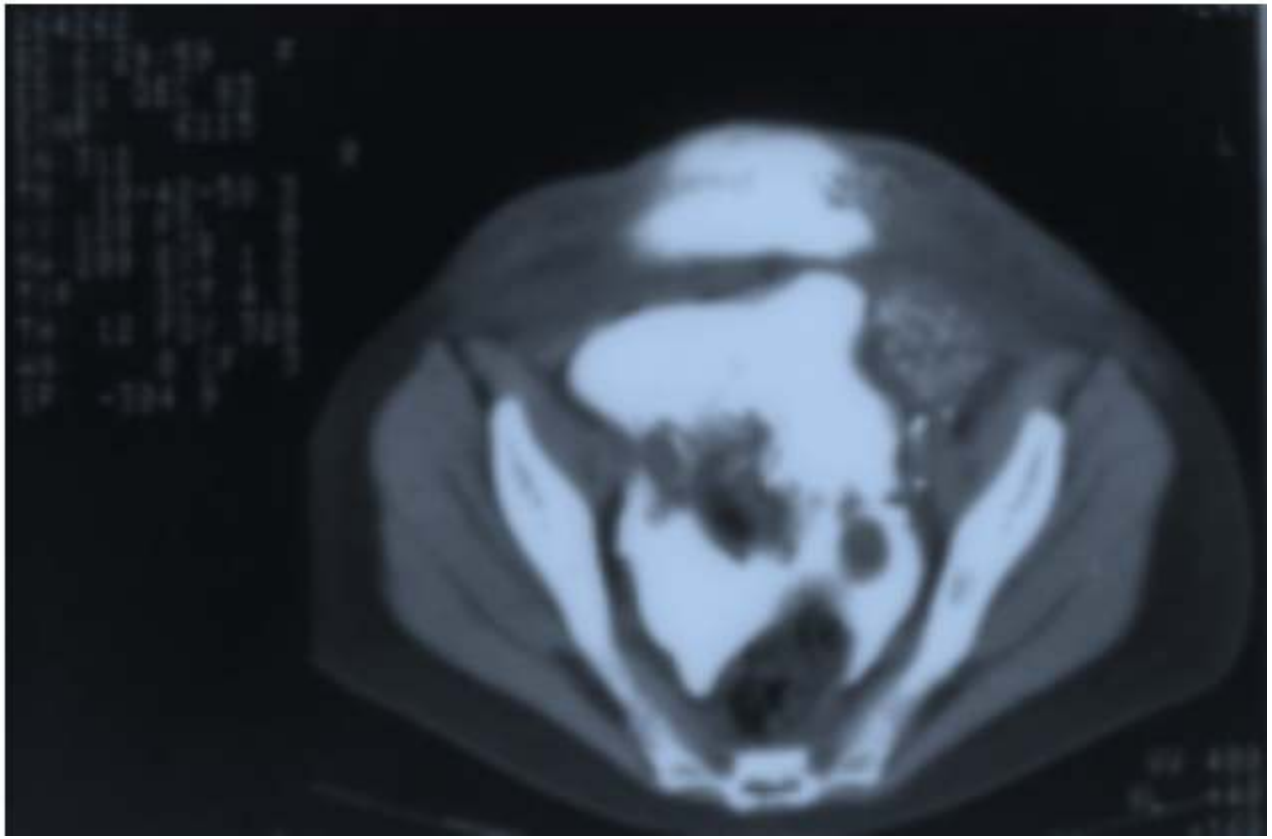
# Delayed wound Healing causing leakage

50-year-old lady , diseased donor renal txp.
Allograft dysfunction from Txp glomerulopathy
Modified immunosuppression using sirolimus,
Eventual graft loss
A coiled swan-neck PD catheter placed.
Prolonged peri-catheter leak from poor wound healing (sirolimus).

J Pearl et al: Clin Transplant 2008;22:672-3

# Delayed wound Healing causing leakage



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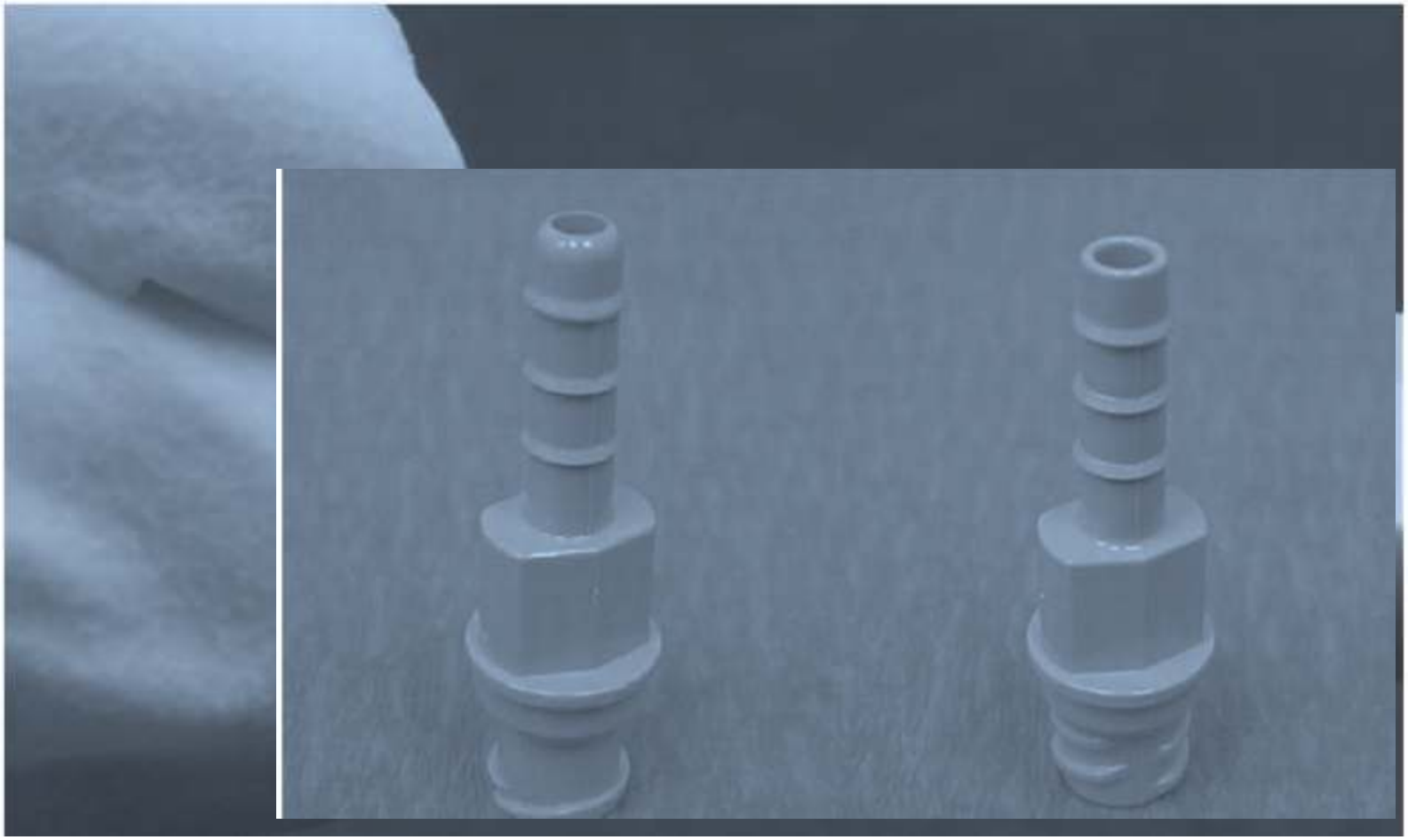
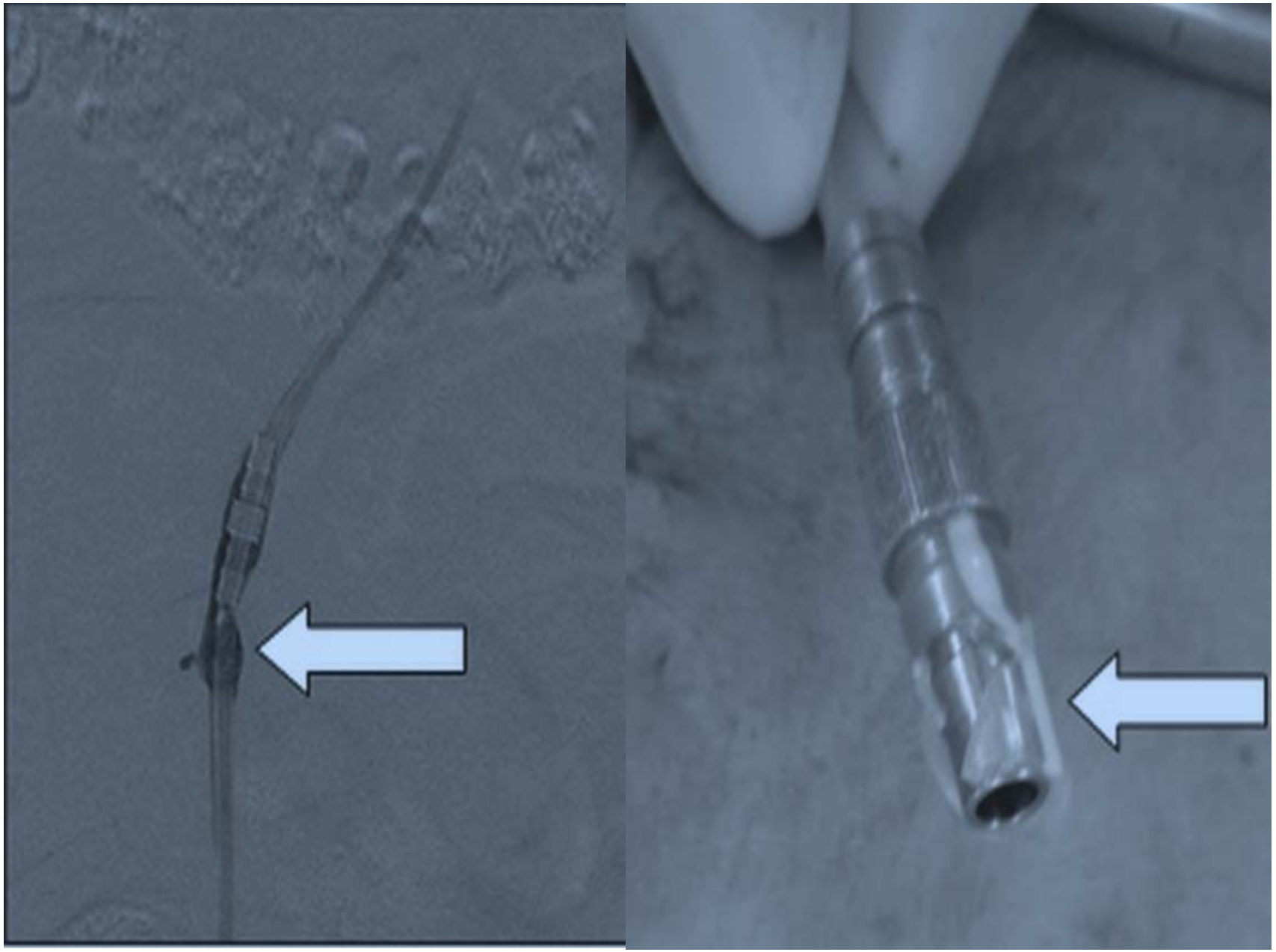


