HIGH-YIELD NEPHROLOGY FOR THE INTERNIST

EDUARDO ALAS MD FASN
OBJECTIVES

• The state of nephrology
• Estimating glomerular filtration rate
• Chronic kidney secondary disease
• Acute kidney injury
• Renal transplant
QUESTION

• How many people are estimated to have CKD in the U.S. currently?

A. 1 million people
B. 5 million people
C. 15 million people
D. 25 million people
E. 30 million people
QUESTION

• How many people are on dialysis at this time in the U.S.?

A. 250,000 people
B. 450,000 people
C. 550,000 people
D. 1,500,000 people
E. 2 million people
THE INTERNIST IS THE PRIMARY PROVIDER TO RENAL PATIENTS

• Kidney disease is the 9TH leading cause of death in the United States.
• An estimated 31 million people in the United States (10% of the adult population) have chronic kidney disease (CKD).
• 9 out of 10 people who have stage 3 CKD (moderately decreased kidney function) do not know it.
• CKD is more common among women, but men with CKD are 50% more likely than women to have their CKD turn into kidney failure (also called end-stage renal disease or ESRD)
• Some racial and ethnic groups are at greater risk for kidney failure. Compared to whites;
  • The risk for African Americans is almost 4 times higher,
  • Native Americans is 1.5 times higher,
  • Asians is 1.4 times higher
  • Compared to non-Hispanics, Hispanics are almost 1.5 times as likely to be diagnosed with kidney failure
Chronic kidney disease (CKD) is a condition in which the kidneys are damaged or cannot filter blood as well as healthy kidneys. Because of this, excess fluid and waste from the blood remain in the body and may cause other health problems.

CKD Is Common Among Adults in the United States

Fast Stats

- 30 million people or 15% of US adults are estimated to have CKD. *
- 48% of those with severely reduced kidney function but not on dialysis are not aware of having CKD.
- Most (96%) people with kidney damage or mildly reduced kidney function are not aware of having CKD.

More than 1 in 7

15% of US adults are estimated to have chronic kidney disease—that is about 30 million people.
NEPHROLOGY WORKFORCE DATA

• In 2013, 9,007 physicians in the United States claimed nephrology as their specialty.
• This translates to ~1 nephrologist per 1,666 adult U.S. residents with advanced CKD (stages 3 to 4).
• In New Mexico approximately 40 practicing nephrologists.
NUMBERS OF FELLOWSHIP TRAINED NEPHROLOGISTS ARE IN DECLINE NATIONALLY

ASN 2018 workforce report
Exhibit 4. Geographical Distribution of Nephrology Fellowship Programs and ESRD Patients per Nephrologist by HRR, 2011

Source: GW Health Workforce Institute analysis of Dartmouth Atlas of Health Care; Fellowship program data from ACGME.
In New Mexico fewer Nephrologists are taking care of more dialysis patients
INCREASED NUMBER OF RENAL PATIENTS


Source: US Renal Data System 2014 Annual Data Report Fig 1.10 (http://www usrds.org/2014/download/Vol2_01_inc-prev_14_slides.pptx)
CHRONIC KIDNEY DISEASE DEFINITION  
(KDIGO 2012)

• CKD is defined as abnormalities of kidney structure or function, present for >3 months, with implications for health.
  • Markers of kidney damage (one or more)
    • Albuminuria (AER >30 mg/24 hours; ACR >30 mg/g [>3 mg/mmol])
    • Urine sediment abnormalities
    • Electrolyte and other abnormalities due to tubular disorders
    • Abnormalities detected by histology
    • Structural abnormalities detected by imaging
    • History of kidney transplantation
  • Decreased GFR
    • GFR <60 ml/min/1.73 m2 (GFR categories G3a–G5)

A 75 yo male arrives to your office for his yearly physical. He has a past history of type 2 diabetes, hypertension and COPD. He is an active smoker still. He is on metformin and a calcium channel blocker and a couple of inhalers. He also uses 2L of O2 via NC. He weighs about a 50 Kg.

