Given the patient's stable clinical status and normal progression of pregnancy we chose to delay initiation of dialysis and monitor closely. Due to the lack of data for pregnant patients with severe renal disease not receiving hemodialysis, we extrapolated the data from the management of pregnant end-stage renal disease patients on hemodialysis. This included correction of anemia with Erythropoietin, correction of metabolic acidosis with sodium bicarbonate and tight control of hypertension. Our patient remained normotensive and did not require antihypertensives. The BUN and creatinine trended up to 72 mg/dL and 7.5 mg/dL. At 35 weeks and 3 days, the patient presented in active preterm labor and underwent c-section with the birth of an apparently healthy male. After delivery, hemodialysis was initiated.

Of note, the course of the pregnancy was remarkable for polyhydramnios, although the patient was not on dialysis. Polyhydramnios in the absence of dialysis has only been reported in one other case, suggesting that the mechanism of polyhydramnios is independent of dialysis. Perhaps in response to high placental BUN, a solute diuresis occurs in the fetus, resulting in excessive amniotic fluid buildup. Overall, our case shows that successful pregnancy in stage 5 CKD can occur without dialysis.

PUB485

Vitamin C-Induced Hyeroxaluria as the Etiology for Reversible Chronic Renal Failure. Shradha Rathi,[1,2] William Kern,[3] Kai Lau.[1,2] *[1]Medicine, University of Oklahoma, OK; [2]VA Medical Center, OK; [3]Pathology, University of Oklahoma.*

Background: Primary hyperoxaluria causes nephrocalcinosis and renal failure. Less well recognized is progressive insidious kidney disease from secondary causes. Vitamin C is both a precursor of oxalate & a promoter of its absorption, two mechanisms synergistically raising urine oxalate. Malabsorption causes Ca chelation with fatty acids, increases oxalate absorption & excretion. We report here a man with severe renal failure due to excessive vitamin C intake & chronic diarrhea. **Case:** A 73 year old man was admitted for weakness, chronic diarrhea, and a serum creatinine of 8.4 mg%. His renal failure was chronic, based on steadily rising creatinine over months (1.2, 1.8, and 3.1 mg% respectively, 4 months, 5 weeks, & 8 days earlier). His diarrhea was due to niacin, laxatives, & MgO, as it resolved on stopping these agents. Since proteinuria was minimal & other etiologies were absent, we suspected tubular disease from chronic nephrotoxin exposure at home. Struck by his 680 mg/d intake of vitamin C, an oxalate-rich diet, chronic diarrhea, & furosemide, we formulated the diagnosis of Ca oxalate-induced interstitial nephritis. This was later confirmed by a renal biopsy showing diffuse intra-luminal crystals & extensive interstitial fibrosis, hyperoxaluria & hypocitraturia. We removed excessive oxalate by 6 hemodialysis treatments. Two weeks after stopping vitamin C, his creatinine spontaneously fell to 3.1 mg%. On a low oxalate diet & vitamin B6, his urine oxalate to creatinine ratio fell steadily from 0.084 pre-treatment to 0.028 (normal < 0.035), as serum creatinine fell further to 1.8 mg% 2 months later. **Conclusion:** 1) High-dose vitamin C can induce hyperoxaluric nephropathy and progressive renal failure, especially if aggravated by diarrhea, oxalate-rich diet, metabolic acidosis, & dehydration. 2) The diagnosis should be suspected in unexplained renal insufficiency when associated with these risk factors. 3).Since prompt treatment could avert end-stage renal disease, we recommend close monitoring of urine oxalate & serum creatinine in patients on high-dose vitamin C & confirm by a renal biopsy.

PUB486

Lifestyle Factors and Development or Progression of Chronic Kidney Disease: A Systematic Review of the Literature. Vinay Deved, Brenda R. Hemmelgarn. *Division of Nephrology, University of Calgary, Calgary, AB, Canada.*

We performed a systematic review to determine the effect of smoking, obesity, alcohol, exercise, protein restriction, and salt restriction on development and/or progression of chronic kidney disease (CKD).

Studies were identified by searching electronic databases (1966 to 2006) and hand searching bibliographies. Eligible studies were randomized controlled trials, cohort studies or meta-analyses which explored the association between the lifestyle factors of interest and the outcome of development and/or progression of CKD. Two reviewers assessed studies for inclusion and extracted data.

A total of 1,554 abstracts were reviewed. Data were abstracted from the following number of articles which met the eligibility criteria: smoking (7), obesity (6), alcohol (2), exercise (1), protein restriction (9), and salt restriction (0). Overall the results obtained from the studies were varied and not amenable to meta-analysis. In the majority of studies, compared to non-smokers, smokers were approximately twice as likely to develop CKD or end-stage renal disease (ESRD). While evidence from cohort studies suggest a graded increase in the risk of ESRD with increasing body mass index (BMI), evidence regarding alcohol consumption and CKD progression is conflicting. Based on the only available RCT, exercise appears to have no effect on progression of CKD. Finally, the benefit of protein restriction on progression of CKD remains controversial, but a low protein diet does seem to protect against renal replacement therapy and death.

