

# How to manage renal colic

김형준 (건양의대)

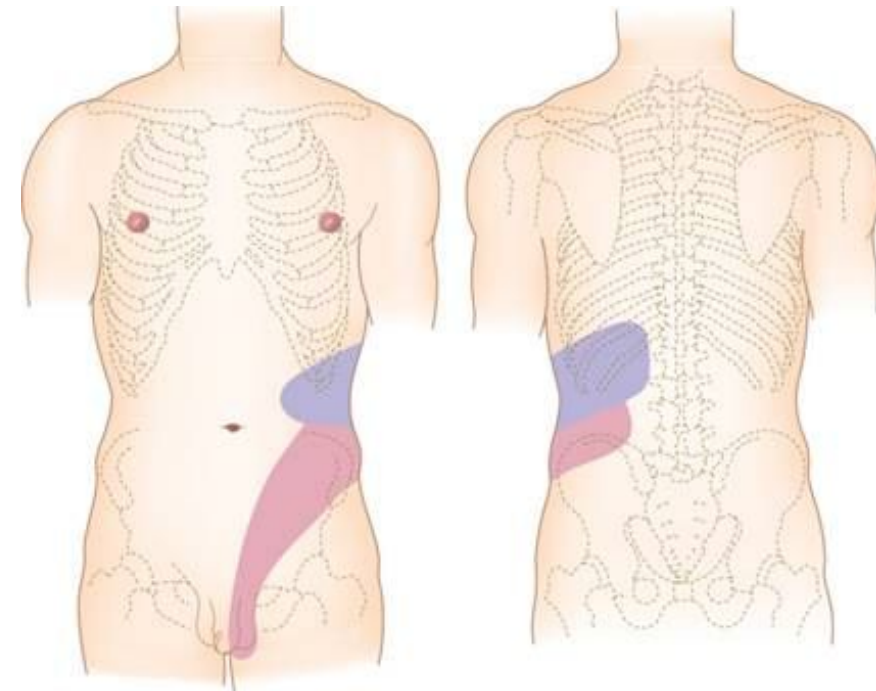
제1회의장 (김대중 컨벤션홀)  
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# What is renal colic?

- Acute and severe loin pain caused by renal stones obstructing urinary flow
  - It is only when stones pass into and obstruct the ureter that they cause colic
- Thereby, ureteric colic is probably the accurate term

- Annual incidence : 3 cases per 1000
- 12% of men and 6% of women will have one episode of renal colic at some stage in their life (Curhan, 2007).

Colic / Colicky pain  
Spasmodic pain from a tubular hollow viscus  
Muscular contraction of hollow tube such as colon, ureter or gallbladder, etc.



# Classical presentation of renal colic

- Colicky and constant ache felt in the loin, and which radiates to the groin or scrotum
  - Intermittent pain occurs as a result of renal capsular distension and ureteric smooth muscle contraction lasting minutes to hours, interspersed with pain-free intervals
  - The severity of pain does not relate to the size of the stone
  - hematuria (erythrocytes >10) are the most significant predictors of acute renal colic
  - Often accompanied by nausea, vomiting and hematuria (Patients are usually restless and cannot get comfortable, unlike in peritonitic conditions where patients tend to remain completely still.
  - Signs of sepsis, including fever, tachycardia and hypotension, may indicate an obstructing stone with infection, necessitating intervention
- ✓ Location of stone and pain characteristics
- Upper ureter ipsilateral testicular pain
  - Mid ureter may simulate appendicitis on the right side or diverticulitis on the left side
  - Lower ureter : lower-quadrant pain radiating to the tip of the urethra, urinary urgency and frequency, and dysuria are characteristic, mimicking the symptoms of bacterial cystitis

# Causes of renal colic

- Most common - urinary tract stone
- Other causes
  - Intrinsic - blood clot, papillary necrosis etc.
  - Extrinsic – lymphadenopathy (mild and more chronic pain)