You decide, given his comorbidities, you would like to check his renal clearance. How would you best first approximate his GFR?

A. Check a random serum creatinine and calculate his clearance based on Cockcroft-Gault
B. Check a random serum creatinine and use the MDRD equation
C. Check random serum creatinine and use the CKD-epi equation for GFR
D. Measure a 24 hour urine for creatinine clearance
E. Check a cystatin-c and use the CKD-epi equation for GFR
DETERMINATION OF GLOMERULAR FILTRATION RATE

• GFR estimating equation have been in use since the 1970’s

• However the 4 variable Modification in Diet in Renal Disease (MDRD) study developed in 2000 was the first eGFR equation widely used in clinical laboratories.
  • It’s strength is in estimating GFR<60 ml/min m²
  • UNDERESTIMATES GFR>60 ml/min m²
  • Relied on available demographic

• Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) was formed in 2003 to improve eGFR equations using both creatinine and cystatin C.
DETERMINATION OF GLOMERULAR FILTRATION RATE

• Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) was formed in 2003 to improve eGFR equations
  • Used both creatinine and cystatin C
  • A more diverse population, larger age range, GFR range, diabetes, and transplants.
  • Has a lesser bias at GFR>60 compared to MDRD
  • https://www.kidney.org/professionals/kdoqi/gfr_calculator
NOVEL CKD GFR ESTIMATING MARKERS THAT HAVE PROGNOSTIC VALUE

- Beta 2 macroglobulin; MHC associated cell surface molecule
  - It is freely filtered by the glomerulus and 99.9% reabsorbed in the proximal tubule.
  - B2M elevation in association with cell turnover.
  - Currently used in risk stratification of multiple myeloma.
  - Already routinely measured in many clinical laboratories, and a clinical standard exists.

- Beta trace protein, lipocalcin prostaglandin D2 synthase
  - First noted in to be elevated in CSF leaks in CKD patients in the late 1980’s.
  - Multiple groups of have studied eGFR equations in transplant patients and been found to be superior than the MDRD.
  - A clinical standard does not yet exist.

- Likely we will use a 4 marker estimating equation.
CURRENT APPROACH IN ESTIMATING GFR

- Normal GFR varies according to age, sex, and body size; in young adults it is approximately 120 ml/min/1.73 m²
- In muscular individuals consider checking a 24 hour creatinine clearance, as often there is a discordance between muscle mass and a random serum creatinine.
- Check a cystatin C
  - But remember it is produced by all nucleated cells so increased cell turnover states affect this measure as does smoking
  - But is useful in determining GFR in decreased on increased muscle mass states (i.e. cirrhosis or excess muscle mass).
- Use the CKD–EPI creatinine – cystatin C calculator
**AS OF 2013, STAGES OF CKD**

<table>
<thead>
<tr>
<th>Persistent albuminuria categories</th>
<th>Description and range</th>
<th>A1</th>
<th>A2</th>
<th>A3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal to mildly increased</td>
<td>&lt;30 mg/g &lt;3 mg/mmol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderately increased</td>
<td>30–300 mg/g 3–30 mg/mmol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severely increased</td>
<td>&gt;300 mg/g &gt;30mg/mmol</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Guide to Frequency of Monitoring (number of times per year) by GFR and Albuminuria Category:

<table>
<thead>
<tr>
<th>GFR categories (ml/min/1.73 m²)</th>
<th>Description and range</th>
<th>G1 (Normal or high)</th>
<th>G2 (Mildly decreased)</th>
<th>G3a (Mildly to moderately decreased)</th>
<th>G3b (Moderately to severely decreased)</th>
<th>G4 (Severely decreased)</th>
<th>G5 (Kidney failure)</th>
</tr>
</thead>
<tbody>
<tr>
<td>60–89</td>
<td>1 if CKD</td>
<td>≥90</td>
<td>60–89</td>
<td>45–59</td>
<td>30–44</td>
<td>15–29</td>
<td>&lt;15</td>
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<tr>
<td>45–59</td>
<td></td>
<td></td>
<td>45–59</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>30–44</td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>15–29</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>3</td>
<td>4+</td>
</tr>
<tr>
<td>&lt;15</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4+</td>
<td></td>
</tr>
</tbody>
</table>

