exon 2 are common (15-18%). Patients with any *RAS* family mutations show no evident progression free survival (PFS) or overall survival (OS) benefit from anti-EGFR treatment.

This case challenges the CRC incontrovertibility that within a pathway, only a single gene might mutate. A diagnosis of CRC indicative of HNPCC/Lynch Syndrome with co-mutations in KRAS and NRAS genes is extremely rare, but not completely unknown. Grellety et al. (2016) reported 2 patients with a co-mutation of *KRAS/NRAS* in both primary, and metastatic tumours.[19] These concomitant mutations arose early during carcinogenesis, and were maintained when the primary tumour metastasized. The clinical implications of such co-mutations are not well understood. There's incomplete data on improving objective response rates on combining EGFR inhibitors with different chemotherapy backbones, and between *RAS* mutations. Effective targeted therapeutics of oncogenic *RAS*-driven tumors remains a major challenge; this patient expired within one year of CRC diagnosis.

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