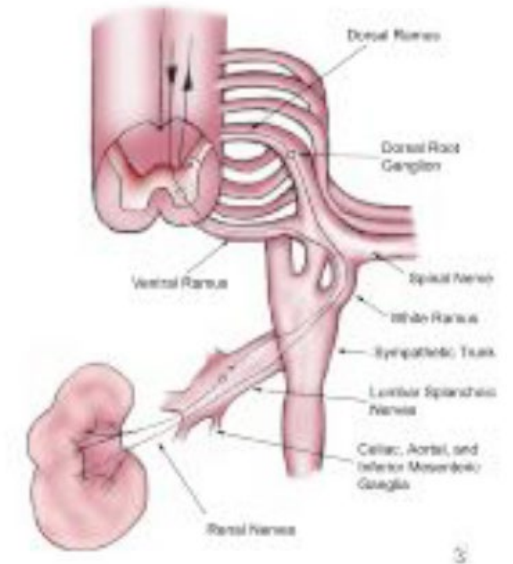


## Differential diagnosis of renal colic

Differential	Features in history and examination
Pyelonephritis	Fever and tender kidney (obstruction with sepsis is an emergency; if obstruction is suspected, immediate imaging is required)
Musculoskeletal pain	Worse with movement
Appendicitis	Tenderness or peritonism in right iliac fossa
Cholecystitis	Worse with eating fatty foods, tenderness in right upper quadrant
Diverticulitis	Associated bowel symptoms, usually tender in left iliac fossa
Leaking abdominal aortic aneurysm	Older age, vascular risk factors
Testicular torsion	Tender testis on examination
Gynaecological problems (for example, ovarian pathology, ruptured ectopic pregnancy)	Younger age, pelvic pain

# Pathophysiology

- Once renal stone get stuck in the ureter,
  - Increase in the intraluminal pressure from ureteric obstruction stretches nerve endings in the mucosa and produces the colicky pain
  - If the stone becomes lodged and unable to move, these muscles go into spasm  
→ Smooth muscle in the wall of the ureter contracts as it tries to move the stone
  - Prolonged isotonic contraction leads to increased production of lactic acid which irritates both slow-type A and fast-type C –fibers
  - Afferent impulses are generated that travel to the spinal cord at the T11-L1 levels, with subsequent projections to higher levels of the central nervous system.
  - This pain can be perceived in any organ sharing the urinary tract innervation such as the gastrointestinal organs and other components of the genitourinary system



# Acute ureter obstruction

- First 1.5 h renal pelvic pressure and renal blood flow both increase
- Next 4 h, renal pelvic pressure remains high but renal blood flow starts to decline
- After that, decline in both renal pelvic pressure and renal blood flow

1) Initial high renal pelvic pressure – prostaglandins : preglomerular vasodilatation

→ diuresis that further increases renal pelvic pressure

Nitric oxide - reduce preglomerular vascular resistance

2) Decrease in renal blood flow ← increase in intrarenal resistance caused by preglomerular vasoconstriction

- Mediators of vasoconstriction : No consensus but candidates are angiotensin II, thromboxane A<sub>2</sub>, antidiuretic hormone and endothelin

3) Decline in ureteric pressure ← decrease in glomerular filtration rate and an increase in the venous and lymphatic reabsorption of urine

- GFR ↓ : ↓ net hydraulic pressure gradient across the glomerular capillaries  
↑ tubular pressure caused by the increase in ureteric pressure

# Treatment

- Two key concepts
  - 1) Effective pain control
  - 2) Maximal preservation of renal function by release of ureteric obstruction

# Analgesics

- Non-steroidal anti-inflammatory drugs (NSAIDs) and Opiates are the mainstay of treatment
- NSAIDs : preferred analgesia in acute renal colic
  - longer duration of efficacy, reduced need for further analgesia compared to opiates
  - fewer side effects compared to opioids and paracetamol
- Thus, NSAIDs should be used as first line analgesia unless a patient has a contraindication to their use  
(e.g. history of peptic ulceration, known or suspected renal impairment, severe asthma)
- After emergent relief of renal colic, oral Tx with diclofenac is effective as short-term prophylaxis of new colic episode especially during the first 4 days.
- IM, IV, Oral, Rectal, Sublingual : Former routes have best analgesic effect.



