SAWA UNIVERSITY

جامعة ساوة الاهلية
كلية التقنيات الصحية والطبية
قسم التخدير - اللجنة العلمية

التخدير-كورس اول

RENAL DISEASE AND ANESTHESIA

Sawa University — جامعـــــة ســـــــاوة الاهلية

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3rd Stage cours 1 — المرحلـــــــــــة الثالثة.

محاضرة رقم 6

Lecture No. 6

نظـــــــري

Theoretical

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Renal Disease and Anesthesia

Normal renal function is important for the excretion of anesthetics and medications, maintaining fluid and acid-base .balance, and regulating hemoglobin levels in the perioperative period

The kidneys play a vital role in

,Regulating the **volume** and **composition** of body fluids -

Eliminating toxins, and -

Elaborating hormones, including **renin**, **erythropoietin**, and the **active** form of **vitamin D** -

Factors related to operative procedures and to - anesthetic management frequently have a significant **:** impact on kidney physiology and function and **may lead to**

,**Perioperative fluid overload -**

:**Hypovolemia, and acute kidney injury, which are major causes of -**

.Perioperative morbidity, mortality, extended hospital length of stay, and increased costs

Effects of Anesthesia & Surgery on Kidney Function

Acute kidney injury -1

Acute kidney injury (AKI) is a common and underappreciated perioperative problem, occurring in 1% to 5% of all hospitalized patients and in approximately 50% of all ICU .patients

AKI is a systemic disorder that can include fluid and electrolyte derangements, respiratory failure, major cardiovascular events, weakened immunocompetence leading to infection and sepsis, altered mental status, hepatic dysfunction, and .gastrointestinal hemorrhage. It is also a major cause of chronic kidney disease (CKD)

- The risk of perioperative AKI is also increased by exposure to nephrotoxic agents such as nonsteroidal anti-inflammatory drugs (NSAIDs), radiocontrast agents, and antibiotics. The clinician must possess a thorough understanding of the risks of AKI, its differential diagnosis, and its evaluation strategy
- AKI is a major contributor to increased hospital length of stay, markedly increasing morbidity, mortality, and cost of care.
- Patients may develop AKI and kidney failure secondary to intrinsic kidney disease.

Risk factors for AKI in the perioperative setting include:

1. Preexisting kidney impairment
2. Diabetes mellitus
3. Cardiovascular disease
4. Hypovolemia
5. The use of potentially nephrotoxic medications by older adult patients

TABLE 1

Definition and staging of acute kidney injury according to the AKIN criteria

Stage	Creatinine concentration	Urine output
1	1.5–1.9 × baseline or ≥ 0.3 mg/dL	<0.5 mL/kg/h for 6–12 h
2	2.0–2.9 × baseline	<0.5 mL/kg/h for >12 h
3	≥ 3.0 × baseline or ≥ 4 mg/dL or dialysis	<0.3 mL/kg/h for ≥ 24 h or anuria for ≥ 12 h

AKIN, Acute Kidney Injury Networks

Chronic Kidney Disease: CKD is defined as either kidney damage or a GFR less than 60-2 mL/min for 3 months or more. Kidney damage is defined as a pathologic abnormality or markers of damage including abnormalities of the blood or on urine or imaging studies.*

Diagnosis of Chronic Renal Insufficiency

Oliguria does not set in until late in the disease and is an unreliable marker of disease progression. confirmed by laboratory testing.

Classification of Chronic Renal Disease:

Stage 1: Kidney damage with normal or GFR (90 ml/min)

Stage 2: Kidney damage with mild GFR (60-89 ml/min)

Stage 3; Moderate GFR (30-59 ml/min)

Stage 4: Severe GFR (15-29 ml/min)