Ohashi Y et al: Advances in Perit Dial 2012;28



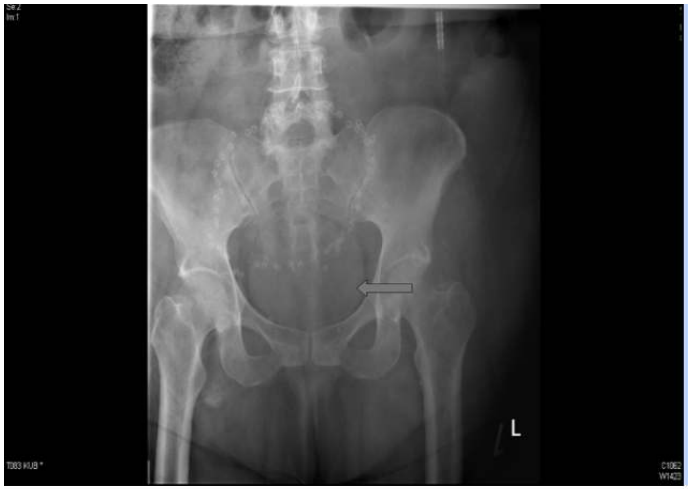


FIGURE 1 Anterior–posterior radiograph of abdomen, showing catheter tip in the pelvis (arrow).

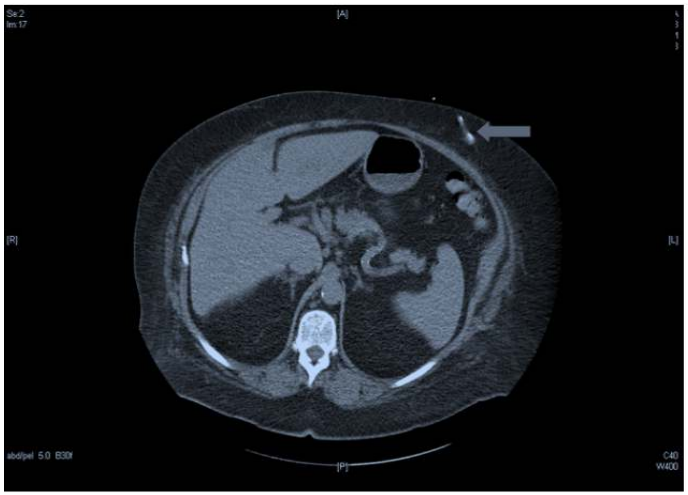


FIGURE 2 Computed tomography image of abdomen, showing catheter location (arrow).

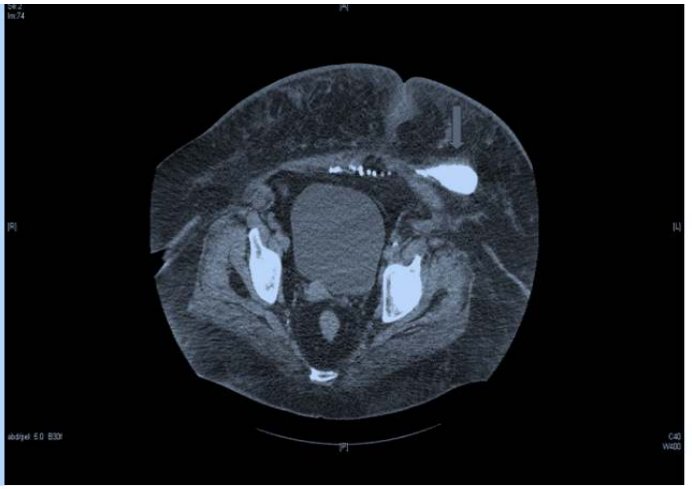


FIGURE 3 Computed tomography image of abdomen, showing dye in the subcutaneous tissue (arrow).

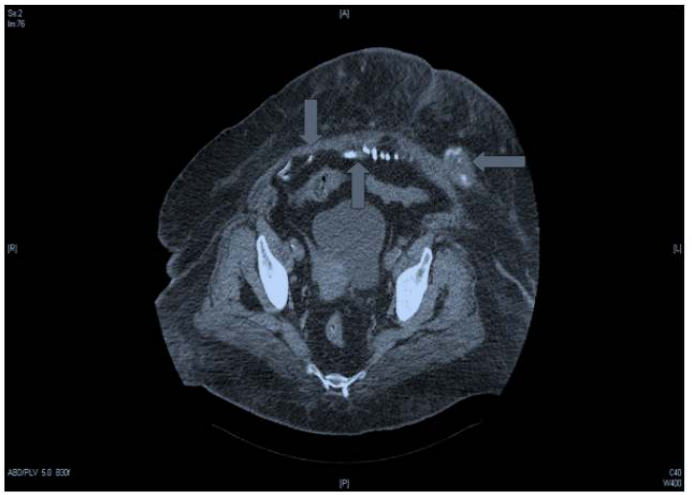
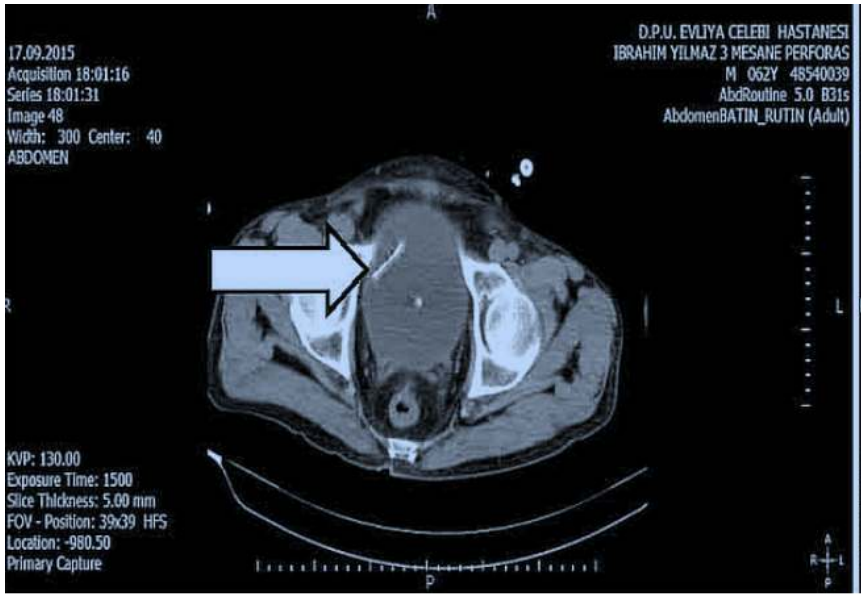