GFR and albuminuria grid to reflect the risk of progression by intensity of coloring (green, yellow, orange, red, deep red). The numbers in the boxes are a guide to the frequency of monitoring (number of times per year).
PREDICTORS OF PROGRESSION

- Identify factors associated with CKD progression to inform prognosis.
  - Level of GFR
  - Level of albuminuria
  - Age
  - Sex
  - Dyslipidemia
  - Race/ethnicity
  - Elevated BP
  - Hyperglycemia
  - Smoking
  - Obesity
  - History of cardiovascular disease
  - Ongoing exposure to nephrotoxic agents
• Acid base balance is maintained by renal excretion of the daily acid load (~1 meq/kg/day) titratable acid and ammonium ions

• In CKD this is maintained by increased ammonium excretion by the nephrons
  • As nephron mass decreases so do the capacity to excrete ammonium
  • At GFR 40-50 the ammonium excretion begins to fall
  • Resulting in increased H⁺ and titratable acids (i.e. phosphoric acid)
  • The acid is buffered by extracellular bicarbonate and by bone
  • CKD serum bicarbonate levels stabilize between 12-20, secondary to buffering mechanisms
PROGRESSION OF CKD LEADS TO INCREASED PREVALENCE OF METABOLIC ACIDOSIS

Fig. 1. Prevalence of metabolic acidosis (defined as proportion of patients with serum bicarbonate of <22 mEq/L) in a population-based cohort of 570,170 US veterans with non-dialysis-dependent CKD stages 1–5, by CKD stage. Numbers within the columns represent the actual number of patients with serum bicarbonate <22 mEq/L within the respective groups. Based on data from reference [56].
PATHOPHYSIOLOGIC CONSEQUENCES OF A METABOLIC ACIDOSIS

- Osteopenia
- Increased secondary parathyroid disease
- Decreased respiratory reserve
- Reduced myocardial contractility and CHF
- Impaired growth in pediatric CKD
- Rapid progression of CKD
TREATMENT

• Typically treat to a serum bicarb level of 23-29
  • Caution with alkalosis, as this is associated with increased mortality
• Treat with oral sodium bicarbonate, sodium citrate, or calcium citrate
• Blood pressure does not typically increase with this sodium load
ANEMIA IN CKD

• Most CKD related anemias are normocytic and normochromic
  • Related to decreased cell survival
  • Decreased erythropoietin production by the kidneys
  • Administration of erythropoietin stimulating (ESA) agents with consumption of iron

• NHANES Study, anemia prevalence
  • Increased by 1% in GFR<60 ml/min
  • 9% at a GFR of 30 ml/min
  • 33-67% GFR<15 ml/min

• Screening occurs, at least twice yearly in GFR<45 ml/min and increases to q3months once they are anemic
# Iron Administration in CKD

## Oral Iron
- High pill burden, multiple times a day
- Increases in GI side effects
- Decreased GI absorption in uremia
  - Aberrant hepcidin clearance
- Medication interactions
  - i.e. use of calcium carbonate as a phosphorus binder

## IV Iron
- Increased oxidative stress; endothelial damage
- Increased in cardiovascular events
- Increased rate of decline of GFR
- Increased infections
- Increased use of venous access
THE IV IRON CONTROVERSY

• FerinjectVR assessment in patients with Iron deficiency anemia and NonDialysis-dependent Chronic Kidney Disease (FIND-CKD) Trial
  • 1 -year, open-label, multicenter, prospective study of patients with nondialysis-dependent CKD receiving oral or iv iron, in the post hoc analysis
    • 617 patients in the safety analysis
    • Pt were randomized to receive
      • Oral iron
      • Low dose iv iron
      • High dose iv iron
NO DIFFERENCE BETWEEN ORAL AND IV IRON

