Christenson, Alan

MRN: 16743072
Description: 81 year old male

Progress Notes Encounter Date: 2/8/2018 7:45 AM

Status: Unsigned

Reyes, Rochelle, PA

Physician Assistant

Thank you for reaching out the to the Tumor Genomics clinical trial team; we have reviewed your patient's genomic report.

IHC

NGS

on tumor from tracheal specimen collected Nov2017

TUMOR TYPE: LUNG SQUAMOUS CELL CARCINOMA (SCC)

Genomic Alterations Identified†

RAF1 rearrangement intron 6
AKT2 amplification – equivocal*
ARID1A Q761fs*72
SPTA1 R1694H, splice site 812+1G>T
TP53 M246V, P278S

Additional Findings‡

Microsatellite status MS-Stable
Tumor Mutation Burden TMB-Intermediate; 9 Muts/Mb

VARIANTS OF UNKNOWN SIGNIFICANCE

Note: One or more variants of unknown significance (VUS) were detected in this patient's tumor. These variants may not have been adequately characterized in the scientific literature at the time this report was issued, and/or the genomic context of these alterations makes their significance unclear. We choose to include them here in the event that they become clinically meaningful in the future.

EPHA3 E481*	*FLT4* E58K	*FOXL2* A301E	*HSP90AA1* K580del	*LRP1B* R799L	*PARK2* A38T
POLD1 H491Y	*PRKDC* D4109H	*SDHA* amplification	*SPEN* S2306del		

Through the Stanford Precision Oncology clinic, the **MATCH clinical trial** is investigating *** in solid tumors with pathogenic variants of NF2, MET, SMO/PTCH1, TSC1/2, GNAQ/GNA11, EGFR, cKIT, mTOR, ROS1, ALK, NTRK, DDR2, or BRAF V600. We are anticipating the opening of an arm investigating *** in solid tumors with pathogenic variants of PIK3CA, PTEN, FGFR, TP53 + MYC, AKT, non-v600 BRAF. NCT02465060. The coordinator is Ginna Freehling, ginfree@stanford.edu.

copanlisib in PIK3CA, PTEN loss by IHC, PTEN-mut + present by IHC

erdafinib in FGFR-amp, FGFR-mut or fusion

Dr Josh Gruber and Dr Melinda Telli have the **Beyond BRCA clinical trial** which is investigating **talazoparib, a PARP-inhibitor,** in non-breast solid tumors with deleterious mutations implicated in the Homologous Repair (HR) pathway. Specifically, ATM, ATR, BARD1, BRIP1, FANC, MRE11, NBN, PALB2, PTEN, RAD50, RAD51C, RAD51D. NCT02401347. The coordinator is Oshra Sedan, osedan@stanford.edu.

Through the Stanford Precision Oncology clinic, the **MyPathway clinical trial** is investigating targeted therapy in solid tumors with pathogenic variants of HER2, EGFR, BRAF, ALK or the Hedgehog pathway SMO or PTCH1. This patient may be eligible to receive ***.
At Stanford, the **MyPathway clinical trial** is investigating **atezolizumab immunotherapy** in solid tumors with high mutational burden or pathogenic variants of DNA repair pathway: ABL1, ATM, ATR, BARD1, BLM, BRCA1, BRCA2, BRIP1, CUL4A/B C11orf30 (ESMY), CHEK1/2, ERCC4, FAM175A, FANC-, GEN1, MRE11A, MLH1, MSH2, MSH6, MUTYH, PMS2, NBN, PALB2, PARP1/2/3/4, PRKDC, PRKDC, RAD- , RPA1, TP53BP1, XRCC2, XRCC3. NCT02465060. The coordinator is Haowu1@stanford.edu.

The Phase I Clinical Research Program has some genomic trials. The PI is Dr Shivaani Kummar. For consideration, send an Epic message to "Clinical Research Phase 1" department or email DLPhase1Team@stanfordhealthcare.org:
- **LOXO-10**, an oral **TRK Inhibitor** in solid tumors with NTRK fusion.
- **PLX8394, + cobicistat,** oral **RAS/RAF/MEK/ERK pathway inhibitors** in solid tumors with activating BRAF mutation.
- **ADCT-502**, an **anti-HER2 antibody- drug conjugate**, in solid tumors with HER2 expression of IHC ?1+ or HER2 amplified/mutated by FISH and/or NGS

Locally through the Sutter Cancer Research Consortium, the **TAPUR** clinical trial is investigating targeted therapies in tumors with actionable variants of VEGFR, BCR-ABL, SRC, LYN, LCK, ALK, ROS1, CDKN2A, CDK4, CDK6, CSF1R, PDGFR, VEGFR, mTOR, TSC, EGFR, ERBB2, BRAFV600E, PTCH1, KRAS, NRAS, BRAF, KIT, PDGF-beta, RAF1, BRCA1, BRCA2, ATM, POLE, POLD1, high mutational load >9mut/Mb. NCT02693535. The coordinator who will connect patients to TAPUR-participating oncologists is Peter Gasper, peter@cpmcri.org.

If the patient is willing to travel, the **MPACT clinical trial** is investigating **WEE1 inhibitor, MK-1775**, and **PARP inhibitor, veliparib,** in advanced solid tumors with pathogenic variants in genes in the DNA repair pathway. Specifically, ATM, ATR, ERCC1, MLH1, MSH2, NBN, PARP1/2, RAD51, TP53.
mTOR inhibitor, everolimus in advanced solid tumors with pathogenic variants in genes in the PI3K or RAS/RAF/MEK pathways. Specifically, AKT1, FBXW7, MTOR, PIK3CA, PTEN
MEK-inhibitor, trametinib in advanced solid tumors with deficient RAS/RAF/MEK pathway: BRAF, HRAS, KRAS, NRAS, NF1
NCT01827384

We do not have treatment recommendations at this time.***
We will review this case with the Molecular Tumor Board.***

Ginna Freehling, CRC
Rochelle Reyes, PA-C
Tumor Genomics Precision Oncology
Stanford Cancer Center
Pager: 650-723-8222 # 25938

Documentation Only Encounter on 2/8/2018

This note has been shared with the patient but is not viewable in MyHealth. Reasons may include one or more (note has not been cosigned, encounter has not been closed, etc.)