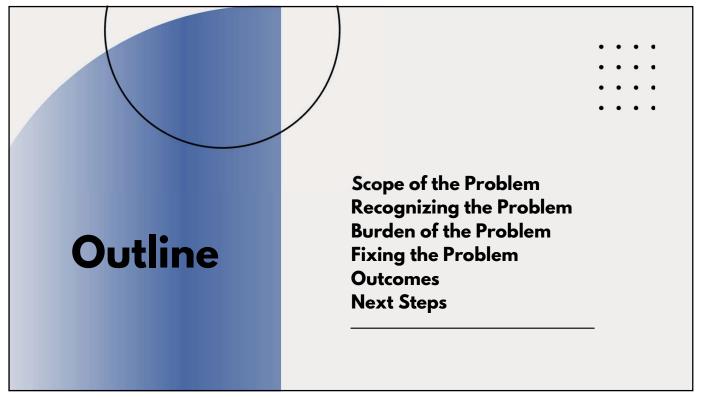
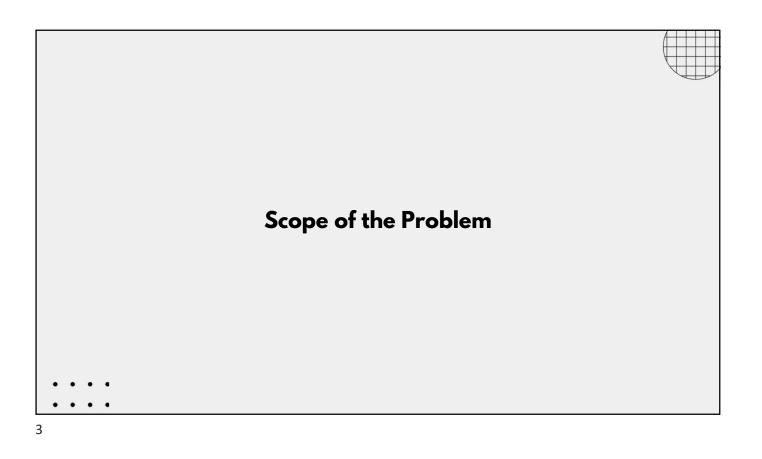
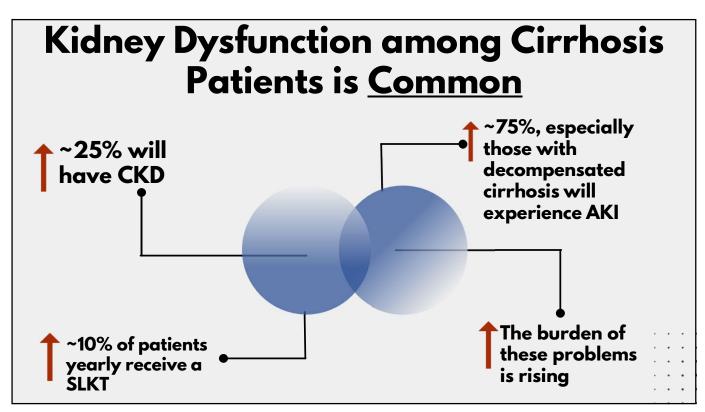
Kidney - Liver Overlap: Recognition of Kidney Disease, Impact on Symptoms and who Needs both Organs