# NSAID

- Block prostaglandin-induced effect

(afferent arteriolar vasodilatation which causes diuresis and pelvic pressure)

- Reduce local edema and inflammation, inhibit the stimulation of ureteral smooth muscle which is responsible for increased peristalsis and subsequently increase ureteric pressure

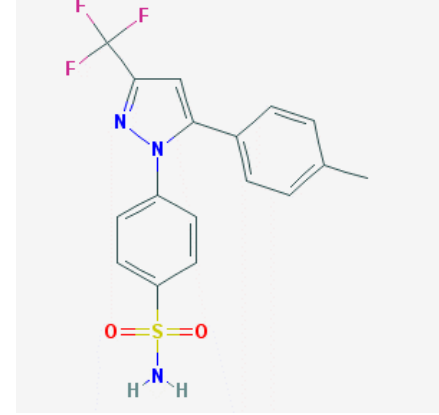
- Potentially interfere with the kidney's autoregulatory response to obstruction with marked reduction of renal blood flow

- Side effects

- NSAIDs can further diminish renal function in patients with an obstruction, particularly those with preexisting renal impairment

- Serious gastrointestinal S/E

# COX-2 inhibitors



- COX-2 inhibitors can prevent the PGE- mediated downregulation of aquaporin channels and major sodium channels in response to obstruction
- Decrease in hydrostatic pressure within the tubules may provide an additional mechanism for the NSAID-mediated reduction in intrapelvic pressures

Low GI side effect

Inhibits renal vasoactive substances

# Other analgesics

- Paracetamol

Showed benefit over opioids

Intravenous paracetamol should be offered if NSAIDs cannot be used or have not been effective.

- Opioids

- There was no benefit of opioids for pain relief over NSAIDs or paracetamol.

Only be considered if both NSAIDs and intravenous paracetamol were contraindicated or not effective.

- Antispasmodics

- No benefit in terms of pain relief when compared with NSAIDs

- Desmopressin

- Synthetic analogue of vasopressin → antidiuretic effect, suppress pelvic muscular contractility

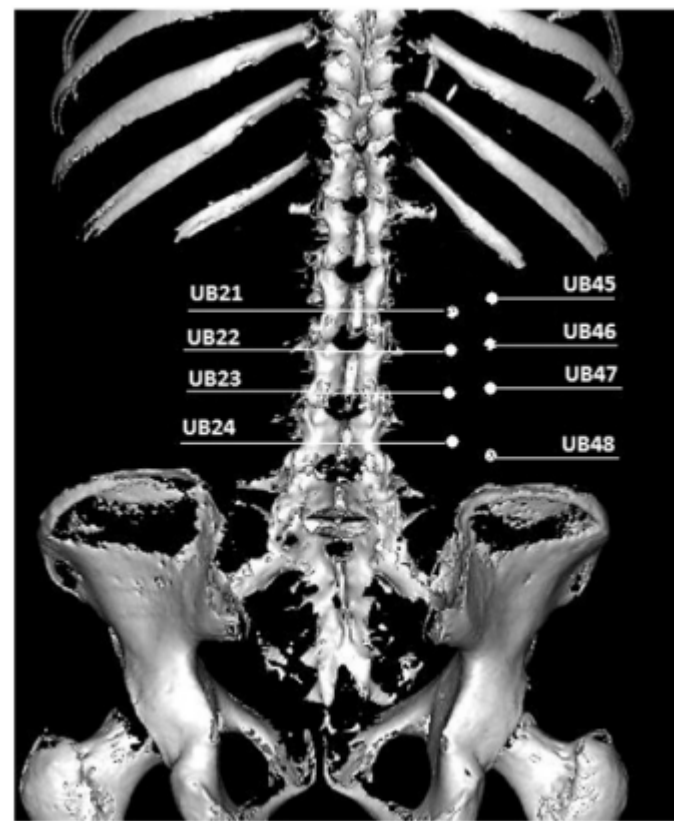
- Diuresis

- Discouraged in the presence of obstruction

(increase intraluminal pressure → reduction of effective peristalsis)



