Case Studies

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Case Studies

Case Study 1

1. What is the most likely diagnoses for this patient and what are the criteria for this diagnosis? What is your rationale?

The most likely diagnosis for this patient is advanced idiopathic pulmonary artery hypertension (PAH) leading to right ventricular heart failure. PAH is a syndrome resulting in increased pulmonary vascular resistance (PVR) leading to right heart failure consequently from restricted flow in the pulmonary arterioles (McLaughlin et al., 2009).

Criteria for diagnosing PAH involve the physical assessment, risk factors, past medical history, symptomology, and diagnostic studies. Classic symptoms of PAH include; worsening exertional dyspnea, fatigue, chest pain, syncope, and edema. In this scenario, the new onset of dyspnea over the last seven months combined with chest x-ray (CXR) results showing cardiomegaly and a dilated pulmonary trunk suggests PAH. The distended neck veins with clear lung fields denote right ventricular involvement along with poor compliance suggestive of prominent a waves in the jugular venous pulse. Cyanotic lips suggest central cyanosis secondary to hypoxemia and is suggestive of late stage PAH (Rich, 2012). A prominent P2 heard upon auscultation is indicative of a high pressure system occurring during closure of the pulmonic valve and a right ventricular S4 heard implies high right ventricular pressures. The Grade III/VI holosystolic murmur located at the left sternal border augmented with inspiration, and left parasternal heave are significant findings indicating progression of this disease. The murmur points to tricuspid insufficiency based on the change with inspiration, also known as carvallo's sign. Pulmonary hypertension usually is the cause of tricuspid insufficiency secondary to increased right ventricular volumes from regurgitant flow (McLaughlin et al., 2009). A left

parasternal heave is suggestive of cor pulmonale which leads to right ventricular hypertrophy (RVH) over time from the increased resistance thus causing increased pressures in the pulmonary vasculature. The electrocardiogram (ECG) revealed significant findings indicating RVH or possible pulmonary embolus from the right axis deviation and right bundle branch block (prominent RSR). The p pulmonale secondary to right atrial enlargement is usually suggestive of primary or secondary pulmonary hypertension. ST depression and T wave inversion in the anterior precordial leads suggests ischemia. Symptomology pertaining to jugular venous distention, slight abdominal distention with a positive hepatojugular reflex and trace edema of ankles are indicative of advanced PAH with right ventricular heart failure (McLaughlin et al., 2009).

Another caveat to this diagnosis is the patient's home medication list. Anorexigenic agents such as fenfluramine can block serotonin reuptake and increase the release causing vasoconstriction and hypertrophy (Rich, 2012). This medication class has potential to increase the incidence for developing PAH. The World Health Organization (WHO) categorizes PAH into five groups encompassing; primary or idiopathic PAH (IPAH) (Group I), left heart disease (Group II), lung diseases and/or hypoxemia associated with PAH (Group III), chronic thrombotic/embolic diseases causing PAH (Group IV), and miscellaneous causes of PAH (Group V) (McLaughlin et al., 2009). Based on the WHO categories, this patient would fall under Group I PAH because of her anorexigen use as the primary cause. There are four functional classes of heart failure from the New York Heart Association (NYHA) that helps determine severity of heart disease. The symptomology of dyspnea with minimal exertion, fatigue at rest, and signs of right ventricular failure would place this patient in a NYHA functional class IV (Barst et al.,

2004). The patient may change functional classes at any point and often will occur during treatment of PAH or progression of disease.

2. What diagnostic test should be performed? Explain your rationale?

Further investigation is warranted if PAH is suspected from physical assessment findings, symptomology, history, and high risk factors. Diagnostic testing includes; CXR, ECG, echocardiogram, V/Q scan, pulmonary function test (PFT), overnight oximetry, lab serologies (HIV, ANA), hepatic panel, six minute walk test, and a right heart catheterization (RHC). The diagnostic tests will help rule out other possible diagnoses or determine potential underlying causes. Establishing the origin and excluding other disorders will facilitate treatment options and necessary adjunctive therapy. The CXR may show cardiomegaly or pulmonary congestion, however is often nonspecific. Obtaining a CXR remains important to determine other potential causes of symptomology. ECG results may reveal right heart strain, right ventricular hypertrophy (RVH), p pulmonale, right axis deviation and ST changes any which may prompt further diagnostic testing. An echocardiogram will help determine right atrial (RA) and right ventricle (RV) size (dilation) and function, chamber pressures, ejection fractions (EF), assessing for any wall abnormalities, displacement of the intraventricular septum and severity of disease. An echo will reveal if right heart failure is present from the calculated right ventricular ejection fraction. The echo will also expose any valvular heart problems such as suspected tricuspid insufficiency and can demonstrate turbulent flow with doppler studies (Rich, 2012). Several studies have shown sensitivity and specificity results of an echo predicting PAH are low compared to right heart catheterization (RHC) results (85% sensitivity and 55% specificity) (Arcasoy et al., 2003). PFT will help determine if an obstructive or restrictive lung disease is present contributing to

