Figure S1. Selected results of Sanger sequencing validation for VHL indels. Each panel indicated an individual sample, which was labeled as NTCG followed by three digits. (A) Mutation at the position 10183748 of chromosome 3 resulted in a fs mutation of the 78th amino acid (AA) Asparagine (N). (B) Mutation at the position 10183699 of chromosome 3 resulted in a fs mutation of the 57th AA Glycine (G). (C) Mutation at the position 10183812 of chromosome 3 resulted in a fs mutation of the 94th AA Glutamate (E). (D) Mutation at the position 10183860 of chromosome 3 resulted in a fs mutation of the 110th AA Histidine (H). (E) Mutation at the position 10183692 of chromosome 3 resulted in a fs mutation of the 55th AA Glutamate (E). (F) Mutation at the position 10188232 of chromosome 3 resulted in a fs mutation of the 126th AA Aspartate (D). (G) Mutation at the position 10183699 of chromosome 3 resulted in a fs mutation of the 57th AA Glycine (G). (H) Mutation at the position 10183841 of chromosome 3 resulted in a fs mutation of the 111st AA Aspartate (D). fs, frameshift.
Figure S2. Selected results of Sanger sequencing validation for *VHL* single nucleotide variation. Each panel indicated an individual sample, which was labeled as renal cell carcinoma followed by two digits. (A) Mutation at the position 10188240 of chromosome 3 resulted in the 128th amino acid (AA) change from Leucine (L) to Proline (P). (B) Two point mutations were found in this sample. Mutation at the position 10183807 (#) of chromosome 3 resulted in the 92nd AA change from Aspartate (D) to Glutamate (E). Mutation at the position 10183808 (##) of chromosome 3 resulted in the 93rd AA change from Glycine (G) to Arginine (R). (C) Mutation at the position 10183734 of chromosome 3 resulted in a stop codon (*) of the 68th AA Aspartate (D).
Figure S3. Selected results of Sanger sequencing validation for protein polybromo-1 indels. Each panel indicated an individual sample, which was labeled as renal cell carcinoma followed by two digits and NTCG followed by three digits. (A) Mutation at the position 52620503 of chromosome 3 resulted in a fs mutation of the 1,096th ammino acid (AA) Arginine (R). (B) Mutation at the position 52621420 of chromosome 3 resulted in a fs mutation of the 559th AA Tyrosine (Y). (C) Mutation at the position 52621387 of chromosome 3 resulted in a fs mutation of the 570th AA Isoleucine (I). (D) Mutation at the position 52621548 of chromosome 3 resulted in a fs mutation of the 516th AA Lysine (K). (E) Mutation at the position 52621397 of chromosome 3 resulted in a fs mutation of the 1,032nd AA Leucine (L). (F) Mutation at the position 52613121 of chromosome 3 resulted in a fs mutation of the 1,159th AA Lysine (K). (G) Mutation at the position 52582122 of chromosome 3 resulted in a fs mutation of the 1,676th AA Aspartate (D). (H) Mutation at the position 52702537 of chromosome 3 resulted in a fs mutation of the 120th AA Phenylalanine (F). (I) Mutation at the position 52613108 of chromosome 3 resulted in a fs mutation of the 1165th AA Phenylalanine (F). fs, frameshift.
Figure S4. Selected results of Sanger sequencing validation for protein polybromo-1 single nucleotide variation. Each panel indicated an individual sample, which was labeled as RCC followed by two digits or NTCG followed by three digits. (A) Mutation at the position 52613116 of chromosome 3 resulted in the 1163rd amino acid (AA) change from Cysteine (C) Arginine (R). (B) Two point mutations were found in this sample. Mutation at the position 52682447 (#) of chromosome 3 resulted in a stop codon of the 242nd AA Tyrosine (Y). Mutation at the position 52682449 (##) of chromosome 3 resulted in the 242nd AA change from Tyrosine (Y) to Histidine (H). (C) Mutation at the position 52682443 of chromosome 3 resulted in the 244th AA change from Aspartate (D) to Glycine (G). (D) Mutation at the position 52682395 of chromosome 3 resulted in a stop codon of the 260th AA Lysine (K). (E) Mutation at the position 52702581 of chromosome 3 resulted in the 106th AA change from Tyrosine (Y) to Phenylalanine (F).
Figure S5. Selected results of Sanger sequencing validation for *BAP1* indels. Each panel indicated an individual sample, which was labeled as RCC followed by two digits. (A) Mutation at the position 52439226 of chromosome 3 resulted in a fs mutation of the 339th ammino acid (AA) Proline (P). (B) Mutation at the position 52436411 of chromosome 3 resulted in a fs mutation of the 695th AA Asparagine (N). (C) Mutation at the position 52442511 of chromosome 3 resulted in a fs mutation of the 78th AA Asparagine (N). fs, frameshift.