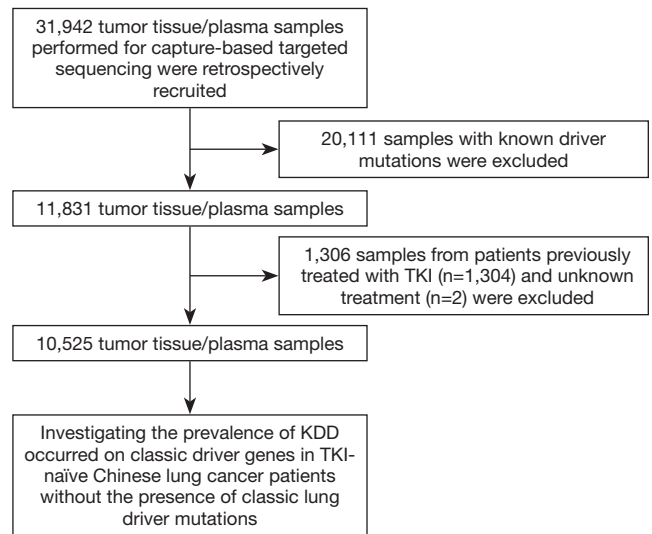


## Supplementary

**Table S1** Driver mutations of genes

Driver gene	Mutations
<i>EGFR</i>	Exon 19 deletion, L858, L861, G719, S768, E709, R776
<i>KRAS</i>	G12, G13, Q61, A146
<i>BRAF</i>	V600, G469, G466
<i>ERBB2</i>	amplification, exon 20 insertion, S310
<i>ALK</i>	fusion
<i>RET</i>	fusion
<i>ROS1</i>	fusion
<i>MET</i>	amplification, exon 14 skipping mutations

EGFR, epidermal growth factor receptor; KRAS, KRAS proto-oncogene, GTPase; BRAF, B-Raf proto-oncogene, serine/threonine kinase; ERBB2, erb-b2 receptor tyrosine kinase 2; ALK, ALK (anaplastic lymphoma kinase) receptor tyrosine kinase; RET, ret proto-oncogene; ROS1, ROS proto-oncogene 1, receptor tyrosine kinase; MET, MET (hepatocyte growth factor receptor gene) proto-oncogene, receptor tyrosine kinase.



**Figure 1** Schematic design of our study. A total of 10,525 tumor tissue/plasma samples performed for capture-based were retrospectively enrolled for investigating the prevalence of KDD occurring on classic driver genes in Chinese lung cancer patients without the presence of classic lung cancer driver mutations. KDD, kinase domain duplication.