Cylindrospiradenomas are benign skin appendage tumors that can develop at multiple sites of hair follicle bearing skin as a result of Brooke-Spiegler Syndrome (BSS). The autosomal-dominant BSS is associated with mutations in the \textit{CYLD} gene that encodes a deubiquitinase which inhibits NF-kB signalling resulting in an anti-inflammatory and anti-proliferative effect. Following-up our previous hypothesis that these tumors arise from hair follicle stem cells (HFSC) or their progeny (Massoumi et al. JID 2005), we first immunostained cylindrospiradenoma sections from three BSS patients for the human HF epithelial stem cell (HFSC) markers keratin 15 (K15) and the immune privilege marker CD 200. Interestingly, multiple K15 and CD200 positive, but \(\beta\)1-integrin-negative cells were found lining the tubular tumor structures, while most of the epithelial tumor nodules were K15-negative but brightly \(\beta\)1-integrin-positive. This suggests that cylindrospiradenoma nodules in BSS are derived from immunoprivileged HFSC-like cells and share some characteristics with highly proliferative, undifferentiated basal layer keratinocytes. Interestingly, BSS patients also show extensive T cell infiltrates in tumors and tumor-free regions of their scalp and strong, ectopic expression of MHC class II molecules on the ORS of their hair follicles. This suggests that inflammatory processes precede or accompany tumor formation and growth. To test whether anti-inflammatory agents inhibit tumor growth, we established a serum-free assay normally used for hair follicle organ culture that allows the maintainence of cylindro(spiradeno)ma fragments for up to 6 days in vitro. Addition of Na-salicylate resulted in increased cell death in treated compared to untreated cylindrospiradenoma fragments. Thus, administration of anti-inflammatory agents may offer a pharmacological alternative to surgical cylindrospiradenoma management.