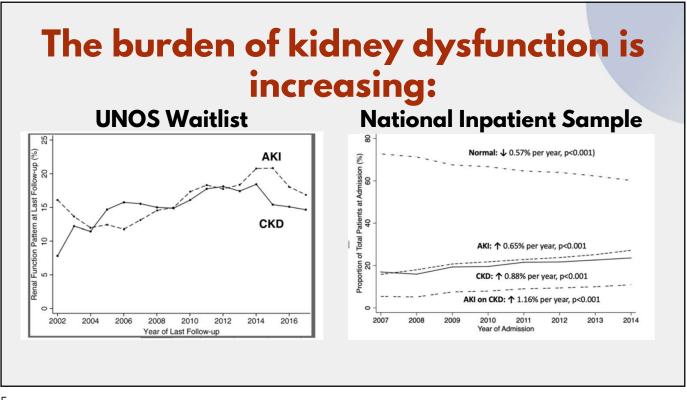
> Giuseppe Cullaro, MD MAS

> > **UCSF** Health

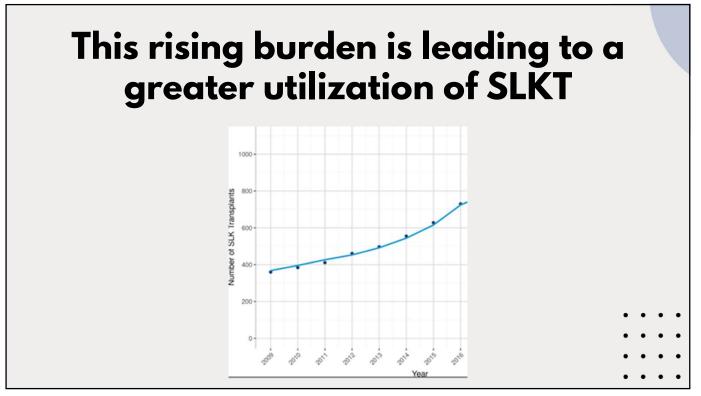




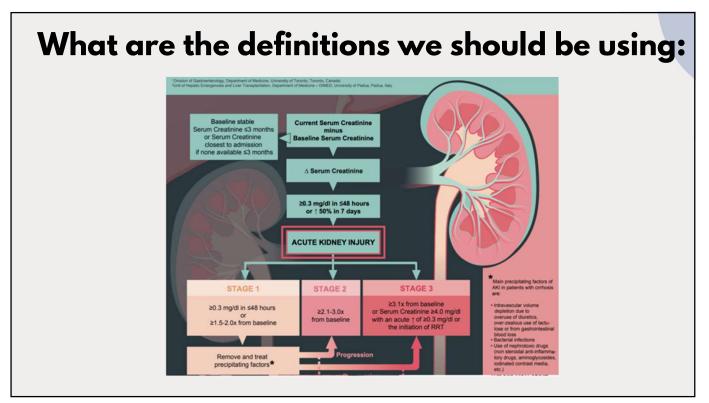


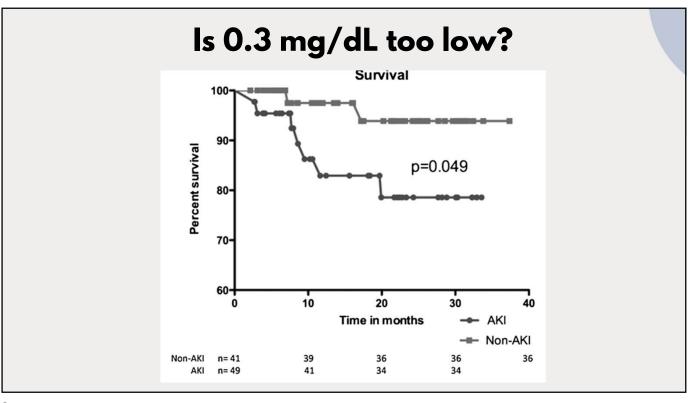


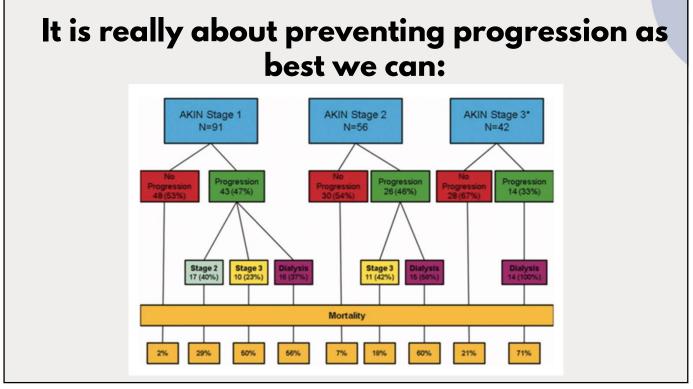








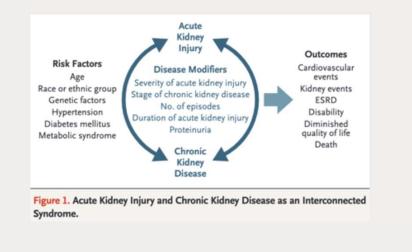




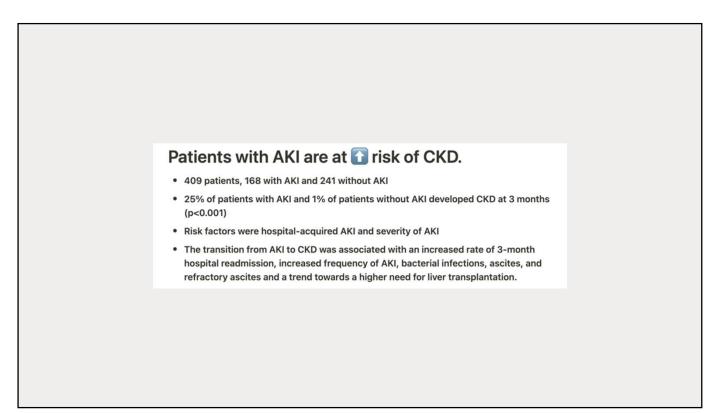
What about CKD?				
Publication eGFR (eSTD) mGI	FR (STD) n		SMD (95% CI)	
	17 (13.05) 41 2.6 (11.1) 151	ŀ	1.37 (0.89-1.85) 1.13 (0.89-1.37) 1.18 (0.96-1.40)	
Asrani, 2018 54.74 (22.97) 38.3 Francoz, 2014 64 (24) 4	17 (13.05) 41 26 (15.55) 125 42 (11) 67 16 (13.2) 52		2.25 (1.69-2.80) 0.84 (0.58-1.10) 1.17 (0.81-1.54) 1.46 (1.03-1.89) 1.40 (0.82-1.97)	
CrCl Caregano, 1994 75.9 (40.1) 56 Heterogeneity: 0 = 0.00; df = 0; P < 0.01; l <sup>2</sup> = 0.016 Model results: z = 2.14; P < 0.01	3.8 (19.8) 27	H	0.60 (0.05-1.14) 0.60 (0.05-1.14)	Paggredlass of the formul
GRAIL Asrani, 2018 53.81 (33.89) 38.2 Heterogeneity: Q = 0.00; df = 0; P < 0.01; l <sup>2</sup> = 0.0% Model results: z = 4.55; P < 0.01	26 (15.55) 125	I	0.59 (0.33-0.84) 0.59 (0.33-0.84)	Regardless of the formule used, eGFR overestimate