PAH. Advanced chronic obstructive lung disease is another cause of right heart failure, specifically cor pulmonale, and would need to be ruled out with spirometry (Barst et al., 2004).

The gold standard test/procedure for diagnosing PAH is a RHC. A RHC will provide conclusive data regarding pulmonary arterial pressures, right atrial pressures (RAP), and PVR all of which play a role in diagnosing PAH, severity of disease and progression. Confirmatory hemodynamics with a right heart catheterization include; mean pulmonary pressures (mPAP) greater than or equal to 25 mm Hg at rest without evidence of elevated pulmonary artery wedge pressures (PAWP) (PAWP < 15 mm Hg) combined with a PVR greater than 3 Wood units are indicative of PAH (McLaughlin et al., 2009; Sitbun et al., 2005). Determining a Fick cardiac output (CO) during the RHC instead of thermodilution CO (not as accurate in the presence of heart failure) will help calculate the PVR (Barst et al., 2004).

Cardiac biomarkers and specific laboratory values are helpful with predictors of PAH. Brain natriuretic peptide (ProBNP) is an independent prognostic value related to PAH survival rates. Elevated ProBNP levels indicate some type of RV involvement with PAH (enlargement or dysfunction). The level also denotes how severe the heart failure is and often remains higher in systolic heart failure when compared to diastolic heart failure (Barst et al., 2004). Serial increases in troponin levels with PAH can indicate RV ischemia leading to a poor prognosis. Additional laboratory tests can help determine potential causes and to obtain baseline values. The lab tests include; complete blood count, thyroid stimulating hormone level, uric acid and a hepatic panel to determine liver function (Rich, 2012). Uric acid levels obtained initially and throughout course of treatment can independently predict mortality as rising values are generally seen with hemodynamic instability (McLaughlin et al., 2009).

A six minute walk test will help determine exercise capacity and functional class setting standards for disease severity, progression of illness, and response to therapy. This test should be performed before and after initiation of treatment (McLaughlin et al., 2009). Improving functional capacity and limiting progression with pharmacological treatment remains the overall goal with PAH and a six minute walk test will objectively illustrate treatment outcomes (McLaughlin et al., 2009).

3. What is the appropriate therapy for this patient? Include all types of therapy and rationale for your choices.

The goal of therapy is to improve overall quality of life, survival, hemodynamics (mPAP, CO/CI, PAWP, and RAP) and exercise tolerance. Generally, therapy can be guided with acute vasodilatory testing using a short active agent (inhaled nitric oxide, epoprostenol, adenosine) via a RHC to determine treatment modalities, specifically if the patient is a candidate for calcium channel blocker therapy. A positive response to the vasodilator test often signifies a better prognosis for the patient. The results influence what treatment algorithm to follow established through guidelines by the American College of Chest Physicians (ACCP). A reduction in the mPAP by 10 to15 mmHg while remaining less than 40 mmHg, and a unchanged or improved cardiac output are indicative of a positive response to the vasodilator test dose (McLaughlin et al., 2009). A negative response to the vasodilator test dose changes the treatment towards more aggressive therapy. Although the acute vasodilatory test is helpful with directing treatment, this particular patient exerts symptoms of overt right heart failure. The guidelines suggest that vasodilator testing should be avoided in patients with signs and symptoms of heart failure as vasodilation from the test dose can decrease coronary perfusion pressures (McLaughlin et al., 2009). Also, calcium channel blockers are contraindicated in the presence of heart failure

secondary to its negative inotropic activity. Calcium channel blockers are indicated initially with a positive response and fall under the NYHA functional class I or II. However, with IPAH, many do not respond to this treatment and subsequently the treatment regimen is changed. The symptomology in this scenario signifies that heart failure is likely and calcium channel blockers are not recommended.