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## Original Contribution

### Comparison of the efficacy of diclofenac, acupuncture, and acetaminophen in the treatment of renal colic ☆

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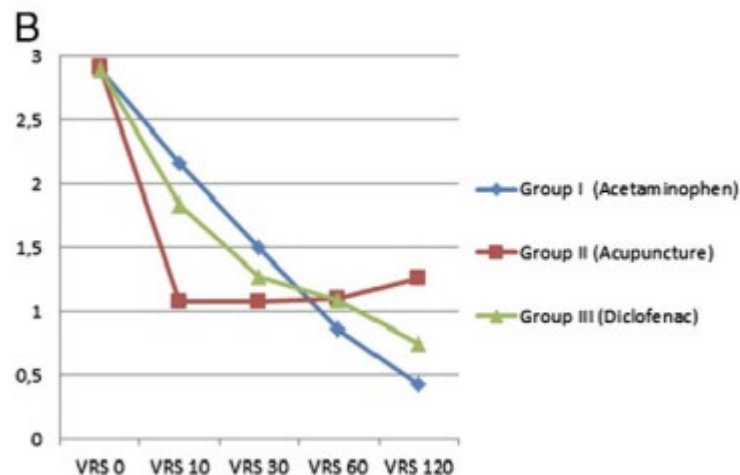
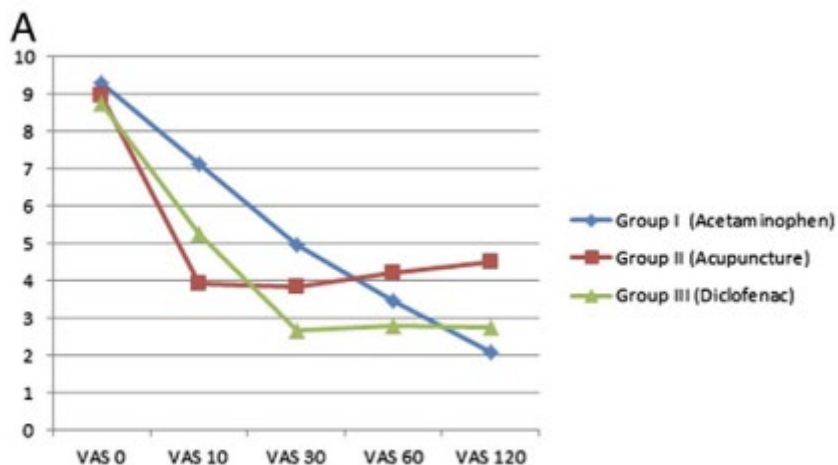
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Class and Name of Drug	Adult Dose	Adverse Effects	Contraindications
NSAIDs			
Ketorolac	30–60 mg IV or IM loading dose, then 15 mg IV or IM every 6 hr; oral continuation dose: 10 mg orally every 4–6 hr (maximum, 40 mg/day), not to exceed 5 days†	Common: dyspepsia, nausea, abdominal pain, diarrhea, headache, dizziness, elevated aminotransferase levels, drowsiness, tinnitus, pain at injection site Rare but serious: anaphylaxis, gastrointestinal bleeding, acute renal failure, bronchospasm, interstitial nephritis, Stevens–Johnson syndrome, agranulocytosis	Absolute: hypersensitivity, active peptic ulcer disease, cerebrovascular hemorrhage, breast-feeding, 3rd trimester of pregnancy Relative: advanced age, hypertension, congestive heart failure, nasal polyps, volume depletion
Diclofenac	50 mg orally 2 or 3 times/day	Thrombocytopenia; others similar to those of ketorolac	Similar to those for ketorolac
Cyclooxygenase-2 inhibitors			
Rofecoxib	50 mg/day	Common: diarrhea, hypertension, nausea, epigastric discomfort, peripheral edema, dyspepsia, fatigue, dizziness Rare but serious: gastrointestinal bleeding, esophagitis, hypersensitivity, bronchospasm, hypertension, congestive heart failure, potentially increased risk of myocardial infarction, hepatotoxicity, blood dyscrasias, renal failure	Absolute: hypersensitivity, NSAID-induced asthma, hepatic failure, renal failure, 3rd trimester of pregnancy, peptic ulcer disease, gastrointestinal bleeding Relative: renal failure, liver failure, hypertension, ischemic coronary artery disease, dehydration, congestive heart failure, fluid retention, advanced age