FIGURE 4 New intraperitoneal peritoneal dialysis catheter (left arrow); abdominal hernia mesh (middle arrow); and broken subcutaneous segment of the old catheter (right arrow).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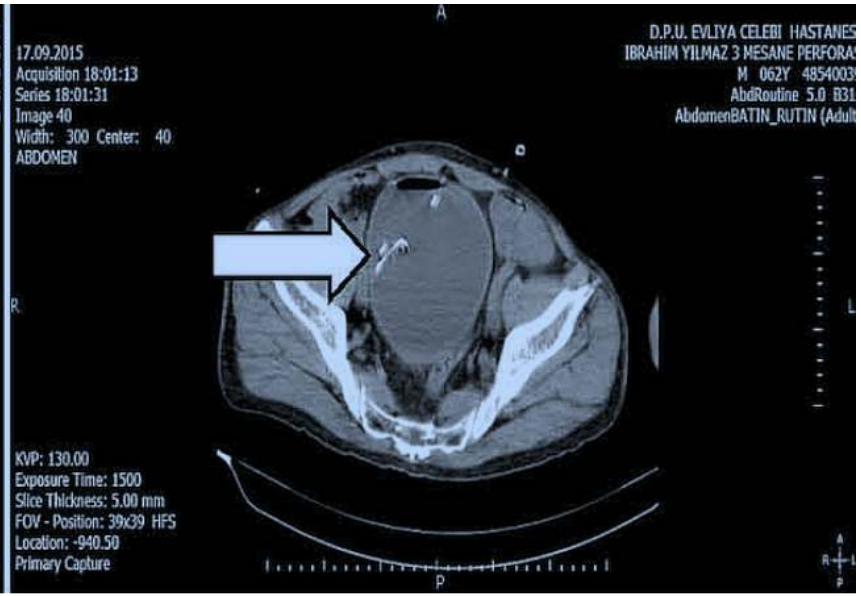
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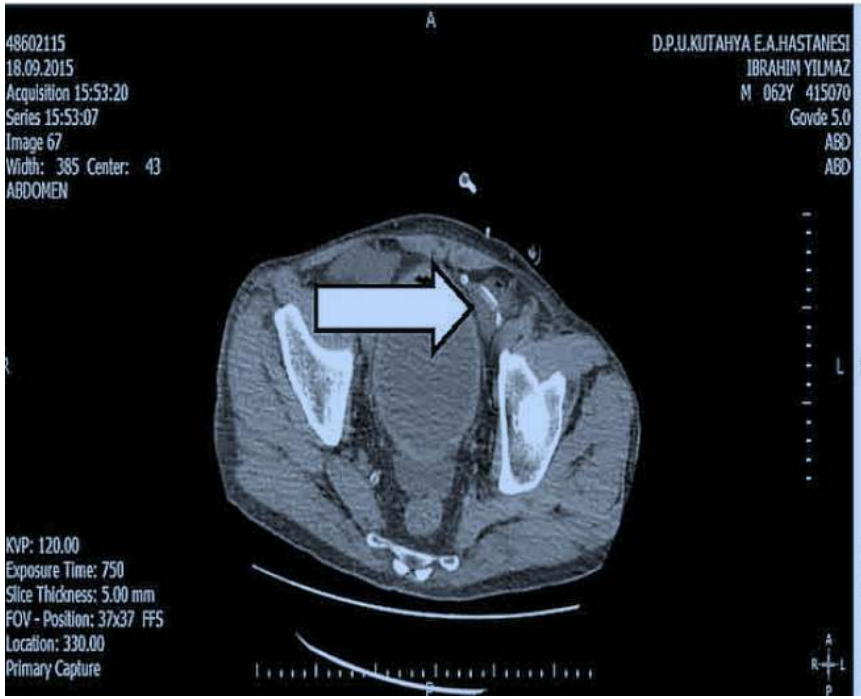
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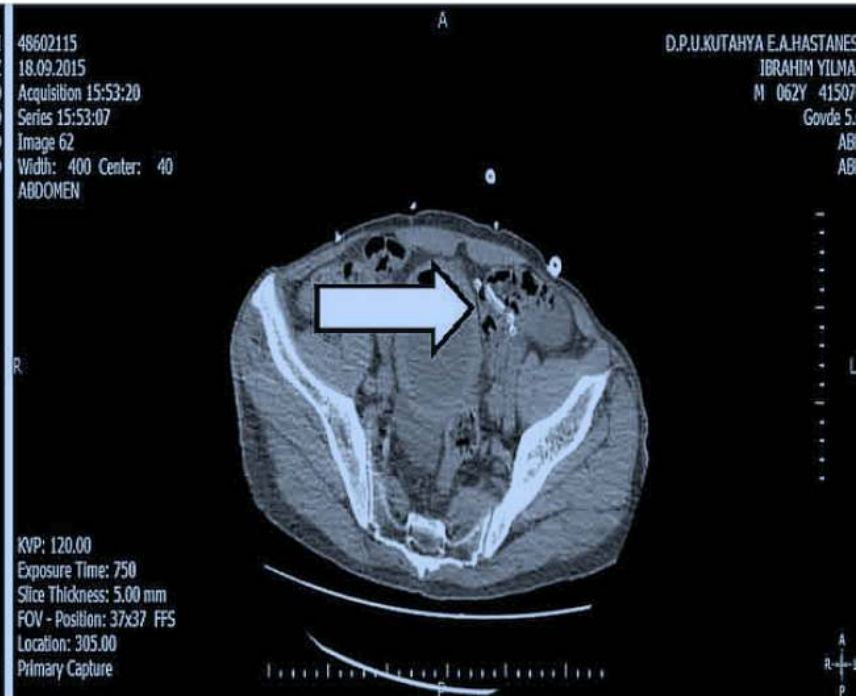
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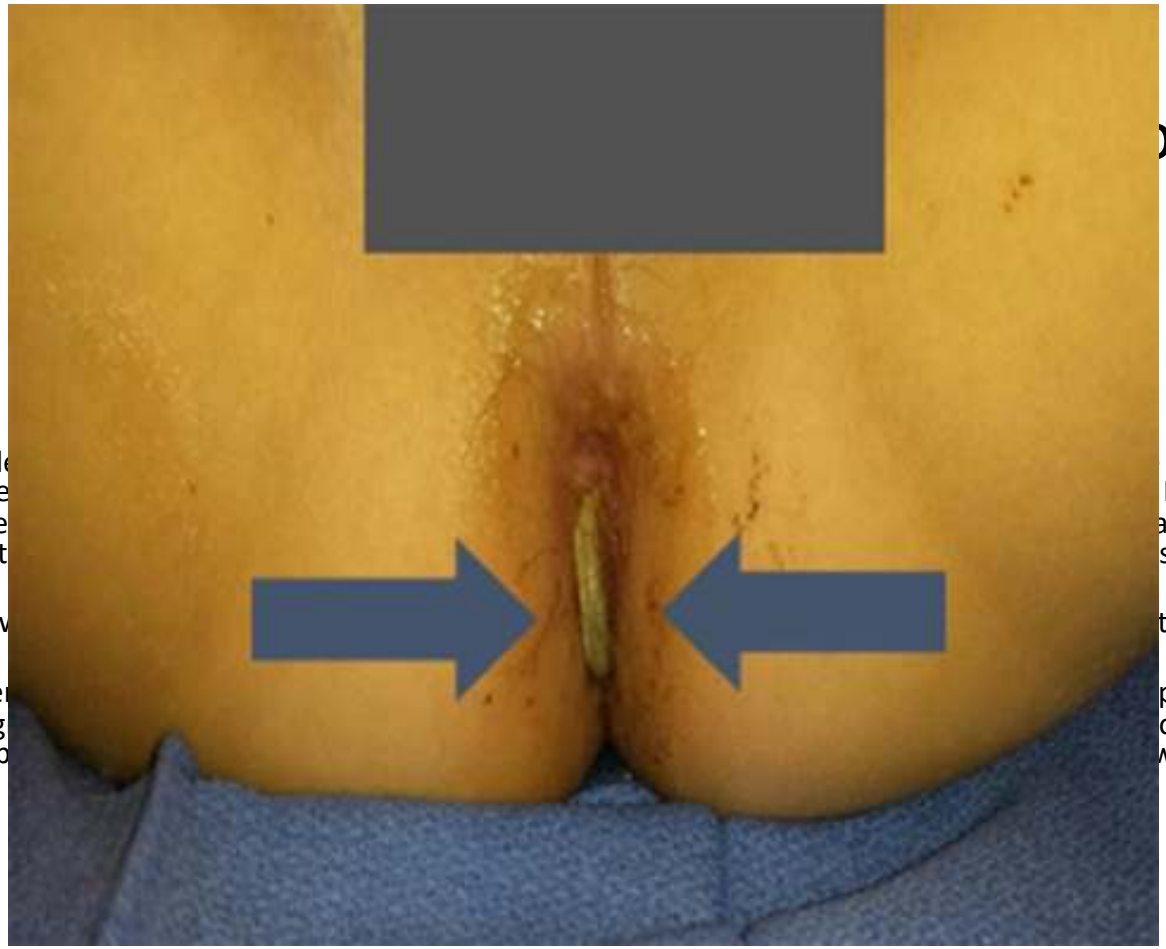
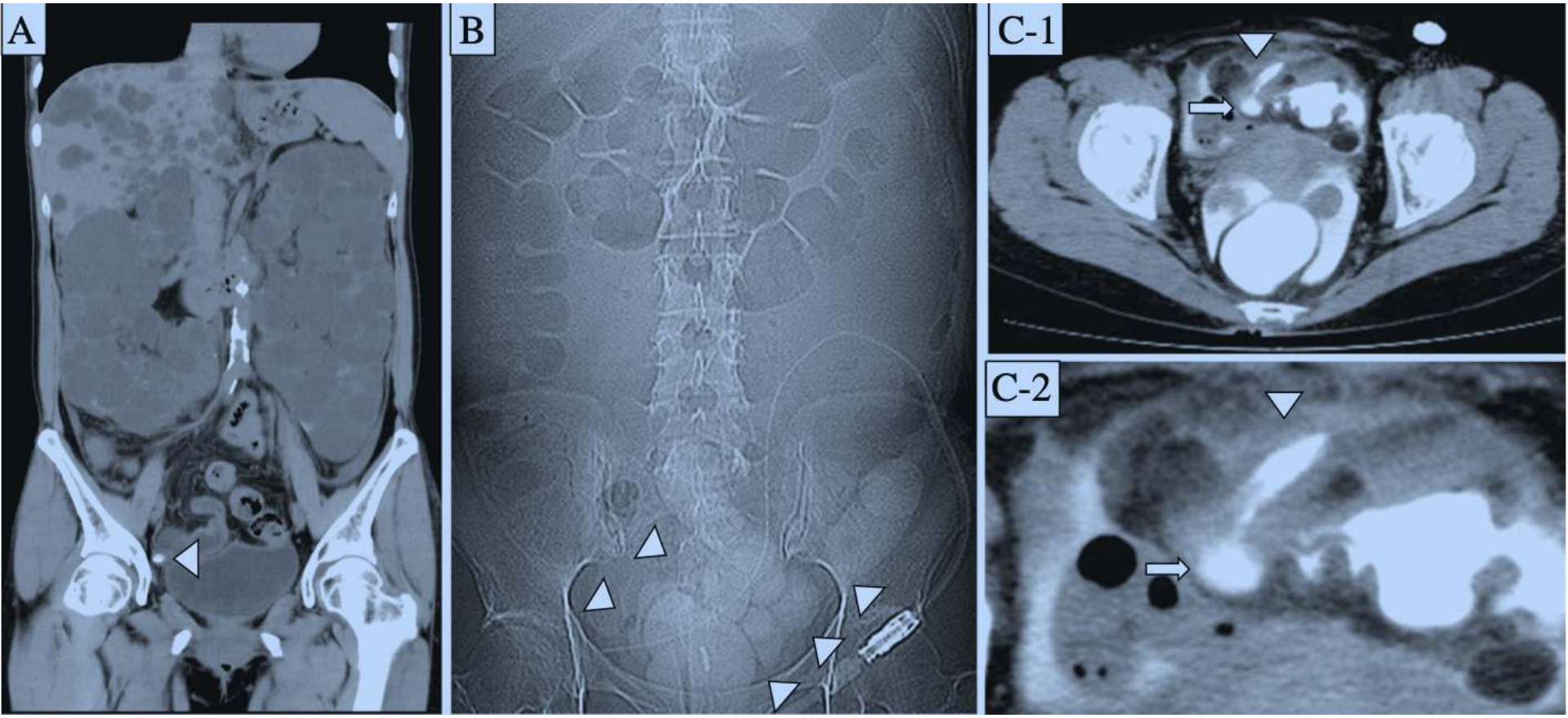
# Sigmo lapsing



The catheter was in the colon 15 cm from anus, indicating that the catheter had entered the colon within the peritoneal cavity. A long tract surrounded by omentum traversing from the left mid abdomen to the right lower abdomen was found. The catheter was found entering the sigmoid, which was repaired after removing the catheter. The patient recovered well and remained stable on HD.