Stage 5: Kidney failure with GFR

Causes of renal failure

Diabetes Mellitus 25% - 1

Glomerulonephritis 14% - 2

Hypertension 8% - 3

Polycystic kidney disease 6% - 4

Pyelonephritis 6% - 5

Renal vascular disease 6% - 6

Others 17% - 7

Uncertain 15% - 8

Systemic effects of renal failure

:Cardiovascular system

Left ventricular hypertrophy -

Atherosclerosis -

Hypertension -

:Respiratory system

Pulmonary edema -

Metabolic acidosis

Coagulopathy

Autonomic neuropathy

:**Fluid and electrolyte**

Volume overload -

Hyperkalemia -

Altered Kidney Function & the Effects of Anesthetic Agents

INTRAVENOUS AGENTS

Propofol & Etomidate

The pharmacokinetics of both propofol and etomidate are minimally affected by impaired kidney function. Decreased protein binding of etomidate in patients with hypoalbuminemia may enhance its .pharmacological effects

Barbiturates

Patients with kidney disease often exhibit increased sensitivity to barbiturates during induction, even .though pharmacokinetic profiles appear to be unchanged

The mechanism appears to be an increase in free circulating barbiturate secondary to decreased protein binding. Acidosis may also favor a more rapid entry of these agents into the brain by increasing the .nonionized fraction of the drug

Ketamine

Ketamine pharmacokinetics are minimally altered by kidney disease. Some active hepatic metabolites are .dependent on renal excretion and can potentially accumulate in kidney failure

Benzodiazepines

Benzodiazepines undergo hepatic metabolism and conjugation prior to elimination in urine. Because they are highly protein bound, increased benzodiazepine sensitivity may be seen in patients with hypoalbuminemia. Diazepam and midazolam should be administered cautiously in the presence of kidney .impairment because of the potential for the accumulation of active metabolites

Opioids

Most opioids used in anesthetic practice (morphine, meperidine, fentanyl, sufentanil, and alfentanil) are inactivated by the liver; some of these metabolites are then

excreted in urine. Remifentanil pharmacokinetics are unaffected by kidney function due to rapid ester .hydrolysis in blood

With the exception of morphine and meperidine, significant accumulation of active metabolites generally does not occur with these agents. Accumulation of morphine (morphine-6-glucuronide) and meperidine (normeperidine) metabolites may prolong respiratory depression in patients with kidney failure, and increased levels of normeperidine may promote seizure activity. The pharmacokinetics of the most commonly used opioid agonist–antagonists (butorphanol, nalbuphine, and buprenorphine) are unaffected .by kidney failure

INHALATION AGENTS

Volatile Agents

Volatile anesthetic agents are ideal for patients with kidney disease because they are not dependent on the kidneys for elimination and they have minimal direct effects on kidney blood flow. Although patients with mild to moderate kidney impairment do not exhibit altered uptake or distribution, accelerated induction and emergence may be seen in severely anemic patients (hemoglobin <5 g/dL) with chronic kidney failure, possibly because of a decrease in the blood:gas partition coefficient. Some clinicians avoid sevoflurane (and avoid <2 L/min gas flows) for patients with kidney disease who undergo lengthy .procedures

MUSCLE RELAXANTS

Succinylcholine

Succinylcholine can be safely used in patients with kidney failure in the absence of hyperkalemia at the time of induction. It should be avoided in patients with kidney failure when the serum potassium is .known to be increased or is undetermined

Although decreased plasma cholinesterase levels have been reported in uremic patients following dialysis, significant prolongation of neuromuscular blockade with succinylcholine use is rarely seen in this .circumstance

Cisatracurium & Atracurium

Cisatracurium and atracurium are degraded by plasma ester hydrolysis and nonenzymatic Hofmann elimination. These agents are often the drugs of choice for muscle relaxation in patients with kidney failure, especially in .clinical situations where neuromuscular function monitoring is difficult or impossible

Vecuronium & Rocuronium

The elimination of vecuronium is primarily hepatic, but up to 20% of the drug is eliminated in urine. The effects of large doses of vecuronium (>0.1 mg/kg) are only modestly prolonged in patients with kidney disease. Rocuronium primarily undergoes hepatic elimination, but prolongation in patients with severe kidney disease has been reported. In general, with appropriate neuromuscular monitoring, these two agents can be used with .few problems in patients with severe kidney disease