Prostanoids are considered as one of the core treatments prescribed for PAH, WHO Group I to improve symptoms and functional exercise capacity. With this case, prostanoids remain first line treatment due to the severity of the disease. This class includes; iloprost, epoprostenol, and treprostinil. Due to the severity of disease leading to right heart failure in this scenario, a continuous infusion of epoprostenol intravenously is the drug of choice to improve overall survival, hemodynamics, and exercise capacity (McLaughlin et al., 2009). Initially epoprostenol is started at 2 ng/kg/min with adjustments made based on symptomology and side effects from the medication. Optimal monotherapy dosing is between 20-40 ng/kg/min. Of all indicated treatments for PAH, epoprostenol remains the only medication that has shown to lengthen survival (Sitban et al., 2005). Possible combination therapy should be considered if the patient does not respond to the therapy. Imperative patient and family teaching include proper sterile technique when preparing medication and care of central line, what symptoms to report immediately and to seek emergency treatment if medication becomes empty without available back up supply. Abrupt cessation of this medication can cause profuse rebound PAH and subsequent death (McLaughlin et al., 2009).

Endothelin receptor antagonists (ERA) also show benefits in treating PAH; improving survival and exercise tolerance. Bosentan has shown promising results in reducing morbidity when compared to prostacyclin in a systematic review (Liu, Chen, Gao, Deng, & Liu, 2013).

Also, bosentan has shown to improve exercise capacity and hemodynamics among idiopathic PAH patients in functional class III and is prescribed as first line drug therapy in patients without advanced disease because of its oral preparation and efficacy (Sitban et al., 2005).

Phosphodiesterase inhibitors are a class of medications used for PAH increasing cyclic guanosine monophosphate concentration causing pulmonary bed vasodilation and relaxation (LexiComp, 2013). Sildenafil has shown to reduce mPAP (McLaughlin et al., 2009). Phosphodiesterase inhibitors or ERA's are recommended in combination with prostanoids when right heart failure is present and not responding to one treatment alone.

Tertiary prevention measures are essential to limit the progression of this disease. To start with, patient teaching to include stopping any and all anorexigens, quit smoking, and discontinuing oral contraceptives while still using preventative measures avoiding pregnancy. The smoking and use of oral contraceptives alone increase her risk significantly for deep vein thrombosis and pulmonary embolism. With PAH, her risk considerably increases and will require pharmacological treatment to reduce the incidence of clot formation along with removing other contributable risk factors.

Anticoagulation can improve overall survival in idiopathic PAH caused by anorexigens. Oral anticoagulation therapy should be initiated and permanently maintained for prevention of thromboembolism in this case secondary to her smoking history and oral contraceptive use. A dilated RA that occurs secondary to a dilated RV can precipitate atrial arrhythmias, specifically atrial fibrillation, contributing to the risk of developing clots. Warfarin is the drug of choice for anticoagulation according to the ACCP guidelines (McLaughlin et al., 2009). Based on the patient's initial international normalized ratio (INR) will identify the starting dose. Maintaining

an INR of 1.5-2.5 with warfarin is recommended, thus preventing thromboembolism formation and further worsening of heart failure (McLaughlin et al., 2009).

Digoxin is recommended to improve cardiac function with overt right sided heart failure. As a positive inotrope, digoxin improves contractility thus increasing CO/CI. Also, digoxin acts as an antiarrhythmic agent preventing arrhythmias from occurring often due to a dilated RA (McLaughlin et al., 2009). Based on the symptoms reported, this patient may be a candidate for digoxin. The echocardiogram and RHC would provide useful information depicting how severe the right heart failure is to decide whether or not adding digoxin to the treatment regime is necessary.

Right ventricular (RV) function assists with prediction of functional capacity and overall prognosis. PAH leading to right heart failure indicates a worsened outcome, thus medical management is essential. Diuretics are recommended in patients who demonstrate right heart failure (elevated jugular venous pressure, abdominal distention and edema) to decrease preload, reducing the amount of blood volume which leads to improvement of RV contraction (McLaughlin et al., 2009). Renal function and electrolytes should be monitored throughout diuretic therapy.