Gonwa, 2004 44.5 (28.8) 22 Francoz, 2014 66 (24) 4	26 (15.55) 125 26 (11.1) 155 24 (11) 67 16 (13.2) 52	•[[I]	0.80 (0.54-1.05) 1.00 (0.76-1.24) 1.28 (0.91-1.64) 1.19 (0.78-1.61) 1.03 (0.82-1.24)	kidney function in
MDRD 5 Gonwa, 2004 43.9 (29.3) 22 Heterogeneity: Q = 0.00; df = 0, P < 0.01; i <sup>2</sup> = 0.0% Model results: z = 8.00; P < 0.01	2.6 (11.1) 155	H	0.96 (0.72-1.19) 0.96 (0.72-1.19)	cirrhosis.
Asrani, 2018 48.71 (21.36) 38.3 Gorwa, 2004 39 (26.2) 22 Francoz, 2014 48 (20) 4	17 (13.05) 41 26 (15.55) 125 2.6 (11.1) 155 42 (11) 67 3.6 (13.2) 52	•I <sup>II</sup>	1,12 (0,65-1,58) 0,56 (0,30-0,81) 0,81 (0,56-1,04) 0,37 (0,03-0,71) 0,96 (0,55-1,38) 0,73 (0,49-0,97)	
Nank Gonwa, 2004 58 (28.3) 22 Heterogeneity: 0 = 0.00: df = 0; P < 0.01; I <sup>2</sup> = 0.016 Model results: z = 12.22; P < 0.01	2.6 (11.1) 148	I	1.64 (1.38-1.91) 1.64 (1.38-1.91)	
RFH fejeda-Maldonado, 2019 55.65 (28) 40.1 Heterogeneity: 0 + 0.00; of = 0; P < 0.01; if = 0.0% Model results: z = 3.08; P < 0.01	17 (13.05) 41	I	0.70 (0.26-1.15) 0.70 (0.26-1.15)	