A Sabahat et al: Am J Case Rep 2020;21:e922828

# Bowel perforation.




T Handa et al: Renal Replacement Therapy 2019;5:24

# Bowel Perforation

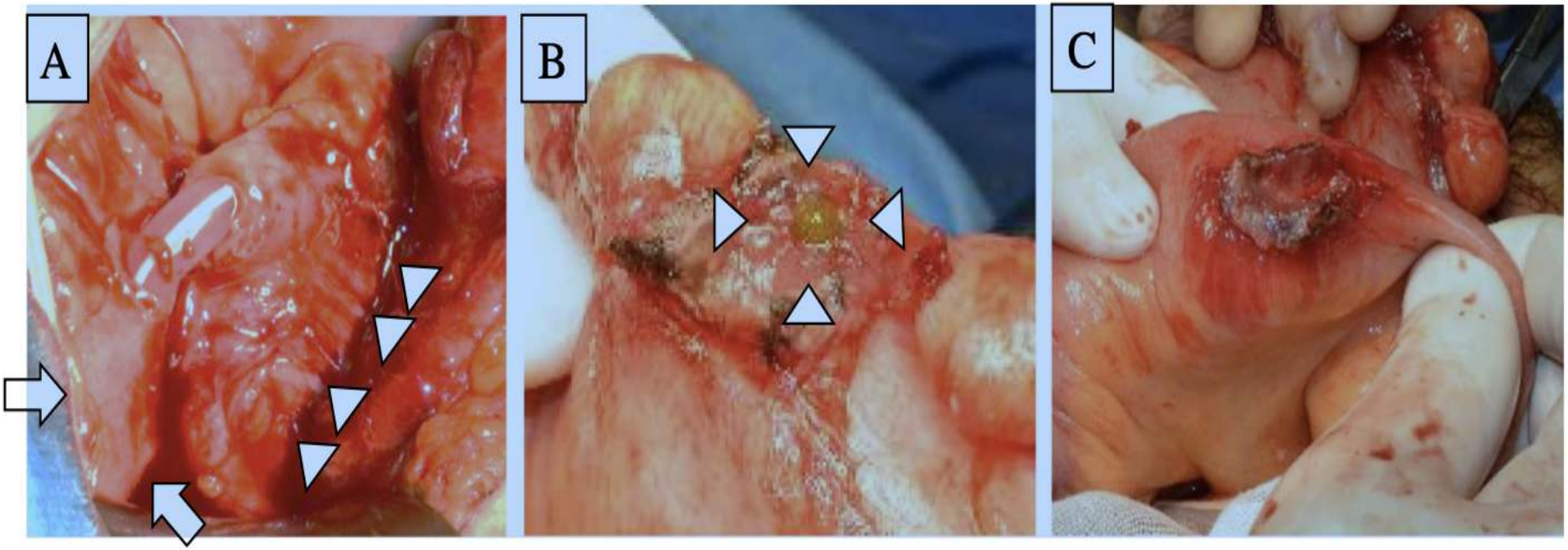



**Fig. 2** Exploratory laparotomy reveals a small orifice in the eroding surface of the sigmoid colon formed where the side hole of the catheter is attached to the colon. **a** The PD catheter was wrapped in the omentum (arrow), and there was erosion on the sigmoid colon (arrowhead). The lateral side of the PD catheter was adhered to the sigmoid colon 5 cm from the catheter tip. **b** A small hole with oozing feces was found where the side hole of the catheter was attached to the colon. The lesion was entirely surrounded by fibrous tissues, which prevented leakage of the intraluminal contents. **c** The PD catheter was also attached to the serosa of the small intestine

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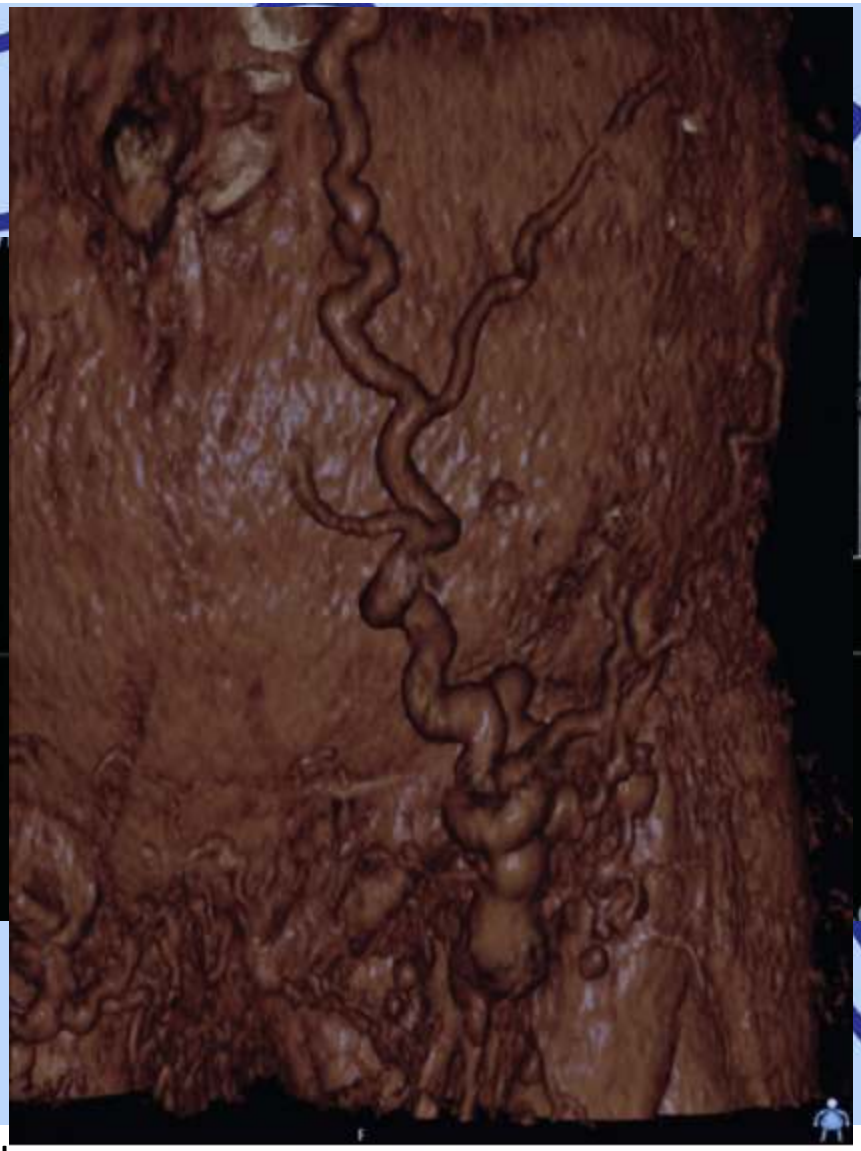
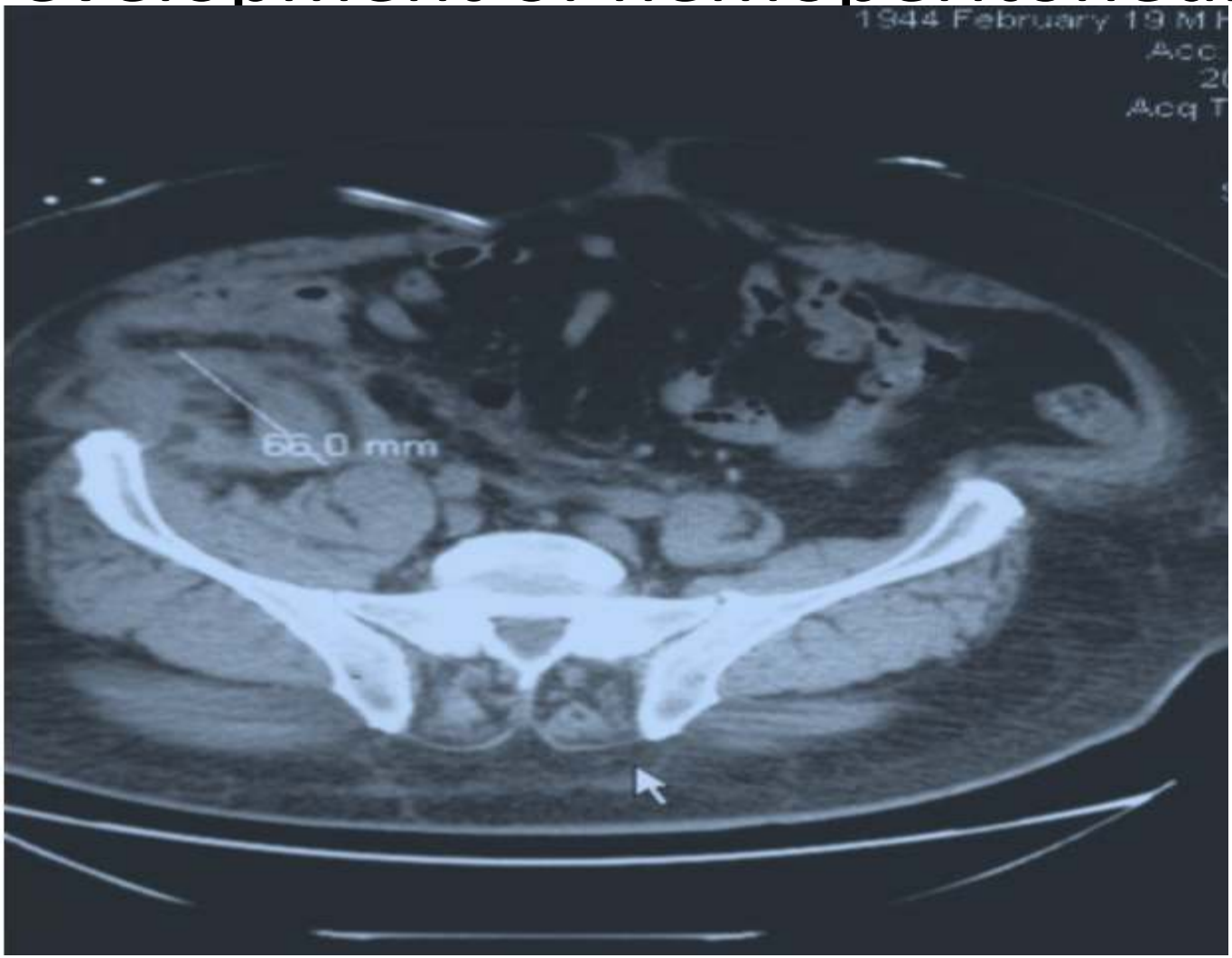
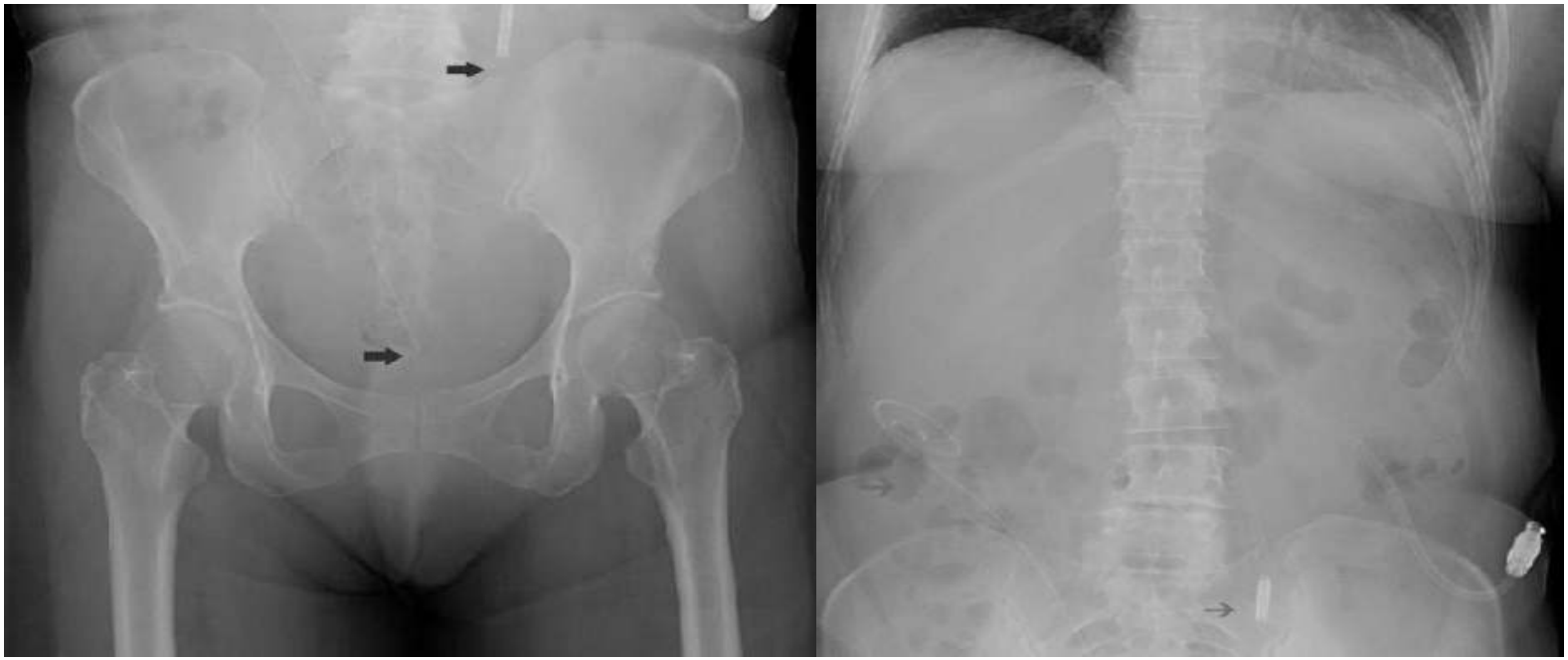