Oxygen supplementation is recommended if needed to keep oxygen saturation levels greater than 90%. This can alleviate some hypoxemia that may be involved with PAH worsening PVR (McLaughlin et al., 2009). Oxygen is often included in the treatment regimen for PAH using combination therapy to improve symptoms and limit progression.

An advanced practice nurse (APN) with a current certificate to prescribe (CTP) can prescribe calcium channel blockers, oxygen, digoxin, and warfarin (per institutional standards) (Ohio Board of Nursing, 2013). An APN with a CTP may also prescribe an ERA,

phosphodiesterase inhibitor, and prostanoids with physician consultation or initiation in a specialty clinic and must be noted on the standard care arrangement with the collaborating physician (Ohio Board of Nursing, 2013).

Case Study 2

1. What is your differential diagnosis? Explain.

Differential diagnoses include; contrast-induced nephropathy (CIN), pre-renal acute kidney injury (AKI), acute tubular necrosis (ATN), intrinsic AKI, and post-renal AKI. Oliguria and an elevated creatinine level illustrated in this scenario suggest some form of AKI.

CIN is commonly caused by exogenous nephrotoxins such as intravenous contrast as described in this case given during coronary angiography. Populations and disease states that increase the risk of CIN include; diabetics, elderly individuals, chronic kidney disease, heart failure, and anemia. This adverse complication is defined by a rise in creatinine 25% or an increase in serum creatinine greater than 0.5 mg/dl above baseline that occurs within 48 to 72 hours after intravenous (IV) radiographic contrast is administered for the procedure/diagnostic test (Leopold & Faxon, 2013). The mechanism of injury from the IV contrast causes renal tubule vasoconstriction and cell damage, thus impairing vasodilation of the renal vasculature. The baseline creatinine of 1.6mg/dL which is slightly above a normal serum creatinine for a male (normal <1.2mg/dL for a male) in this case shows previous underlying renal impairment adding to renal insult from IV contrast given during coronary angiography. Also, this patient is a type II diabetic which increases his chance of having this adverse complication of CIN. Symptoms of diabetic retinopathy (dot hemorrhages and hard exudates of the fundus) generally indicate poor blood glucose control (Riordan-Eva, 2013). The patient has a history of coronary artery disease (CAD) which is usually a systemic problem affecting the kidneys either directly causing renal

artery stenosis or exists as a common area for atheroembolic disease (Libby, 2013). An S4 gallop often indicates some form of diastolic dysfunction resulting from active myocardial ischemia potentiating the risk of CIN after coronary angiography. Also, the patient was treated with an ACE inhibitor which can decrease the glomerular filtration rate (GFR) by causing efferent arteriolar vasodilation and with pre-existing renal compromise can cause hyperkalemia (Lin & Denker, 2013). Angiotensin II maintains glomerular capillary hydrostatic pressure keeping the GFR up through vasoconstriction in states of hypovolemia; however an ACE inhibitor halts this process adding to reduction of GFR in a hypovolemic state (Lin & Denker, 2013). Bilateral renal artery stenosis also should be assessed via renal ultrasound as an ACE inhibitor can potentiate worsening of this problem and is contraindicated if confirmed. Aspirin blocks prostaglandin synthesis which is a vasodilator thus causing vasoconstriction of the renal vasculature (Lin & Denker, 2013). All of these profound contributing factors pose a risk for CIN after coronary angiography.

Other differentials would need to be ruled out prior to committing to the diagnosis of CIN. Pre-renal AKI can be caused from decreased perfusion to the kidneys from hypotension (Lin & Denker, 2013). The patient in this case study was receiving IV nitroglycerin (a potent venous vasodilator) potentially causing hypotension. This patient at baseline has hypertension based on the symptoms of worsening hypertension described in this scenario. In light of this information, the patient's kidneys are likely use to higher blood pressures and a drop in blood pressure caused by nitroglycerin can decrease the perfusion to the kidneys. Though this is a possibility and should be investigated as a cause of pre-renal AKI, a more likely cause of AKI is CIN.

Intrinsic AKI can be caused by prolonged hypoperfusion of the kidneys, sepsis, ischemia,

glomerulonephritis, interstitial nephritis, endogenous and exogenous nephrotoxins. ATN is a form of intrinsic AKI resulting from ischemia and nephrotoxins. Ischemia in the kidney can develop from hypoperfusion states if not corrected leading to ATN. Severe sepsis will develop into a low flow state thus decreasing perfusion to the kidneys (ischemia). High myoglobin levels (rhabdomyolysis) from muscle breakdown results in tubule obstruction from the large particles may also cause intrinsic AKI. Additionally, glomerulonephritis and interstitial nephritis are other disease states that can result in intrinsic AKI from a reduced GFR, proteinuria, and alterations in excretion of sodium (Lin & Denker, 2013).