<table>
<thead>
<tr>
<th>Event</th>
<th>High ferritin FCM (n = 154) (138.5 PY)</th>
<th>Low ferritin FCM (n = 150) (129.0 PY)</th>
<th>Oral iron (n = 312) (242.8 PY)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac disorders (per 100 PY)</td>
<td>7.2</td>
<td>5.4</td>
<td>5.8</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>1.4</td>
<td>-</td>
<td>1.6</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>0.7</td>
<td>-</td>
<td>1.2</td>
</tr>
<tr>
<td>Cardiac failure congestive</td>
<td>1.4</td>
<td>0.8</td>
<td>0.4</td>
</tr>
<tr>
<td>Acute coronary syndrome</td>
<td>1.4</td>
<td>-</td>
<td>0.4</td>
</tr>
<tr>
<td>Infections (per 100 PY)</td>
<td>4.3</td>
<td>3.9</td>
<td>4.9</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>-</td>
<td>0.8</td>
<td>1.6</td>
</tr>
<tr>
<td>Renal and urinary disorders (per 100 PY)</td>
<td>4.3</td>
<td>1.6</td>
<td>5.4</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>0.7</td>
<td>0.8</td>
<td>2.5</td>
</tr>
</tbody>
</table>
THE IV IRON CONTROVERSY

- Randomized trial to evaluate intravenous and oral iron in chronic kidney disease (REVOKE)
  - Single center, prospective study of 136 patients with CKD and anemia to receive either oral iron sulfate or intravenous iron sucrose
  - Terminated early due to increased risk of SAEs, cardiovascular SAEs, and infection in patients receiving IV iron compared with those receiving oral iron

- Possible explanations for the conflicting findings (per the authors)
  - Differences in methods of SAE adjudication and reporting as well i.e. multiple events within one patient was reported
  - Events were reported up beyond the point at which another anemia therapy was initiated and/or the randomized study medication was discontinued.
  - They included CKD who were progressing rapidly, and they could reach ESRD within 12 months of study initiation
<table>
<thead>
<tr>
<th>Event type</th>
<th>Oral Iron (n=69)</th>
<th>IV Iron (n=67)</th>
<th>Adjusted incidence rate ratio IV/Oral (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects (n)</td>
<td>Incidence rate (events/100 PY)</td>
<td>Subjects (n)</td>
<td>Incidence rate (events/100 PY)</td>
</tr>
<tr>
<td>Overall SAEs</td>
<td>40</td>
<td>176</td>
<td>168.4</td>
</tr>
<tr>
<td>Infections</td>
<td>11</td>
<td>27</td>
<td>25.8</td>
</tr>
<tr>
<td>Skin</td>
<td>6</td>
<td>6</td>
<td>5.7</td>
</tr>
<tr>
<td>Bone</td>
<td>2</td>
<td>7</td>
<td>6.7</td>
</tr>
<tr>
<td>Lung</td>
<td>4</td>
<td>4</td>
<td>3.8</td>
</tr>
<tr>
<td>UTI</td>
<td>3</td>
<td>5</td>
<td>4.8</td>
</tr>
<tr>
<td>Sepsis</td>
<td>1</td>
<td>2</td>
<td>1.9</td>
</tr>
<tr>
<td>Other</td>
<td>2</td>
<td>3</td>
<td>2.9</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>19</td>
<td>36</td>
<td>34.4</td>
</tr>
<tr>
<td>CHF</td>
<td>9</td>
<td>15</td>
<td>14.3</td>
</tr>
<tr>
<td>Angina</td>
<td>2</td>
<td>2</td>
<td>1.9</td>
</tr>
<tr>
<td>MI</td>
<td>8</td>
<td>9</td>
<td>8.6</td>
</tr>
<tr>
<td>Stroke</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>4</td>
<td>4</td>
<td>3.8</td>
</tr>
<tr>
<td>PVD</td>
<td>1</td>
<td>2</td>
<td>1.9</td>
</tr>
<tr>
<td>Other</td>
<td>4</td>
<td>4</td>
<td>3.8</td>
</tr>
<tr>
<td>Renal</td>
<td>18</td>
<td>29</td>
<td>27.7</td>
</tr>
<tr>
<td>AKI</td>
<td>15</td>
<td>22</td>
<td>21</td>
</tr>
</tbody>
</table>
THE IV IRON CONTROVERSY