T Natroshvili et al: Case Reports in Surgery 2020; article ID 1309418

# Development of hemoperitoneum.



Nonfunctioning PD catheter from separation from the catheter into 2 fragments within abdominal cavity causing outflow failure.



PK Guru et al: Saudi J of Kidney Dis and Transplant 2017;38:405-9

# Problems with pre-sternal catheter, 3 cases .

**Case # 1**: Isolated dialysate collection from fracture of the catheter

A 41-year-old paraplegic male who had a pre-sternally placed PD catheter in order to avoid infectious complications.

After a year on PD, he developed a painless swelling near the left supra-umbilical region with impaired drainage of dialysate when seated in an upright position. Swelling was close to the site of a previous catheter-related incision. There was a painless reducible 5 x 5 cm swelling.

This was thought to be a hernia and it was repaired when a pocket of dialysate solution was noted adjacent to a fracture in the catheter just below the titanium connector. The catheter did not require replacement. Instead, it was repaired and the patient experienced no problems

BS Pavey et al: Advances in Perit Dial 2008;24

# Pre e #2:

**Recur adapter.**

62-year luid growing
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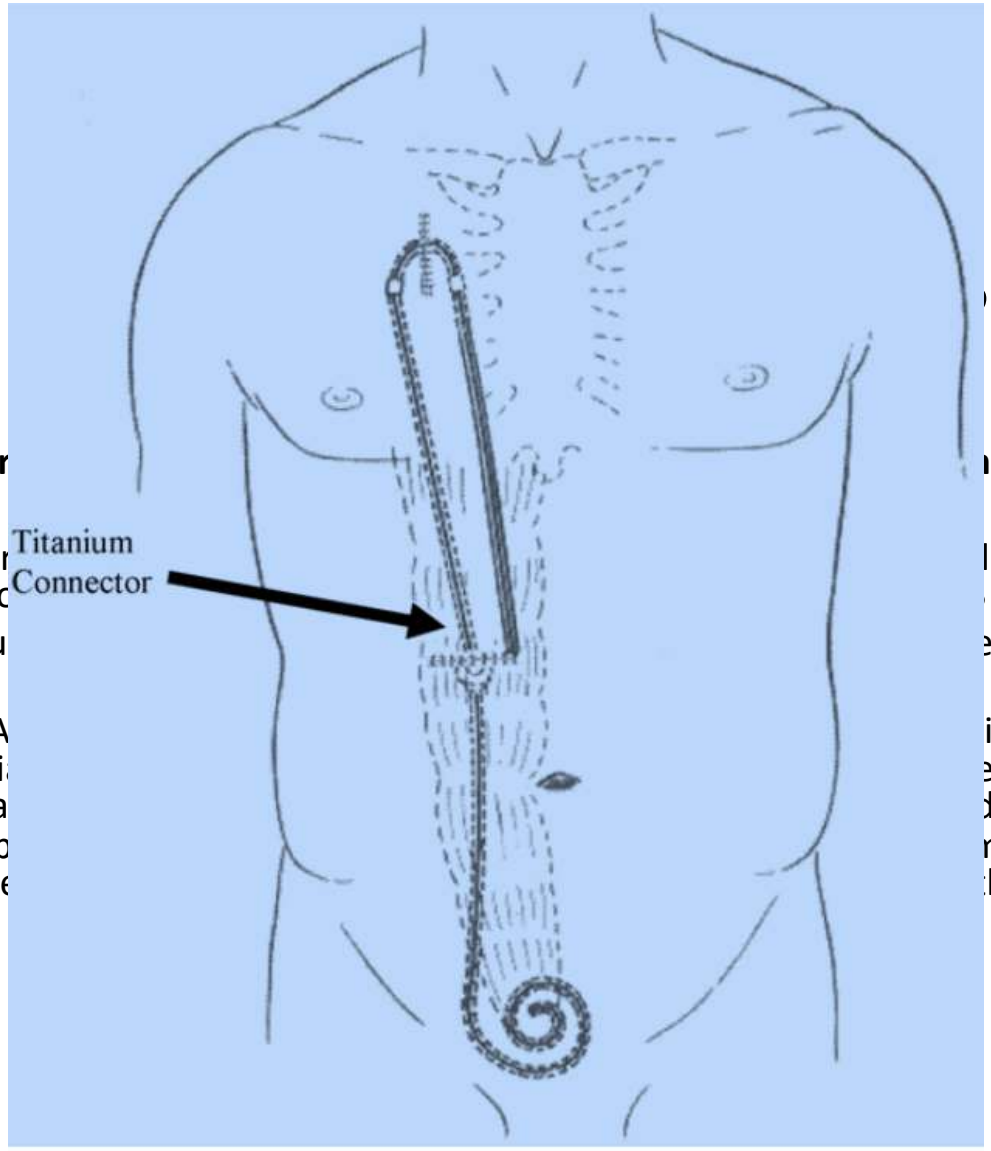

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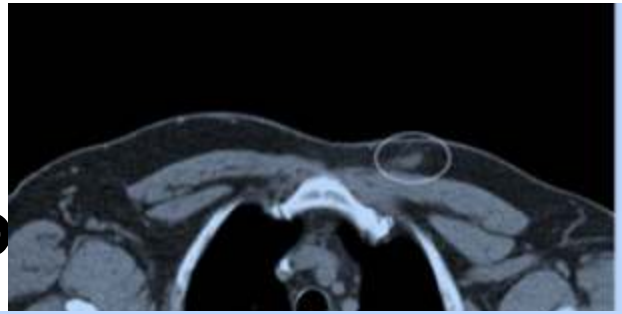