There are limited findings listed in this case scenario suggesting post-renal AKI and will likely be ruled out once laboratory test results are available. Hematuria may be present on a urinalysis depending on the cause. Post-renal AKI is caused by an obstruction of urinary flow usually from a partial or total obstruction in the renal pelvis or ureter (Harris & Neilson, 2013). The information provided (recent coronary angiography, type II diabetic, CAD) in this case points to a separate diagnosis.

2. What is your next step to diagnose the problem? Explain. Of what value is a urinalysis and urinary electrolytes?

The next step is to distinguish a diagnosis among the differentials listed above through process of elimination. In order to rule out other potential causes of AKI, laboratory tests can point to a diagnosis. A urinalysis and urine electrolytes are beneficial in ruling out pre-renal, intrinsic, and post-renal AKI. More specifically both of these tests can be helpful in distinguishing between pre-renal azotemia and pre-renal ATN. Renal ultrasound is indicated if post-renal AKI is suspected to determine the presence of ureteral and renal pelvic dilation (Lin & Denker, 2012).

Urinalysis results entail; urine specific gravity, urine osmolality, urinary sediment, casts, red blood cells, white blood cells and if protein excretion is present. With pre-renal AKI the urinalysis may be clear or show hyaline casts. An abnormal urinalysis showing sediment, granular, muddy brown, renal tubule epithelial cell, or mixed casts and excretion of protein is usually indicative of ATN. Urinary sediment usually shows granular and epithelial casts if caused by a tubular injury resulting from IV contrast. Red blood cell and white blood cell casts can point to other causes of kidney failure from either intrinsic renal disease or glomeruli disease states (Lin & Denker, 2013). Urine osmolality measures solute concentrations of particles per kilogram and generally correlates with urine specific gravity. An increase in urine specific gravity greater than 1.020 is often a marker of large particles caused by IV contrast. A cohort study revealed a high specificity using a urinalysis to find various casts (90-96%) with a low sensitivity (6.6-14.5%) (Schinstock et al., 2012). In this same study, a higher sensitivity was found using neutrophil gelatinase-associated lipocalin for early detection of AKI (66.7%) (Schinstock et al., 2012).

Urine electrolytes determine if the kidney failure is related to pre-renal or ATN ensuring a correct diagnosis. Urinary electrolytes can provide information to obtain a fractional excretion of urea (FeNa) result. A FeNa less than 1% is usually indicative of pre-renal AKI and a FeNa greater than 1-2% is present in ATN. The FeNa equation utilizes urine electrolytes to obtain a result (Lin & Denker, 2013). In one study, sensitivity and specificity accounted for 78% and 75% using FeNa as a diagnostic test to confirm pre-renal AKI versus ATN (Pepin, Bouchard, Legault, & Ethier, 2007). The urine sodium in pre-renal AKI is usually less than 20mEq/L whereas it is usually greater 20mEq/L in ATN. Overall, the clinical picture combined with clinical findings will help facilitate diagnosing the type of AKI.

3. What are the indications for dialysis in AKI (acute kidney injury)? Be specific.

Electrolyte abnormalities, severe metabolic acidosis, uremia with signs and symptoms, volume overload despite use of diuretics and toxic ingestions all indicate the possible need for dialysis (Harris & Neilson, 2013). Marked hyperkalemia with electrocardiogram changes suggests the need to lower potassium levels. Although hyperkalemia can be corrected temporarily with medications (dextrose and insulin, albuterol, kayexelate, and high doses of loop diuretics), the underlying problem needs to be addressed. Severe metabolic acidosis (pH < 7.0) is life threatening and warrants immediate attention/correction to prevent untoward complications. Uremia causing mental status changes or seizures are symptoms often caused by severe electrolyte disturbances requiring immediate attention. Hemodynamic instability from any of the above indications may facilitate further need for imminent dialysis (Harris & Neilson, 2012).

