**Supplemental Figure Legends**

**Supplemental Figure 1.** CNS tumor cells with BRAF<sup>V600E</sup> show high rates of induced autophagy by microscopy. (A) Cells expressing mCh-GFP-LC3 were exposed to either standard media or starvation EBSS media for 4 hours, with and without the presence of 10μM CQ to inhibit the degradation of LC3 and the GFP signal. Cells were fixed with paraformaldehyde and fluorescent images obtained. GFP-LC3 puncta parameters were defined using pixel width, intensity, and minimum distance between each puncta. Using these defined parameters allowed for accurate and uniform counting across conditions. Number of GFP puncta per cell was determined in 50 cells per experimental condition by ImageJ. (B) Quantitation of cell puncta measurements with median values indicated by line.

**Supplemental Figure 2.** RNAi inhibition of autophagy results in increase PI positive cells in BRAF<sup>V600E</sup> cells and decreased LC3II. (A) Cells expressing control, ATG5, or ATG12 shRNAs were plated 48 hours after RNAi in standard media and allowed to grow for an additional 72 hours and evaluated for the percentage of PI positive cells. (B) A representative immunoblot demonstrating inhibition of autophagy as evidenced by decreased LC3II levels with RNAi of Atg5 or Atg12. Cell lysates were collected 72 hours after RNAi.

**Supplemental Figure 3.** Increasing doses of CQ results in reduced cell growth in BRAF<sup>V600E</sup> cells and corresponds to evidence of autophagy inhibition by accumulation of LC3II. (A) Cells were plated and treated with increasing doses of CQ for 48 hours. They were monitored every 4 hours by light microscopy using real time in vitro imaging. Quantitative analysis of confluence was performed using the IncuCyte system. Data shown are mean ± SEM of a representative experiment. (B) WT BT16 and V600E 794, AM38 and NMC-G1 cells were treated with
increasing doses of CQ for 4 hours and protein lysate was collected and analyzed for LC3II accumulation by immunoblot analysis.

**Supplemental Figure 4.** Autophagy inhibition does not improve tumor cell kill with Raf inhibition in BRAF WT cells. BT16 cells were treated with increasing doses of vemurafenib in media with and without 10μM CQ for 72 hours and tritiated thymidine uptake assays were performed to assess cell proliferation.

**Supplemental Figure 5.** Vemurafenib has minimal effects on autophagic flux. Cells with mCh-GFP-LC3 were exposed to either standard media or media with 1μM vemurafenib as indicated and analyzed by flow cytometry for the change in ratio of mCh to GFP signal as a measure of autophagic flux.

**Supplemental Figure 6.** Comprehensive timeline of treatments, clinical, and radiologic responses of patient case report.