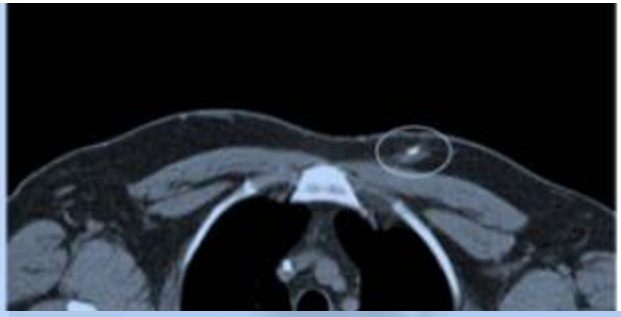

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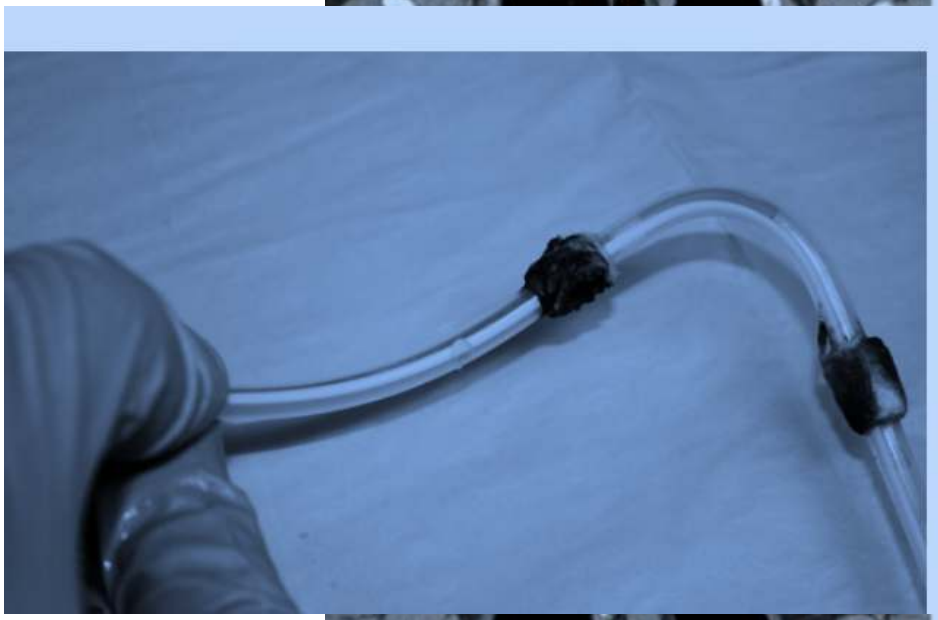
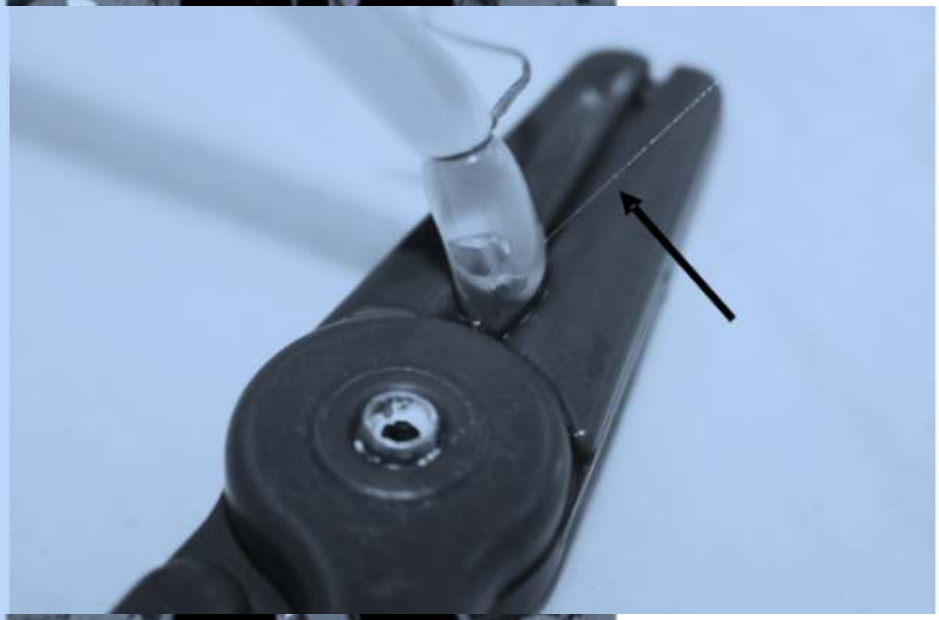
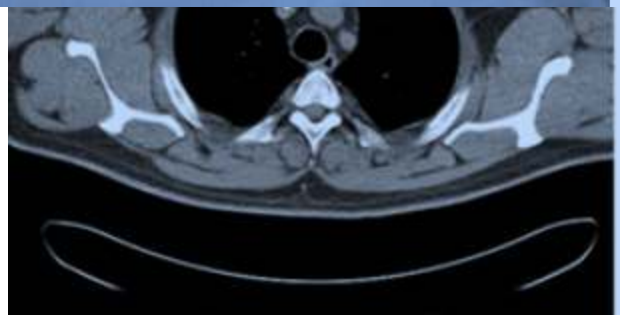
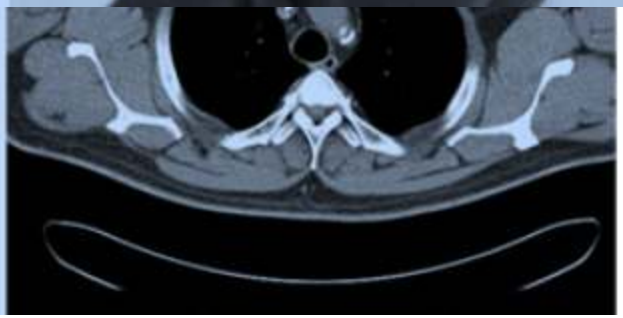








# In summary:

Persistent leaks & delayed wound healing

Erosions & Fractures

Holes & Catheter Rupture

Visceral perforation (bladder, colon)

Disconnection, outflow failure, w. & w/out infection

Entering a vein

Retroperitoneal Hematoma &/or hemoperitoneum

Annual Dialysis Conference

presented by the *University of Missouri Division of Nephrology*

# Managing Electrolyte Imbalances in PD

**Kurt Tarwater, MD**
**Associate Professor**
**Division of Nephrology**
**University of Missouri**

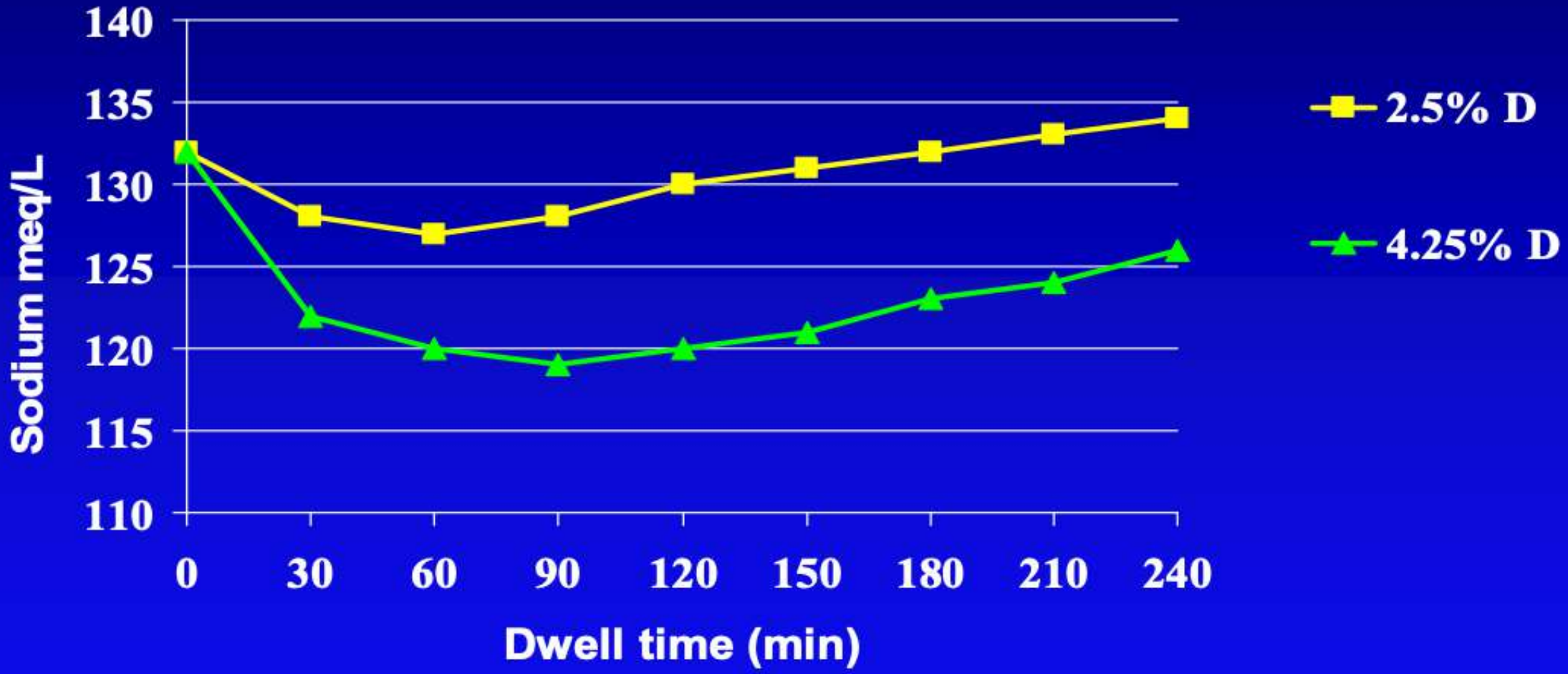
University of Missouri

# Overview

- Case based review of hyponatremia and hypokalemia in PD patients

# Hyponatremia in PD

- Develops due to excess water intake relative to Na intake
  - PD effluent is hypotonic to isotonic to plasma




Heimburger et al. Kid Int 38: 495, 1990

# Hyponatremia in PD

Case

- 56 y/o anuric male on CAPD
- Stable PD regimen for 2 years (4 x 2.5 L exchanges daily)
  - Stable serum sodium of 135 mEq/L
  - Stable weight of 60 kg
- Gradually over 20 days
  - Plasma Na drops to 127 mEq/L
  - Weight increases to 70 kg with associated pitting edema
- Plasma Na stabilized at 127 mEq/L but weight/edema continued to increase

# Hyponatremia in PD

- In the first 2 years
  - Daily dialysate Na losses repeatedly were measured at 140.5 mEq/day
  - UF was always around 1L daily
- Currently
  - UF remains around 1L daily
  - Dietary Na intake unchanged
  - Daily fluid intake has increased by 0.5 L/day the past several weeks

# Hyponatremia in PD

- Increased water intake caused the hyponatremia.

- What is the mechanism for his volume overload?

# Hyponatremia in PD

- In first 2 years
  - Pt was in steady state
    - Daily Sodium Intake = Daily Sodium Removal with PD
    - Daily Water Intake = Daily UF Volume + Insensible Losses
    - Plasma Na stable at 135 mEq/L
  - Daily Na losses of 140.5 mEq/day so Na intake was also 140.5 mEq/day

# Hyponatremia in PD

- New steady state (Na 127 mEq/L)
  - Intake unchanged at 140.5 mEq/day
  - Pt continuing to gain weight by 0.5 kg/day
  - UF unchanged at 1L/day

# Hyponatremia in PD

- New steady state (Na 127 mEq/L)
  - Na removed daily = 11L x 127 mEq/L = 1397 mEq (assuming drained PD fluid measures at 127 mEq/L)
  - Na infused daily = 10L x 132 mEq/L = 1320 mEq
  - Daily sodium balance = 1320 infused – 1397 drained + 140.5 dietary = **63.5 mEq/day positive balance**

# Hyponatremia in PD

- New steady state (Na 127 mEq/L)
  - Pt is drinking an extra 0.5 L/day
  - Positive daily Na balance of 63.5 mEq/day
  - 63.5 mEq / 0.5 L = **127 mEq/L added to ECF daily**
- **With no changes pt will continue to have isonatric volume expansion of 0.5 kg daily**
- **Treatment**
  - **Reducing fluid intake by 0.5 L/day will return [Na] to previous baseline**
  - **To remove excess volume will have to increase UF**

# Key Points

- In CAPD, increasing water intake above the steady state level leads to hyponatremia and volume expansion
  - The plasma sodium level will drop but eventually reach a new steady state level
  - ECF volume will not achieve a new steady state. It will increase daily by the amount the daily water intake was increased

# Key Points

- In steady state the Na removed in drain fluid = dietary intake
  - When excess water intake reduces plasma [Na] the daily Na lost in drain fluid decreases and a daily positive Na balance develops assuming dietary intake remains constant
  - The daily Na excess / daily water excess will be hypotonic to plasma and will expand the ECF
  - A new steady state will occur when the plasma [Na] drops to the point that it = daily Na excess / daily water excess

# Key Points

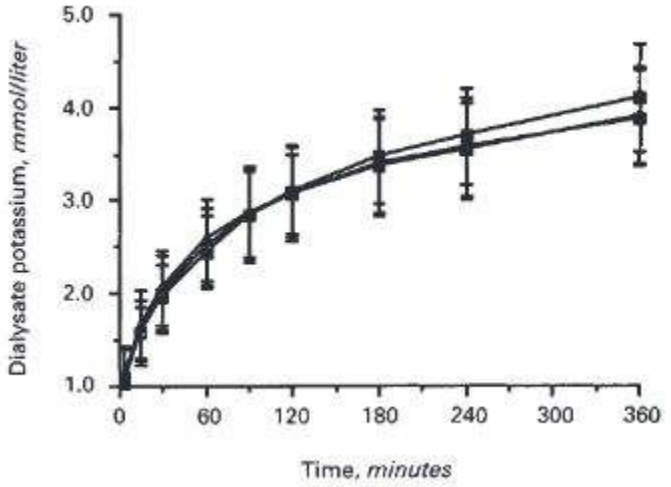
- **<u>In Our Case</u>**
  - **Serum sodium was 127 mEq/L**
  - **Daily fluid gain was 0.5 L**
  - **Daily positive Na balance was 63.5 mEq**
  - **63.5 mEq / 0.5 L = 127 mEq/L**