- Generally the weight of clinical evidence indicated IV iron is both safe and effective
- However, future trials will confirm long-term safety
- For the time being we will be using IV iron for long term use in CKD, not on dialysis, patients.
DUAL PURPOSE ORAL IRON REPLETION /PHOSPHORUS BINDER

- Ferric citrate (Auryxia®) was approved in November 2017 for iron deficit anemia.
- It is also an oral phosphorus binder.
- Phase 3 clinical trial of 234 patients with CKD 3-5 over a 16 week period.
- You may see patients on this medication for phosphorus control, please do not stop it.

Fishbane, JASN 2017
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CHRONIC KIDNEY DISEASE MINERAL BONE DISORDERS (CKD-MBD) AKA RENAL OSTEODYSTROPHY

- Develops from the kidney’s inability to excrete phosphorus properly
- Increased phosphorus levels stimulates FGF-23 and parathyroid hormone (PTH)
- FGF-23 reduces phosphate levels
  - Increased renal excretion
  - PTH stimulation
  - Calcitriol inhibition (leads to decreased GI absorption)
- The resulting hypocalcemia leads to an increase in PTH secretion; secondary hyperparathyroidism (SHPT)
SECONDARY HYPERPARATHYROIDISM

- SHPT is associated with increased bone turnover
- Risk of fractures
- Vascular calcifications
- Risk of cardiovascular and all-cause mortality
- Observational data indicate that PTH >600 pg/mL is associated with a higher risk of cardiovascular mortality as well as all-cause cardiovascular hospitalization
SECONDARY HYPERPARATHYROIDISM & MORTALITY

Figure 5. Associations of parathyroid hormone (PTH) levels with mortality and hospitalizations among Dialysis Outcomes and Practice Patterns Study participants not receiving secondary hyperparathyroidism treatment during the first 1 year of study observation. Adjusted for demographics, comorbidities, albumin, hemoglobin, calcium, and phosphorus. Outcomes are in relationship to the patient’s first reported PTH after 1 year of study observation without prescription for PTH-controlling medications. Mortality: 5387 patients, 1061 deaths. Hospitalization: 5381 patients, 2258 hospitalizations.
TREATMENT OF SHPT

• Primarily reduce phosphorus intake
• Administer vitamin D analogues
• Administer calcimimetics
  • Cinacalcet
  • IV etelcalcetide
• Parathyroidectomy

• Three randomized controlled trials have analysed the effects of treatment with cinacalcet on hard clinical outcomes such as vascular calcification, bone histology and cardiovascular mortality and morbidity.
• “… hard outcomes remain(s) elusive”
OSTEOPOROSIS CARE IN CKD PATIENTS

- Fracture Reduction Evaluation of Denosumab in Osteoporosis Every 6 Months (FREEDOM) study suggested that denosumab 60 mg every 6 months was safe increasing BMD in women with postmenopausal osteoporosis and CKD stages 1–4.
- However there is increased evidence that denosumab therapy in CKD patients is associated with transient decreases in serum calcium, (denosumab-induced hypocalcemia).

D. Miyaoka, Osteopor Int 2018
HOW ABOUT THE ROLE OF BISPHOSPHONATES IN CKD

• Generally, bisphosphonates are excreted via the kidneys.

• Accumulation of bisphosphonate in the bone is suspected to be faster in advanced-stage CKD patients.

