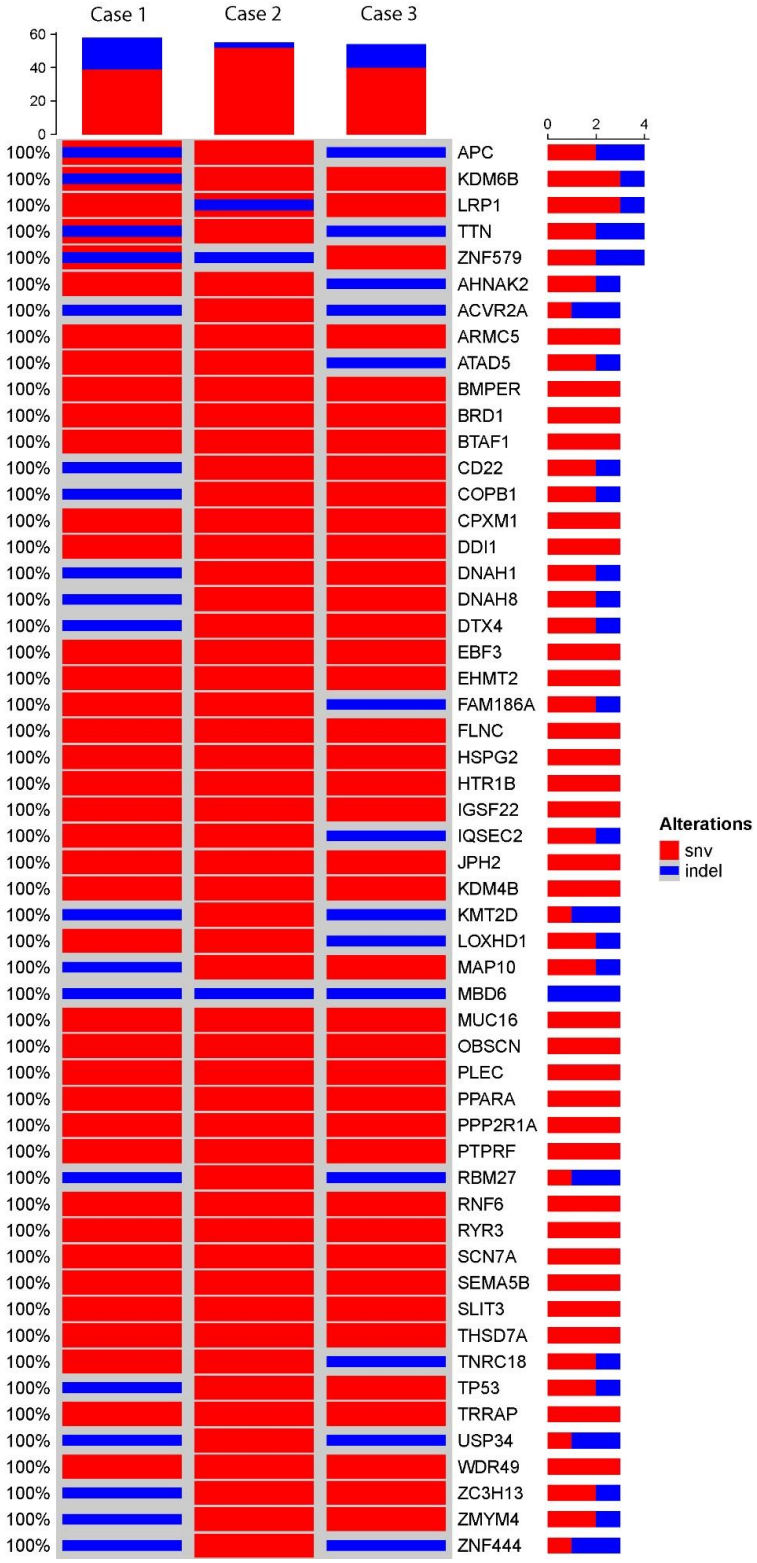
Case 1
Case 2
Case 3
60
40
20
0
0
2
4
100% APC
100% KDM6B
100% LRP1
100% TTN
100% ZNF579
100% AHNAK2
100% ACVR2A
100% ARMC5
100% ATAD5
100% BMPER
100% BRD1
100% BTAF1
100% CD22
100% COPB1
100% CPXM1
100% DDI1
100% DNAH1
100% DNAH8
100% DTX4
100% EBF3
100% EHMT2
100% FAM186A
100% FLNC
100% HSPG2
100% HTR1B
100% IGSF22
100% IQSEC2
100% JPH2
100% KDM4B
100% KMT2D
100% LOXHD1
100% MAP10
100% MBD6
100% MUC16
100% OBSCN
100% PLEC
100% PPARA
100% PPP2R1A
100% PTPRF
100% RBM27
100% RNF6
100% RYR3
100% SCN7A
100% SEMA5B
100% SLIT3
100% THSD7A
100% TNRC18
100% TP53
100% TRRAP
100% USP34
100% WDR49
100% ZC3H13
100% ZMYM4
100% ZNF444
Alterations
snv
indel

Supplementary Figure 1: Oncoprint plot for 54 non-synonymous changes common for all three cases, each case shown by one lane. The upper bar plot shows the total number of alterations detected in each case. The right panel shows relative prevalence of alterations in each gene (for example, *APC* had both indel and SNVs for case #1, SNVs for case #2, indel for case #3 – thus total of 4 alterations is shown at the right barplot). All three cases (as noted in Table 1) were MMR-deficient, confirmed by immunohistochemistry, and MSI-high according to whole-exome sequencing of tumor tissue. Importantly, all cases also had high tumor mutational burden: 43, 351, and 31 mutations/Mb for cases #1, #2, and #3, respectively. With a wide spectrum of mutations found, 54 non-synonymous mutations were common for all three cases including genes commonly mutated in CRC, such as *APC* and *TP53*. Cases #1 and #2 had missense mutations in both *POLE* and *POLD1* genes. Case #2 also bore mutations *KRAS A59T* and *BRAF* P192S, while case #3 had *BRAF* V600E mutation. The tumor mutational burden (table 1) not surprisingly is different across different platforms.