- **Continuing the same PD Rx and Na/water intake will yield daily volume expansion by 0.5 L at a stable plasma [Na] of 127 mEq/L**

# Hypokalemia in PD

General Considerations

- Serum K is 0.5 mEq/L higher than plasma
  - Therefore PD effluent K is 0.5 mEq/L lower than measured serum K
  - This will be ignored during the case review
- PD fluid has no K
- PD fluid K will equal plasma K in about 6 hours of dwell time




*Heimbürger et al: Solute and fluid transport in CAPD*

# Hypokalemia in PD

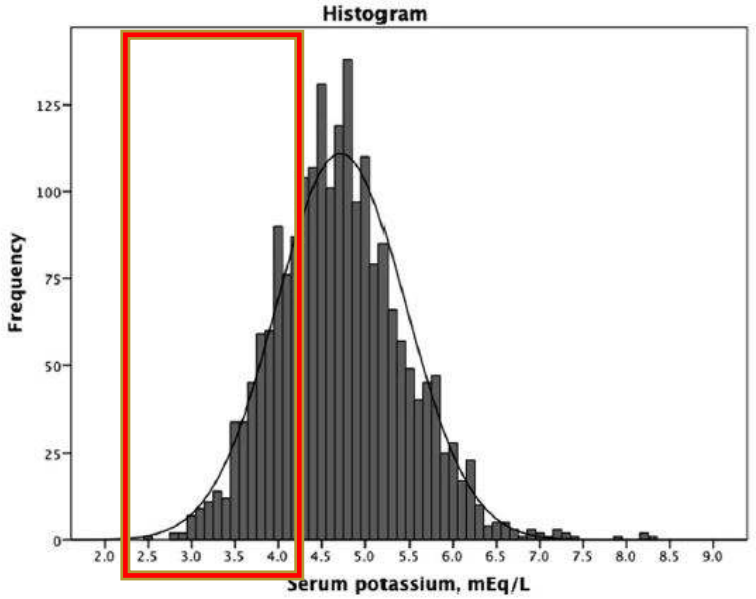



Fig. 1 Histogram of serum potassium distribution. Frequency distribution histogram for potassium levels in 146 patients (2055 measurements), expressed in mEq/L

International Urology and Nephrology (2020) 52:393–398

# Hypokalemia in PD

Case

- 43 y/o anuric women
- On CAPD for 1 year with regimen of 4 x 2.5L exchanges daily
- Serum K has been 4 mEq/L on several occasions
- Daily diet estimated to be 60 mEq/day
- UF volume of 1.5L/day and her weight have both been steady

# Hypokalemia in PD

- The patient decides to eliminate many K-containing foods from her diet and K intake drops to 30 mEq/day

- What impact will her reduced dietary K have on her serum K level?

# Hypokalemia in PD

- Prior to the dietary K reduction this patients daily K intake had to equal daily K losses because the serum K was steady at a level of 4 mEq/L

- In Steady State
  - Daily K Intake = Daily K Losses
  - Daily K Intake = K in PD drain fluid + Stool K
  - Daily K Intake = [(Serum [K]) x (drain fluid V)] + Stool K

# Hypokalemia in PD

Calculate Serum K After Diet Change

- First – Calculate K in drain fluid prior to diet change
  - Rx: 4 x 2.5L exchanges daily --- 4 mEq/L x 10L = 40 mEq
  - UF: 1.5L/day --- 4 mEq/L x 1.5L = 6 mEq
  - Total: 46 mEq/day in drain fluid

- Next – Calculate daily stool K losses prior to diet change
  - 60 mEq/day intake – 46 mEq in drain fluid = 14 mEq/day in stool

# Hypokalemia in PD

- Lastly – Solve for new steady state serum K level
  - Daily K Intake = [(Serum [K]) x (drain fluid V)] + Stool K

Equation can be rearranged to:

  - [Daily K Intake – Stool K] / [PD Drain Vol] = Serum K concentration

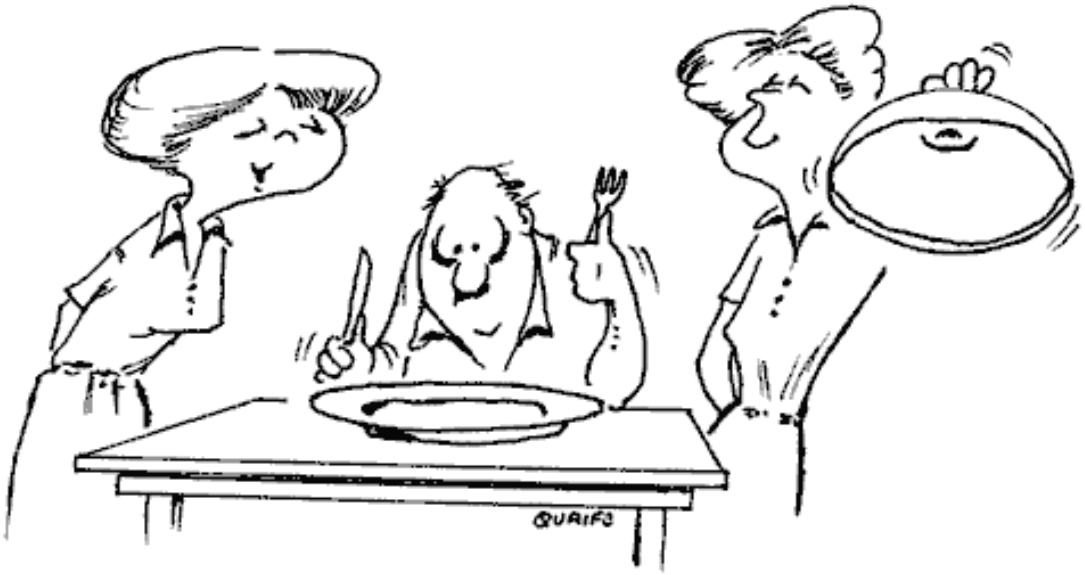
  - [30 mEq – 14 mEq] / [11.5 L] = **1.4 mEq/L (new steady state K level)**

# Hypokalemia in PD

- A more common clinical situation would be that the patients steady state plasma K level changes and they either didn't tell you or didn't know they decreased their K intake.
- In this situation you can use the previous equations to solve for the daily K intake

# Key Points

- PD is very effective at K removal
- K losses from diffusion and convection can be easily estimated from serum K concentration and drain volume
- It is helpful to know PD patients daily K intake at steady state so that if serum K changes to a new steady state the new dietary K intake can be calculated



...and this dish is totally potassium-free!

www.lightersideofdialysis.com

# Gastroparesis in Peritoneal Dialysis (PD)

**Dr Mayuri Trivedi**

Assistant Professor and In-charge
Nephrology Division, Department of Medicine
L.T.M.G Hospital, Mumbai

Consultant Nephrology and Transplant physician
Hinduja Healthcare Surgical, Mumbai

@mayuritrivedi80

- **Session title**: PD Fundamentals-Complications of PD
- **Lecture** : Gastroparesis in PD
- **Disclosures**: No conflict of Interest
- **Objective:** To discuss the pathophysiology, diagnosis, and treatment of gastroparesis in PD and the literature review

# Outline of the talk

- Relevance of the topic
- Statistics
- Definition and complications of gastroparesis
- Physiology of normal gastric emptying
- Pathophysiology of gastric emptying in PD
- Diagnosis
- Treatment

# Why is this topic relevant?

# Relevance of discussion

- Despite the very high prevalence, gastrointestinal (GI) disorders in Peritoneal dialysis (PD) patients are vastly under reported and under studied

- Impact on QOL, morbidity, and drop out from PD are high

# Statistics

- 57.9% of PD patients have varying degrees of GI symptoms

  Li Y et al Perit Dial Int 2009; 29

- 85% patients on PD report at least one GI complaint as compared to 51% on HD

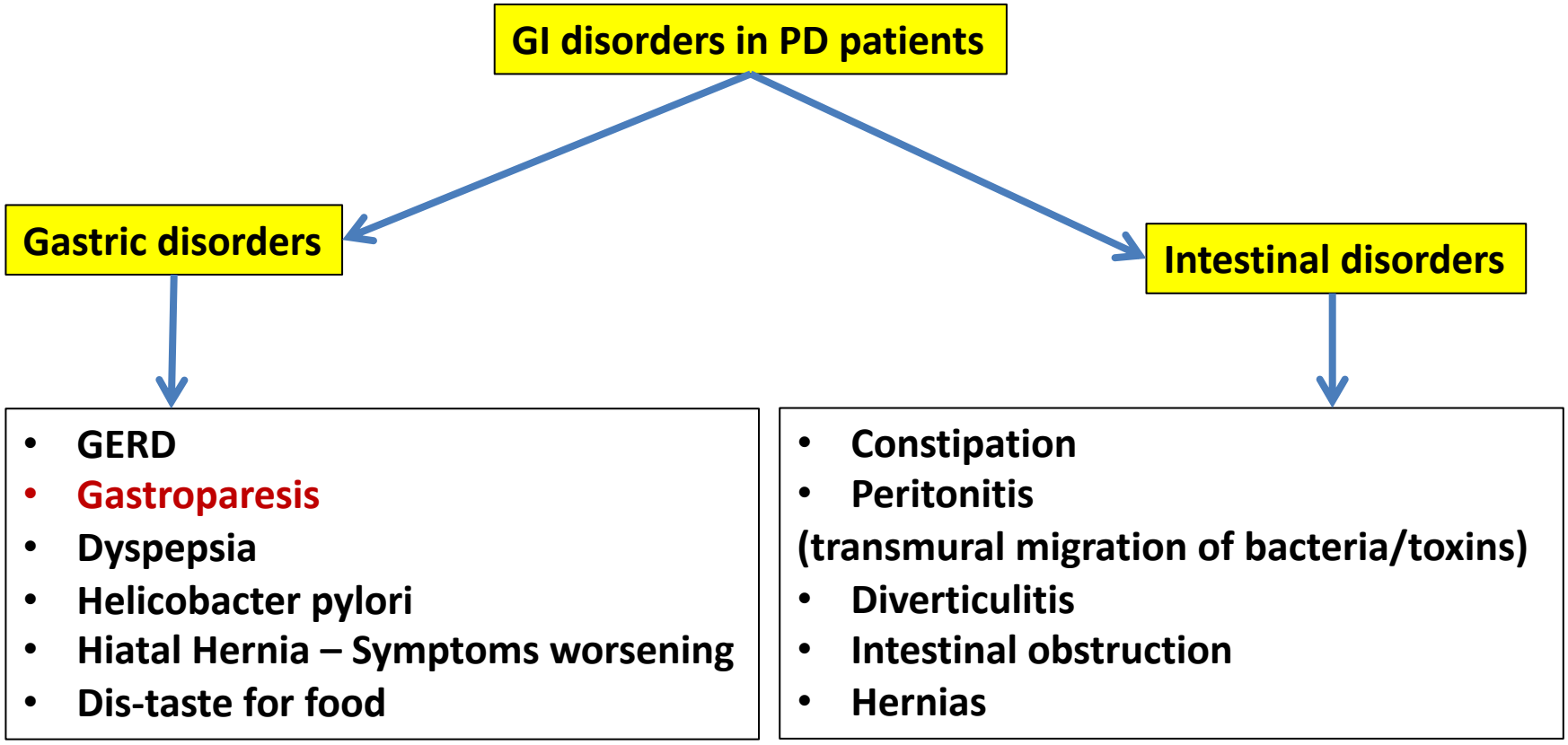
- At least 50% of patients on PD have gastroparesis which may or may not be symptomatic