4. Write a set of admitting orders for the patient. Be specific.

Admission:

- Admit to the coronary care unit (CCU)
- Consult nephrology

Diagnosis:

- Contrast Induced Nephropathy (CIN)
- AMI s/p cardiac catheterization/coronary angiography
- Secondary diagnoses: Type II diabetes, diabetic retinopathy and CAD

Condition:

- Serious

Vital Signs:

- Continuous cardiac monitoring.

- Obtain HR, respirations, BP, and SPO2 Q1hr.
- Obtain temperature Q4 hours
- ECG stat

Allergies:

- No known drug allergies (NKDA)

Activity:

- As tolerated
- Out of bed TID
- Ambulate in hallway with continuous cardiac monitoring

Nursing:

- Place foley catheter
- Daily weights at 0500
- Monitor FSBG AC & HS
- Encourage coughing and deep breathing along with incentive spirometry Q1 hour while awake

Diet:

- Diabetic 1800cal/renal/low sodium
- Specifically 20-30 kcal/kg/day
- 0.8-1.0 g/kg/day of protein restriction (helps prevent metabolic acidosis) (Palevsky et al., 2013)

Intake & output:

- Monitor and document urine output Q1hr
- Strict I/O documentation Q8 hours

- Call NP if low urine output < 30 ml/hr

Medications:

- Normal saline 0.9% @ 100ml/hr for 12 hours, then decrease to KVO
- Nitroglycerin gtt (25mg/250ml NS) via non PVC tubing increase or decrease titration of gtt based on chest pain relief (max dose 200mcg/min) (Hold for MAP < 60 or HR > 120 and call NP) (call NP if worsening chest pain or no relief)
- Metoprolol 12.5mg po BID (hold for HR < 60bpm and/or MAP <60)
- ASA 81mg po daily
- Atorvastatin 40mg po QHS
- Heparin 5000 units subcutaneous Q12 hours
- Senna S (1 tablet) po BID
- Colace 100mg po BID
- Subcutaneous regular insulin high sliding scale AC & HS: Goal blood glucose 110-150 mg/dL

Fasting Blood Glucose (mg/dL)	Dose
<70	Hypoglycemia protocol
70-110	0
110-150	2 units
151-190	4 units
191-230	6 units
231-260	8 units
261-300	10 units

301-350	12 units
>350	Give 14 units and call practitioner

- Discontinue lisinopril
- Avoid nephrotoxic agents: diuretics, ACE inhibitors/ARBs, contrast, NSAIDS

PRN Medications:

- Morphine 2-4mg IV Q2 hours (hold for RR <12bpm, MAP < 60)
- Zofran 4mg IV Q6hrs prn nausea
- Temazepam 15mg po QHS prn insomnia

Labs:

- CBC with Diff now and then daily
- Renal, Mg, PO4 now and then Q12 hours
- Lipid profile stat
- Troponin Q6 hours X 3
- Urinalysis stat
- Urine electrolytes stat
- Urine microalbumin (send to lab)
- Hgb A1C

Notify CCU team if:

- HR <55bpm or >120bpm
- MAP <60
- SBP >160 or < 90

- SPO2 < 90%
- Respirations < 8bpm or > 30bpm
- Temperature > 101.5˚F or < 96˚F

Special:

- Maintain SPO2 > 90%
- Renal US to rule out bilateral renal artery stenosis and/or renal obstructions

Please call the CCU pager at 230-9219 with any questions or concerns.

Thank you,

Kimberley Roberts, ACNP student

5. Complete the following chart with values present in prerenal and acute renal failure. Be certain to reference the chart.

Table 1. Differential Criteria Values for Pre-renal and Acute Renal Failure

Laboratory Test	**Pre-renal**	**Acute Renal Failure**
FeNa	<1%	>2%
BUN to creatinine ratio	>20:1	10-15:1
Urine specific gravity	>1.020	1.010-1.020
Urine osmolality, mOsm per kg	>500 mOsmol	<350 mOsmol
Urine sodium concentration, mEq per L (mmol per L)	<20 mEq/L	>40 mEq/L
Urine sediment	Usually not present or may reveal hyaline casts	Granular, muddy brown, renal tubule epithelial cell, or mixed casts

Table 1. Adapted from: Lin, J., & Denker, B. (2013). Azotemia and urinary abnormalities. In D. Longo, A. Fauci, D. Kasper, S. Hauser, J. Jameson, & J. Loscalzo, *Harrison's principles of internal medicine*. New York: McGraw-Hill.

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