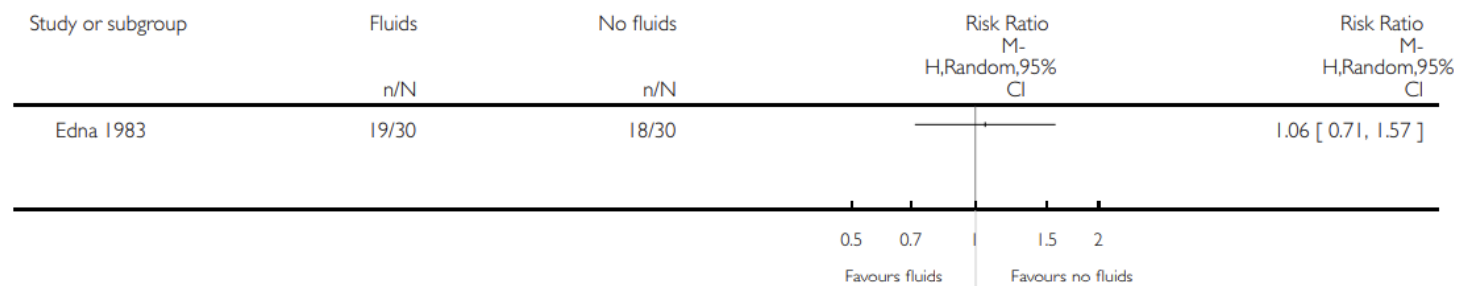
Class and Name of Drug	Adult Dose	Adverse Effects	Contraindications
Meperidine	1 mg/kg of body weight IM every 3–4 hr	Common: dizziness, lightheadedness, sedation, nausea, vomiting, dysphoria, dry mouth, urinary retention, hypotension, agitation, disorientation, constipation, flushing Rare but serious: respiratory depression, respiratory arrest, seizure, cardiac arrest, arrhythmia, shock	Absolute: hypersensitivity, use of monoamine oxidase inhibitors within 14 days Relative: advanced age, respiratory depression, seizure disorder, liver failure, renal failure, hypothyroidism
Morphine sulfate	0.1 mg/kg IM or IV every 4 hr	Biliary spasm, paralytic ileus, toxic megacolon, increased intracranial pressure, miosis, bradycardia; others similar to those of meperidine	Absolute: hypersensitivity, paralytic ileus Relative: chronic obstructive pulmonary disease, biliary disease, acute alcoholism
Narcotic combinations			
Acetaminophen with codeine	300 mg of acetaminophen with 30 mg of codeine, 2 tablets orally every 4–6 hr	Common: lightheadedness, sedation, dizziness, constipation, nausea, vomiting, hypotension, rash, biliary spasm, urinary retention, miosis Rare but serious: pancytopenia, thrombocytopenia, liver damage, respiratory depression, hemolytic anemia, neutropenia	Absolute: hypersensitivity Relative: glucose-6-phosphate dehydrogenase deficiency
Antidiuretics			
Desmopressin	40 µg/spray (if single dose ineffective after 30 min, consider NSAIDs or narcotics)	Common: headache, rhinitis, nausea, dizziness, epistaxis Rare but serious: hyponatremia, water intoxication, seizure, anaphylaxis, thrombosis	Absolute: type IIB von Willebrand's disease, hypersensitivity Relative: coronary artery disease, hypertension, hyponatremia, young age, advanced age, risk of thrombosis

