m PFS was 10.1 (CI95%:7.7-12.3) and 8.7 (CI 95%:7.5-10.9) (p = 0.34) months and m OS was 23 and 20.4 months respectively (p = 0.08). All comparative data between groups 1 and 2, univariate and mutivariate analysis will be presented.

Conclusions: This large retrospective study confirms that, in some circumstances, continuous use of EGFR-TKI beyond PD does not hamper OS and should be considered. Prospective studies will help to determine which patients benefit more this strategy. Clinical trial information: Supported by an academic grant from Boehringer Ingelheim, Hoffman Roche.

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