Based on this systematic review of the literature, recommendations can only be made for smoking, obesity and protein restriction. Smoking cessation should be encouraged in all smokers to reduce the risk of developing CKD and ESRD (Grade D). Obese (BMI > 30 kg/m^2) and overweight (BMI 25.0 – 29.9 kg/m^2) individuals should be encouraged to reduce their weight to decrease the risk of developing CKD and ESRD (Grade D). A protein controlled diet (0.80 – 1.0) g/kg/day is recommended for adults with CKD (Grade D). Dietary restriction of < 0.70 g/kg/day should include careful monitoring for clinical and biochemical markers of malnutrition (Grade D).

PUB487

Safety Concerns on the Use of Low Molecular Weight Heparin in Dialysis Patients. Maurizio Gallieni, Mario Cozzolino, Elena Missaglia, Cesar Crovetto, Giusy Chiarelli, Diego Brancaccio. *Chair of Nephrology, San Paolo Hospital-University of Milan, Italy.*

The use of low molecular weight heparin (LMWH) in the treatment of deep vein thrombosis and pulmonary embolism is widespread, because of its superior efficacy, safer profile, and greater cost effectiveness over unfractionated heparin (UFH).

The activity of UFH is easily monitored by measuring the PTT, while LMWH is monitored by measuring the ability of plasma from treated patients to inhibit factor Xa. This is not considered necessary in patients with normal renal function.

Several LMWHs can be used during hemodialysis for anticoagulation. This can generate the error of assuming that even high doses of LMWH can be used safely in severe CKD. However, this is not the case. A recent meta-analysis (Ann Int Med 2006;144:673) pointed out that CKD patients with a creatinine clearance of 30 mL/min or less who are treated with standard therapeutic doses of enoxaparin have elevated levels of anti-Xa and an increased risk for major bleeding. There were insufficient studies to assess the risk with other LMWHs and prophylactic doses of enoxaparin.

We investigated how this important safety information is diffused among Italian nephrologists, through 150 face to face interviews during medical meetings. Results are displayed in the table.

We conclude that more educational efforts should be undertaken, to avoid major bleeding incidents in dialysis patients erroneously treated with full doses of LMWH.

The established advantages of LMWH over UFH anticoagulant treatment can not be extended to patients with advanced renal failure (creatinine clearance < 30 ml/min) until a widespread use of tests for anti-Xa activity will be available. UFH remains the drug of choice for the prevention and treatment of thrombosis in patients with renal failure.

	YES	NO
Have you used LMWH for dialysis anticoagulation?	90%	10%
Have you used high dose LMWH for treatment of thrombosis in dialysis patients?	95%	5%
At full dose?	52%	48%
Can you recall any major bleeding in patients taking LMWH?	12%	88%
Do you check anti-factor Xa levels?	0%	100%
As far as you know, can your laboratory test anti-factor Xa activity?	0%	100%

PUB488

Three Year Trends in Renal Function, Cardiovascular Risk Factors, and Coronary Artery Calcification among Patients with and without Chronic Kidney Disease. Amit Sharma,[3] Dylan Wessman,[1] Frank Mullens,[2] Robin Parker,[2] Sandra Gilbert,[1] Louis Brenner,[4] Scott Chasan-Taber.[4] *[1]Cardiology, Naval Medical Center, San Diego, CA; [2]Radiology, Naval Medical Center, San Diego, CA; [3]Clinical Research, Boise Kidney & Hypertension Institute, Meridian, ID; [4]Genzyme Corporation, Cambridge, MA.*

We conducted a single-center, longitudinal, observational study to examine the temporal association between renal function, traditional and novel cardiovascular disease (CVD) risk factors, and coronary artery calcification (CAC).

Subjects included adults without symptomatic coronary artery disease who were grouped according to eGFR < or ≥ 60 ml/min using the MDRD formula. Physical measures included height, weight, heart rate, and blood pressure. Laboratories included complete blood count, chemistry profile, intact parathyroid hormone, fasting lipid panel, hemoglobin A1c, C-reactive protein, fibrinogen, homocysteine, and urinary microalbumin. Coronary artery calcification was measured by multidetector row computed tomography (MDCT) scan and expressed as an Agatston score. Physical measures, laboratories, and MDCT scans were performed at baseline, 1 year, and 3 years.

Eighty seven subjects (average age 55 years, 49% male, and 51% caucasian) completed the study, of whom 30 had an eGFR < 60 ml/min at baseline. Although the average SCr and eGFR remained stable over time, the average CAC score increased progressively among patients with and without CKD (table 1). Three-year trends for markers of bone mineral metabolism and CVD risk factors were more heterogeneous, suggesting that the development and progression of CAC is a multifactorial process.

Table 1. Three Year Trends in Renal Function and Coronary Artery Calcification

	Baseline		Year 1		Year 3	
	CKD	Control	CKD	Control	CKD	Control
SCr (mg/dl)	1.8 ± 0.6	1.0 ± 0.2	1.8 ± 0.7	1.0 ± 0.3	1.9 ± 1.0	1.0 ± 0.3
eGFR (ml/min)	41.4 ± 11.7	79.8 ± 15.6	44.2 ± 14.4	76.1 ± 18.0	42.4 ± 15.9	75.2 ± 18.1
CAC score	112.9 ± 278.4	146.8 ± 398.3	156.1 ± 336.1	171.1 ± 456.0	201.3 ± 452.3	200.8 ± 471.3

Data presented as mean ± standard deviation

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