# Severity of renal colic pain and appropriate treatment

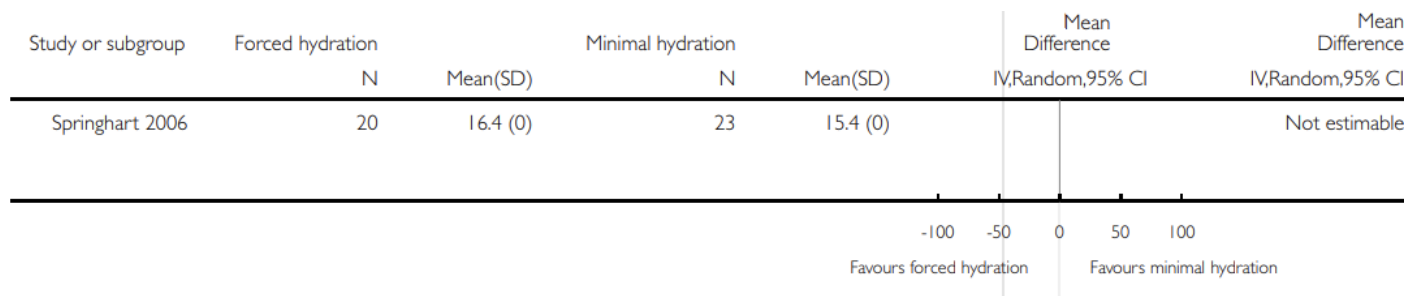
Level of pain	Moderate	Severe	Unbearable
Drug dose	Diclofenac 100 mg oral/rectal <i>or</i> ibuprofen 600–800 mg oral/rectal	Diclofenac 50–100 mg IV (30 min) <i>or</i> dipyron 1–2 g IV (30 min) <i>or</i> 5 g dipyron IV and 500 mg tramadol per 24 h <i>or</i> Desmopressin 40 µg intranasal	Diclofenac 50–100 mg IV (30 min) <i>or</i> dipyron 1–2 g IV (30 min) <i>or</i> 2–3 mg per dose morphine titrated to effect <i>or</i> pethidine 25 mg per dose IV <i>or</i> 5 g dipyron IV and 500 mg tramadol per 24 h <i>or</i> Desmopressin 40 µg intranasal

# Hydration

Fluids versus no fluids, Outcome 1 Pain > 6 hours.



Forced (2 L IV over 4 hours) vs minimal (20 mL per hour) hydration, Outcome 2 Analgaesic use (morphine equivalents).



No reliable evidence in the literature to support the use of diuretics and high volume fluid therapy for people with acute ureteric colic.

Potential positive therapeutic impact of fluids and diuretics to facilitate stone passage



# Immediate intervention

Indications)

- The presence of an obstructed infected kidney
- Obstruction of a solitary kidney
- Bilateral obstruction
- intractable pain or vomiting

→ Immediate D-J insertion or PCN

Catheter-related, Stent-related symptoms should be considered

# Medical Expulsive Therapy

- Calcium channel
    - Ureteral contraction is induced by increase of cytoplasmic free calcium and the function of calcium channels
    - CCBs inhibit the spontaneous rhythmic activity of the ureter and calyces
    - Prostaglandins and calcium influx to the cells induce contractions
    - CCBs such as verapamil and nifedipine have led to the use of these substances for MET
  - Adrenergic receptors
    - $\alpha 1A$ ,  $\alpha 1B$ , and  $\alpha 1D$  / distal ureter has the higher distribution of  $\alpha 1$  adrenergic receptors
  - Phosphodiesterase (PDE) types 1, 2, and 4
    - Intracellular messenger molecule (cAMP, cGMP) regulates cellular response to extracellular stimulation
- Possible Agents for MET  
:  **$\alpha$ -blockers**, anticholinergics, PDE5 inhibitors, **calcium channel blockers**, beta-3 agonist, and corticosteroids

# Summary

- NSAIDs (ketorolac, diclofenac), which act directly by inhibiting prostaglandin-induced effects, and opioids (pethidine, meperidine, morphine, and tramadol) are commonly used as analgesic agents to relieve renal colic
  - First choice should be NSAIDs if not contraindicated.
  - Opioid should be avoided for possible side effect and addiction. Nonetheless, opioid is medical therapy of choice during pregnancy
- Ureteral stenting, PCN insertion should be considered for intractable pain or vomiting, infectious condition or impaired renal function caused by obstruction