Pancuronium

Pancuronium is primarily dependent on renal excretion (60–90%). Although pancuronium is metabolized by the liver into less active intermediates, its elimination half-life is still primarily dependent on renal excretion (60–80%). Neuromuscular function should be closely monitored if pancuronium is used in patients with abnormal

kidney function

Reversal Agents

Renal excretion is the principal route of elimination for edrophonium, neostigmine, and pyridostigmine. The half-lives of these agents in patients with kidney impairment

are therefore prolonged at least as much as any of the above relaxants

	Drugs safe in CRF	Drugs safe in limited or reduced doses	Drugs contraindicated in CRF
Premedication	Lormetazepam, midazolam, temazepam		
Induction	Propofol	Ketamine, etomidate, thiopental	
Maintenance	Isoflurane, desflurane, halothane, propofol	Sevoflurane	Enflurane
Muscle relaxants	Suxamethonium, atracurium, cisatracurium	Vecuronium, rocuronium	Pancuronium
Opioids	Alfentanil, remifentanil	Fentanyl, morphine	Pethidine, codeine, tramadol
Local anaesthetics	Bupivacaine, lidocaine (reduce dose by 25%)	Oxycodone	
Analgesics	Paracetamol		NSAIDs

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Anesthetic considerations •

:Pre-operative Assessment •

.Routine anesthetic assessment along with special attention to renal functions is made -

.Hypertension and ischemic heart disease are commonly seen in chronic renal failure -

.Proteinuria and hypoalbuminemia predispose to edema -

.Urinalysis is a cheap, readily available, and informative laboratory test -

A complete blood count may reveal anemia, other causes of the anemia include excessive -
.hematuria, and reduced production of erythropoietin by failing kidneys

.Chest X-ray and ECG may be required -

:Summary of pre-operative assessment

Patients should be optimized in the preoperative period, hypertension should be managed with anti-hypertensives, antibiotic coverage for urinary infections. Routine transfusion is not recommended in chronic kidney disease and may predispose to CHF. Electrolytes should be corrected appropriately and dialysis may be needed in severe renal failure. Pre-medications may be necessary and antacids prophylaxis may be considered

• *Intra-operative:*

* open or laparoscopic renal surgery, general anesthesia with positive pressure ventilation using - .muscle relaxation is recommended

.Rapid sequence intubation is preferred in patients with chronic renal failure -

Induction of anesthesia may be achieved with intravenous and inhalational agents. Maintenance of - anesthesia is achieved with inhalational agents. The induction agent of choice in renal disease is .Propofol as it is metabolized by the liver and its excretion is not renal dependent

.Atracurium is the preferred muscle relaxant as it is metabolized by Hoffman degradation -

- A large-bore intravenous line is required as there may be a sudden risk of bleeding.

A limb with an arteriovenous fistula must not be used for intravenous infusions -

:Monitoring

Routine standard monitoring is a must. Patients with end stage disease may require central venous pressure monitored fluid administration. Temperature monitoring is required as renal surgery may take .many hours. Warm intravenous fluids and a warming blanket may be used

- ***Fluid therapy:***

Patients may be dehydrated as they are given bowel preparation and may be on dialysis, particularly in old age individuals. Appropriate fluid resuscitation to avoid sudden hypotension at induction with crystalloid fluid aiming for urine output should be 0.5-1 ml/kg/h is required in patients with signs and symptoms of dehydration

. ***Post-operative pain relief:***

There can be significant pain, especially in an open approach to kidneys. Multimodal analgesia is required for early mobilization and to reduce the incidence of postoperative pulmonary complications. Epidural analgesia should be used

unless contraindicated. Regional analgesia is contraindicated in presence of coagulopathy, thrombocytopenia, anticoagulation, or recent hemodialysis. Fentanyl and other short-acting opioids are useful as they are largely metabolized in the liver. Nonsteroidal anti-inflammatory drugs are contraindicated because of their nephrotoxic potential. Paracetamol is a safe drug and is a good adjuvant .analgesic

Any questions?