Afatinib for the treatment of NSCLC with uncommon EGFR mutations: an updated database of 1023 cases

Introduction
- Around 7–23% of patients with EGFR-r+ NSCLC have tumours harbouring uncommon mutations (non-Del19/L858R); up to 25% of EGFR-r+ tumours harbour compound mutations (>1 EGFR mutation)
- There is a lack of clinical data assessing the activity of EGFR TKIs in patients with NSCLC harbouring uncommon EGFR mutations
- Increased use of NGS for mutation detection and plasma-based assays will increase identification of uncommon EGFR mutations in everyday clinical practice
- Previously, we developed a database of 693 patients with NSCLC and uncommon ex20ins

Methods
- Aims
  1) Investigate afatinib as a treatment for patients with uncommon EGFR mutations
  2) Update the uncommon mutations database with new findings, with a focus on individual ex20ins and other uncommon mutations
- Patients categorised into four groups
  - TKI naïve (N=587), TKI pretreated (N=425)
  - Comorbidities: Asian / non-Asian, % 62.8 / 37.2
- Key endpoints
  - ORR, objective response rate
  - TTF, time to treatment failure
- Patients with compound mutations were also analysed

Results
- 1023 patients were identified, with most patients treated in clinical studies/compassionate use programmes
- 587 TKI naïve (59.0%)
- 425 EGFR TKI pretreated (41.0%)

Results (cont’d)

Key findings and conclusions
- Data are in line with previously published data
- Afatinib demonstrated strong activity against major uncommon, compound, and ‘other’ uncommon mutations
- Afatinib showed excellent activity against E709X and L747X mutations in TKI-naïve patients
- Afatinib demonstrated activity against certain exon 20 insertions at residues A763, M766, N771, and V769
- Afatinib showed activity against the osimertinib resistance mutations G724S, L718Q, L718V, and C797S

Results (cont’d)

Evidence of activity against certain Exon 20 insertions at residues A763, M766, N771, and V769

References
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#1212P

Presented at the European Society for Medical Oncology (ESMO) Congress, Virtual Format, 16-21 September 2021

This study was funded by Boehringer Ingelheim. The authors were fully responsible for all content and editorial decisions, were involved at all stages of poster development and have approved the final version.

Medical writing support for the development of this poster, under the direction of the authors, was provided by Paige Phillips, MS of Ashfield MedComms, an Ashfield Health company, and funded by Boehringer Ingelheim. To access Dr Yang’s conflicts of interests please scan the QR code