• There are no large-scale clinical safety data on bisphosphonate use in patients with CKD4 or eGFR <35 mL/min/1.73 m².

• Use of any bisphosphonate drug generally avoided in advanced CKD patients.
COMMUNITY ACQUIRED ACUTE KIDNEY INJURY (CA-AKI)

- 36 millions American use OTC NSAIDS
- Recent Beers Criteria specifically identifies acute kidney injury (AKI) only for indomethacin and ketorolac
  - suggests that there is a risk of AKI with noncyclooxygenase (COX) and COXselective NSAIDs only when creatinine clearance falls below 30 mL/min
- CA-AKI typically have disease states that reduce kidney perfusion and require a compensatory increase in kidney prostaglandins to promote vasodilation of the afferent arteriole to maintain adequate glomerular capillary pressure and filtration
CA-AKI

• Patients who have an episode CA-AKI develop new-onset chronic kidney disease (CKD)
  • This is concerning for geriatric populations, where it has been shown that up to 70% of patients aged ≥67 years who have an episode of AKI develop CKD within 2 years
• These patients have faster CKD progression at rates similar to patients who develop nosocomial AKI (eg, from trauma or sepsis)
• When NSAID therapy was given concomitantly with both diuretics and RAAS inhibitor, a 31% higher rate of AKI was observed (RR = 1.31; 95% CI = 1.12-1.53)

Pai, A. Annals of Pharmacother 2018
BACTRIM TOXICITY

- In a VA Study in Texas in 2012 of 573 veteran patients
  - Electronic record review of a 3 year interval
  - 11.2% patients developed AKI
  - 5.8% of the AKI was judged to be due to directly to Bactrim
  - 4.9% possibly due to Bactrim use
  - AKI resolved after discontinuation of the medication
  - 1 patient required dialysis.
- Multivariate analysis showed patients with DM2 and HTN were at greatest risk
- There was no dose or duration effect

Fraser, T. J Antimicrob Chemother 2012
• Metformin is associated with increased risk of lactic acidosis in CKD patients
• Most studies with this medication excluded patients with CKD
• This fear is largely from our experience with phenformin, which was withdrawn in 1978 as well as case reports of metformin and lactic acidosis.
• In April of 2016 the FDA cleared the use of metformin “metformin can be used safely in patients with mild impairment in kidney function and in some patients with moderate impairment in kidney function.”
METFORMIN FDA LABELING RECOMMENDATIONS

- Before starting metformin, obtain the patient’s eGFR.
- Metformin is contraindicated in patients with an eGFR below 30 mL/minute/1.73 m2.
- Starting metformin in patients with an eGFR between 30-45 mL/minute/1.73 m2 is not recommended.
- Obtain an eGFR at least annually in all patients taking metformin. In patients at increased risk for the development of renal impairment such as the elderly, renal function should be assessed more frequently.
- In patients taking metformin whose eGFR later falls below 45 mL/minute/1.73 m2, assess the benefits and risks of continuing treatment. Discontinue metformin if the patient's eGFR later falls below 30 mL/minute/1.73 m2.
- Discontinue metformin at the time of or before an iodinated contrast imaging procedure in patients with an eGFR between 30 and 60 mL/minute/1.73 m2; in patients with a history of liver disease, alcoholism, or heart failure; or in patients who will be administered intra-arterial iodinated contrast. Re-evaluate eGFR 48 hours after the imaging procedure; restart metformin if renal function is stable.

• In a 2018 JAMA Int. Med. paper of 75413 diabetics on metformin with a GFR on record from 2004-2017 in a single community based health system
  • 2335 acidosis events were recorded
• Compared with alternative DM medications metformin was not associated with increased acidosis risk (adjusted hazard ratio [HR], 0.98; 95%CI, 0.89-1.08)
• eGFR 45 to 59 mL/min/m$^2$ (adjusted HR, 1.16; 95%CI, 0.95-1.41)
• eGFR 30 to 44 mL/min/m$^2$ (adjusted HR, 1.09; 95%CI, 0.83-1.44)
• At a eGFR less than 30 mL/min/m$^2$ it was associated with an increased risk of (adjusted HR, 2.07; 95%CI, 1.33-3.22).
• How many transplant centers are in New Mexico?

