Differences Among Specific EGFR Mutations

TRANSCRIPT & FIGURES
Within the overview of EGFR mutations, there's several different types – the two most common are EGFR exon 19 deletion and EGFR L858r; together, these represent more than 80% of EGFR mutations. In addition to these two common types, there are some uncommon types, but some of these are related to responsiveness to EGFR tyrosine-kinase inhibitors, so rarer ones like G719A, L861, these are rare, but we have pretty good evidence that they lead to response to drugs, just as those patients who have L858r or exon 19 deletion.

There are patients who have exon 20 insertions – in general, EGFR exon 20 insertions are associated with resistance to EGFR tyrosine-kinase inhibitors like erlotinib, gefitinib and afatinib, and so, as a consequence, that's not our first line therapy for those patients with EGFR exon 20 insertions.

Going back to the two most common EGFR mutations, exon 19 deletion and EGFR L858r, since these are the most common ones, we have more data on patient outcomes for these two mutations. When we look at a broad variety of data, typically with afatinib, we see that afatinib may actually be more effective for those patients with EGFR exon 19 deletions than it is for patients with EGFR L858r.

There has been similar data reported for patients treated with gefitinib and erlotinib as well. Though the data is not quite as clear cut, it does seem that those drugs also work a bit better for patients with EGFR exon 19 deletions.

Now what the consequence of those differences is, is quite controversial. I think that, in general, I still recommend treatment with an EGFR tyrosine-
kinase inhibitor – and I don’t prefer one or the other, for patients with EGFR exon 19 deletions, EGFR L858r, as well as the rarer ones like G719A or L861Q.
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