

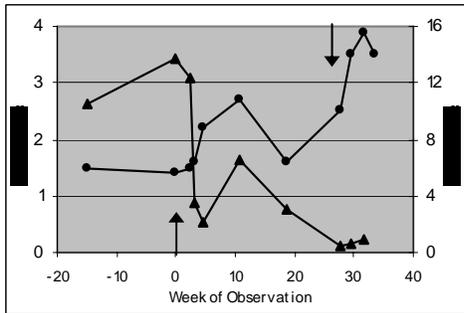
SUCCESSFUL TREATMENT OF MULTI-DRUG RESISTANT FSGS WITH BASILIXIMAB. Glenn H. Bock, Barbara Leuber, Kimberly Capp; Dep't of Pediatrics; Geisinger Medical Center; Danville, PA

In children with idiopathic nephrotic syndrome (INS), corticosteroid (CS)-resistance is predictive of a poor outcome, more so in the child whose INS does not remit with other forms of conventional immunotherapy. Postulating a central pathogenic role of activated T-cells in INS, we treated a multi-drug resistant 14 y old boy with the chimeric monoclonal anti-interleukin 2, basiliximab (Bxb).

The patient developed INS at age 14 mo. Prior treatments included CS, cyclophosphamide, cyclosporine, tacrolimus, and MMF. He was intractably nephrotic. He received 2 series of Bxb, 6 months apart:

	Bxb dose	Day(s) given
Series 1	0.4 mg/kg/dose (20 mg)	0, 4, 14
Series 2	0.8 mg/kg/dose (40 mg)	0, followed by
	0.4 mg/kg/dose (20 mg)	14, 28, 42, 70

Treatment included CS and MMF, primarily as HAMA prophylaxis. Within 1 week there was abrupt diuresis with total wt loss of 27% by week 3. Spontaneous diuresis *preceded* increase of serum albumin or decrease of proteinuria (figure [arrows = series 1&2]). Sustained



remission, normal sCr, albumin, and cholesterol, and no adverse effects after 9 mo.

We conclude that (1) INS remission occurred after Bxb treatment in a multidrug-resistant patient and thus may be a promising and novel therapeutic approach; (2)

The timing of diuresis is consistent with recent observations suggesting abnormal systemic vascular permeability in INS. In addition to the possible therapeutic benefit, Bxb in INS may provide a human model to investigate the mechanisms of edema formation in nephrotic states.