A. 1
B. 2
C. 3
D. 4
E. 5
QUESTION

- What solid organs are transplanted in New Mexico currently?
  A. Liver
  B. Lung
  C. Heart
  D. Kidney
  E. Pancreas
RENAL TRANSPLANT DOES OCCUR IN NEW MEXICO

- We have 2 transplant centers in New Mexico

**Program Summary**

**Adult**

<table>
<thead>
<tr>
<th>TRANSPLANT VOLUME</th>
<th>TRANSPLANT RATE</th>
<th>OUTCOME ASSESSMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>41 ADULTS</td>
<td>15.2 PER 100 PEOPLE PER YEAR</td>
<td>AS EXPECTED</td>
</tr>
</tbody>
</table>

**Pediatric**

<table>
<thead>
<tr>
<th>TRANSPLANT VOLUME</th>
<th>TRANSPLANT RATE</th>
<th>OUTCOME ASSESSMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 CHILDREN</td>
<td>98.8 PER 100 PEOPLE PER YEAR</td>
<td>AS EXPECTED</td>
</tr>
</tbody>
</table>
THE NEED FOR TRANSPLANT IS INCREASING IN OUR STATE

**Waiting List**

**As of January 2018**

- **256** people were on the waiting list
  - **74** people joined the list
    - **46** received transplants
    - **0** recovered
  - **81** people were removed
    - **46** transplanted
    - **7** deteriorated
    - **18** other
    - **3** transferred to another center
  - **7** died

**At the end of December 2017**

- **249** people were on the waiting list
CHARACTERISTICS OF RENAL TRANSPLANTS IN NEW MEXICO

Transplants
BETWEEN JANUARY 2018 AND DECEMBER 2017

46 PEOPLE
RECEIVED TRANSPLANTS

3 with a living donor
43 with a deceased donor

Select a characteristic below to see basic characteristics of transplants recipients at this program:
EXCITING NEWS IN NM TRANSPLANT

The University of New Mexico Health Sciences

Newsroom

The Altruistic Donor

New Mexico man’s act triggers nationwide chain of live kidney donations

Albuquerque Journal

Doctors perform NM’s first pancreas transplant
TRANSPLANT MEDICATIONS AND SOME SIDE EFFECTS

- Calcineurin inhibitors (tacrolimus and cyclosporin)
  - Post transplant diabetes in 20% of patients
  - Gout
  - Pancytopenia
  - Gingival hyperplasia

- Mtor inhibitors (sirolimus and everolimus)
  - Decreased wound healing
  - Proteinuria
  - Hyperlipidemia
  - Pancytopenia
  - Azospermia

- Mycophenolate (myfortic and cellcept)
  - Leukopenia
  - Diarrhea
METABOLISM AND DRUG INTERACTIONS OF IMMUNOSUPPRESSANTS

• Tacrolimus is metabolized in the cytochrome P450 (CYP) 3A subfamily
  • Increased tacrolimus levels
    • ketoconazole, cyclosporine, diltiazem, erythromycin, and fluconazole
  • Decreased tacrolimus levels
    • Carbamazepine, phenytoin, rifampin, St. Johns wort

• Sirolimus is metabolized in the cytochrome P450 3A4 isozyme (CYP3A4)
  • Increase sirolimus levels
    • antifungal medications, clarithromycin, erythromycin, isoniazid, protease inhibitors
  • Decrease sirolimus levels
    • carbamazepine, phenobarbitol, phenytoin, rifampin
OBJECTIVES

• The state of nephrology
• Estimating glomerular filtration rate
• Chronic kidney secondary disease
• Acute kidney injury
• Renal transplant
THANK YOU

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