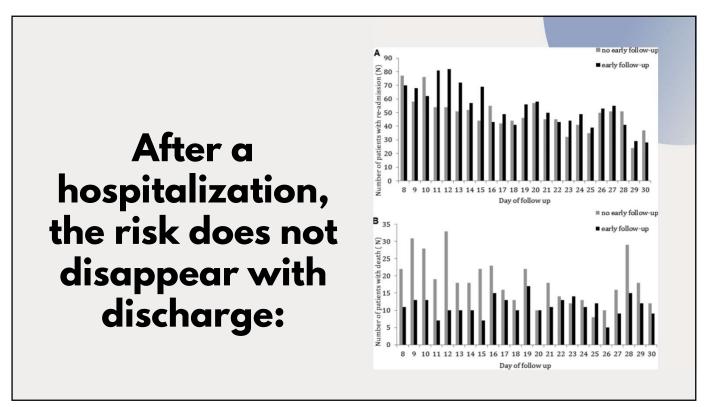


## AKI and CKD are interrelated - and start an important cycle.

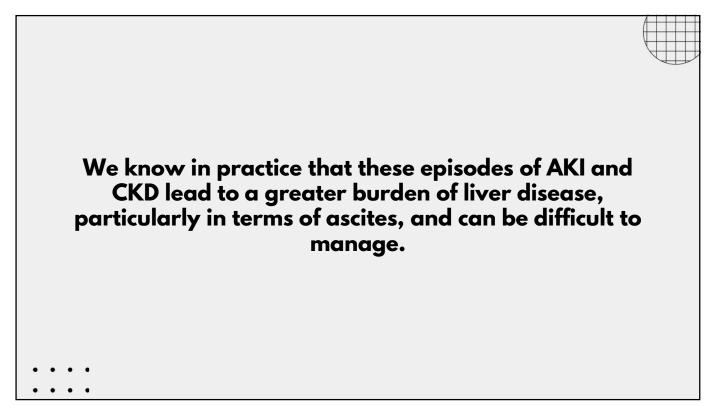


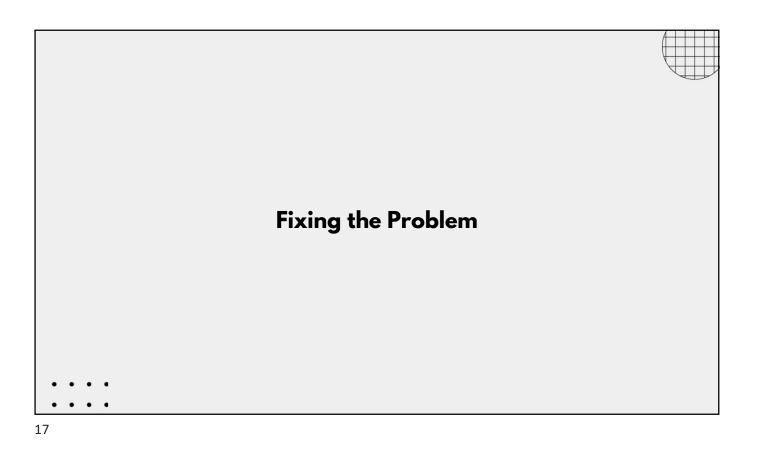


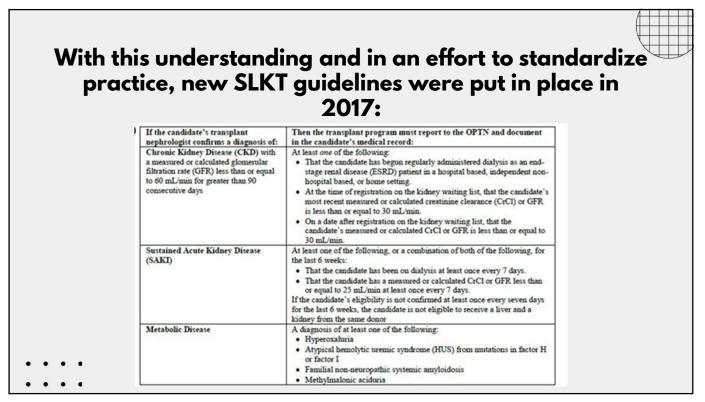


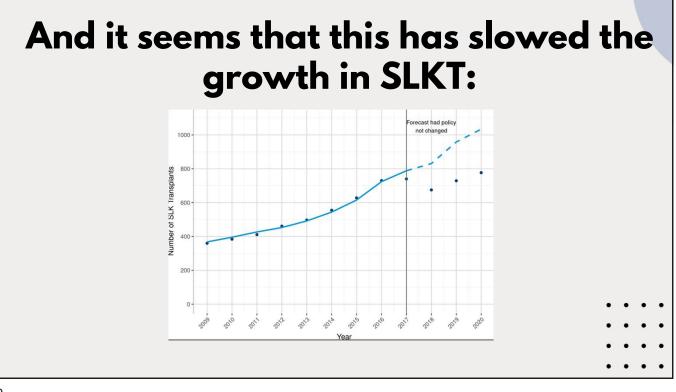


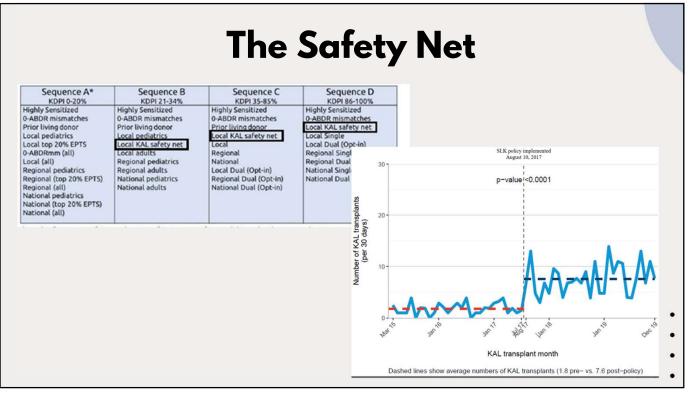


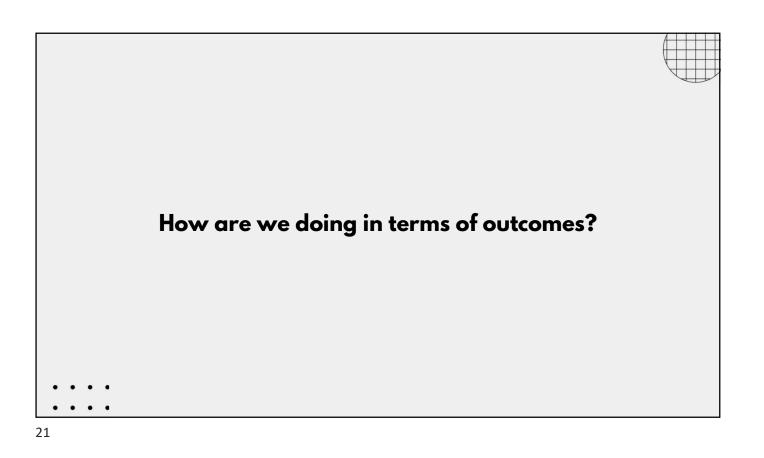


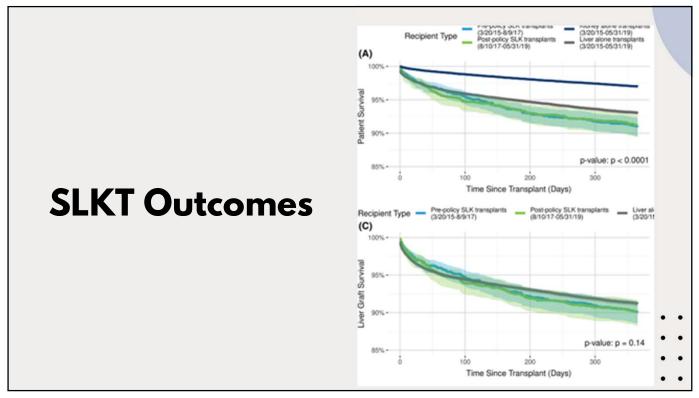


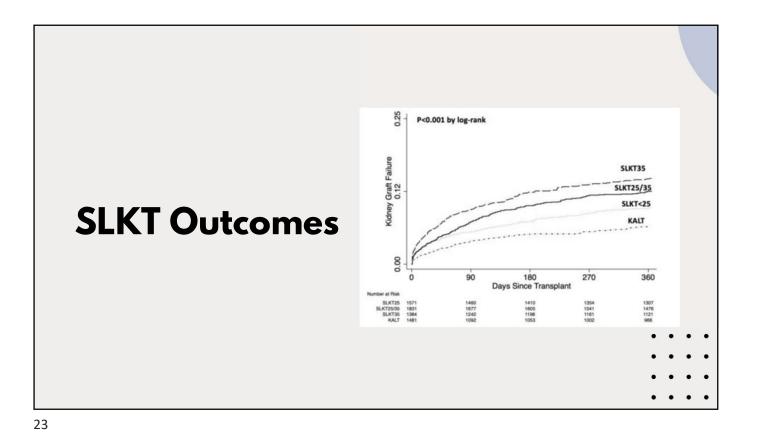


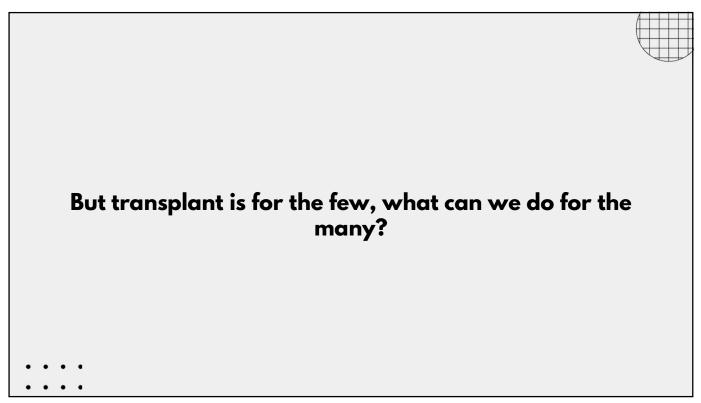


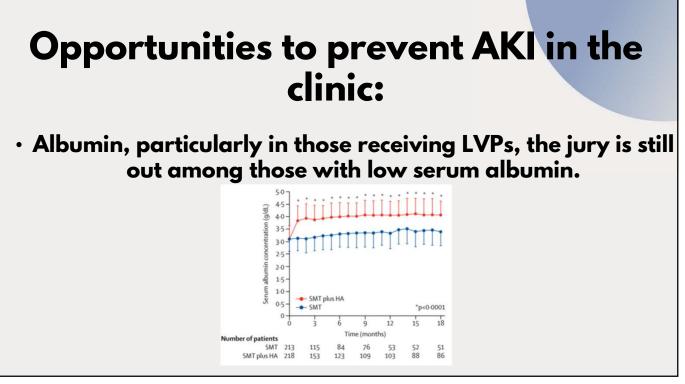












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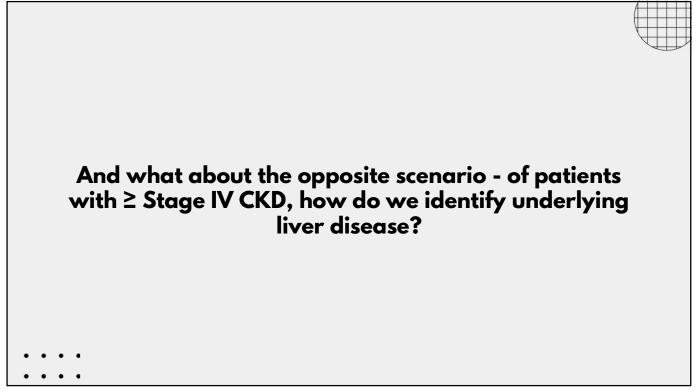
## Opportunities to prevent AKI in the clinic:

2. Pay attention to those with CKD, low MAP, DM/HTN/NAFL, and refractory ascites - they are the ones most at risk.

3. Trials have attempted to augment MAP, but have been mostly negative.

## Opportunities to prevent CKD when patient hospitalized with AKI:

- Early recognition and intervention.
- If HRS-AKI, earlier treatment with vasoactive agents. Terlipressin soon; Trials for HRS-AKI ongoing; please consider referral.
- Watch for recurrence, be vigilant, and monitor diuretics and potential other insults.



## Of Patients with CKD, who to refer for SLKT?

Limited data, as data for fibrosis, are lacking in SRTR.

Often, we are sent these patients by the Kidney Transplant team with a request to risk stratify.

If a patient has an elevated FIB-4 or splenomegaly on ultrasound, we tend to consider first-noninvasive tests of fibrosis (FIBROSCAN, MRE)

If a patient clearly has CSPH - varices on endoscopy or ascites, then we will recommend SLKT.

If a patient has clear signs of fibrosis but no clear CSPH, then we will complete an HVPG and depending on trajectory and degree consider TIPS and KTA or SLKT.