- 55% patients on PD correlate their GI symptoms to “Onset of PD” as compared to 12% patients on HD

- 53% patients on PD report change in dietary habits to relieve GI symptoms as compared to 14% patients on HD

  Salamon K et al. J Ren Nutr 2013; 23: 114–118.

# Classification of GI disorders in PD



# Definition and complication of Gastroparesis

# What is gastroparesis?

**Gastroparesis is delay in gastric emptying without obstruction to outflow**

**Cardinal symptoms:**

Nausea, vomiting, early satiety, bloating and upper abdominal pain

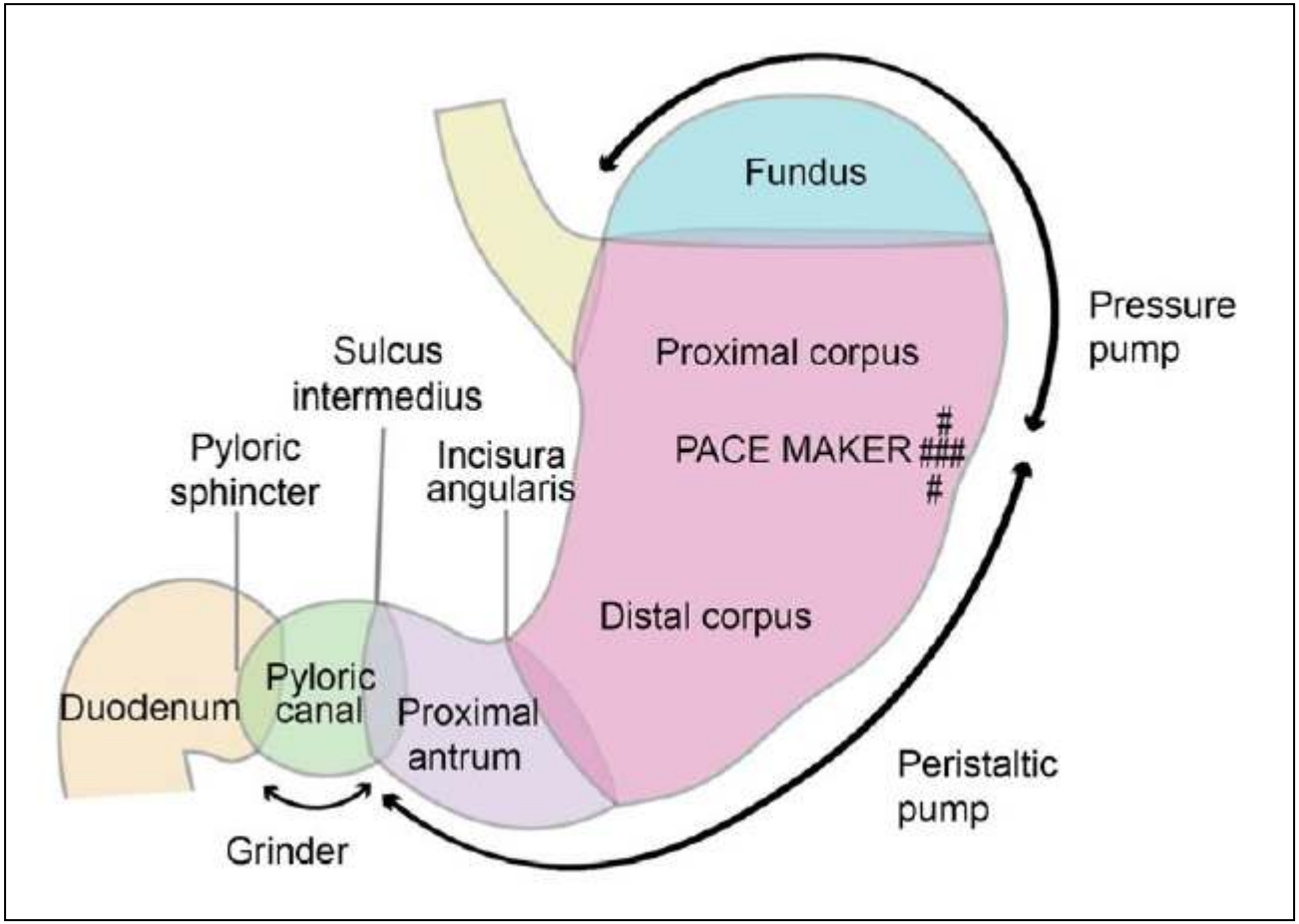
**Complications:**

Electrolyte imbalance, dehydration, frequent hospitalisations malnutrition and impaired QOL

**Severe gastroparesis:**

Oesophagitis, Mallory-Weiss tear, PUD ,Bezoar formation

**What is the physiology/pathophysiology of Gastric emptying ?**




The anatomical and functional parts of the stomach

Goyal RK et al Neurogastroenterol Motil. 2019 Apr;31(4):e13546.

# Physiology of gastric emptying

- Gastric motility is controlled by the gastric myo-electrical activity which propagates from the proximal body to distal antrum

- Gastric myo-electrical activity is controlled by

**Neuronal circuits:**

1. Gastric inhibitory vagal motor circuit (GIVMC)
2. Gastric excitatory vagal motor circuit (GEVMC)

- Measured non invasively using abdominal surface electrodes called electro-gastrography (EGG)

# Physiology of gastric emptying

| Slow gastric emptying | Fast gastric emptying |
|---|---|
| Cholecystokinin | Ghrelin |
| Leptin | Motilin |
| Glucagon-like peptide-1 | |
| Glucagon | |
| Oxyntomodulin | |
| Peptide YY | |
| Gastrin-releasing peptide | |
| Enterostatin | |
| Pancreatic amylin | |
| Pancreatic polypeptide | |

Goyal RK et al Neurogastroenterol Motil. 2019 Apr;31(4):e13546

# Physiology of gastric emptying

**Relevant definitions:**

**Dominant Power (DP):**Mean power in the total gastric power spectrum during the EGG recording

Normally the DP increases with oral intake

**Normal slow wave frequency (NSWF) :** 2-4cycles/min

**Tachygastria:** Frequency of 4-9 cycles/minute

**Bradygastria**: Frequency of less than 3 cycles/min

Lee SW et al Am J Kidney Dis 2000; 36:566–573

# Pathophysiology of gastric emptying in PD (Myo-electrical)

- Impaired irrespective of dialysate composition or when tested with empty peritoneal cavity

- Most impaired with glucose or metabolically active substrate containing dialysate

Van V et al.Perit Dial Int. 2002 Jan-Feb;22(1):32-8

- Abnormal myo-electrical activity on EGG

- Direct uremic toxins or autonomic dysfunction or electrolyte imbalance

# Abnormalities in myoelectrical activity

- CAPD causes enhanced gastric dysrhythmias
- No increase in DP and decrease in DP following infusion of PD fluid
- Increase in tachygastria post CAPD as compared to pre CAPD associated with impaired gastric motility
- Increased degree of early satiety post CAPD as compared to post HD

Lee SW et al Am J Kidney Dis 2000; 36:566–573

# Abnormalities of gastric emptying in PD (Hormonal)

1. Increased levels of hormones in ESKD:
   - Insulin
   - Gastrin
   - Glucagon
   - Cholecystokinin
2. These changes in hormone levels are improved after HD but not with CAPD

Impaired gastric emptying-Gastroparesis

Sirinek KR et al Am J Surg 148:732-735, 1984
Bonomini V et al Blood Purif 8:54-68, 1990

# How do you diagnose gastroparesis?

# Symptoms and signs

- Nausea
- Vomiting
- Bloating
- Early satiety
- Abdominal pain (Burning , vague or crampy epigastric pain)
- Rarely abdominal tenderness on palpation

# Scoring systems used for gauging severity

- Gastrointestinal symptom rating scale (GSRS)
- GI symptoms questionnaire (GSQ)
- Gastrointestinal symptom score (GIS)
- Rome criteria (Version IV)
- **Gastroparesis Cardinal symptom index (GCSI)**

- Assessment of symptom severity over 2 weeks
- Incorporates 9 symptoms focusing on 3 areas
  early satiety, nausea/vomiting, and bloating
- High co-relation with actual gastric emptying

# Diagnostic tests and Imaging

- Esophagogastroduodenoscopy
- Gastric emptying scintigraphy (GES)
- Wireless capsule motility
- Antral-duodenal manometry
- Breath test
- Other imaging: MRI, 3-D USG

# Gastric emptying scintigraphy (GES)

- Currently considered the gold standard
- Non invasive ,quantitative radio labelled scintigraphy based test
- Limited use due to lack of standardisation and interference due to drugs , smoking, hyperglycaemia etc.
- Delayed gastric emptying defined as Gastric retention:

1. >90% at 1 hour
2. >60% at 2 hours
3. >10%at 4 hours

# Wireless capsule motility

- Indigestible capsule provides a nonradioactive and comparable alternative to GES
- Measures gastric-emptying time by sensing luminal pH, pressure, and temperature after swallowing
- Gastric emptying is demarcated when there is a sudden change in pH - acidity of the stomach to the alkaline of the duodenum

# Antro-duodenal manometry

- Coordination of gastrointestinal motor function in fasting and postprandial periods through pressure measurements
- Pressure sensors measure the gastric and duodenal contractions
- Helps differentiating between myopathy and neuropathy causes

# Breath test

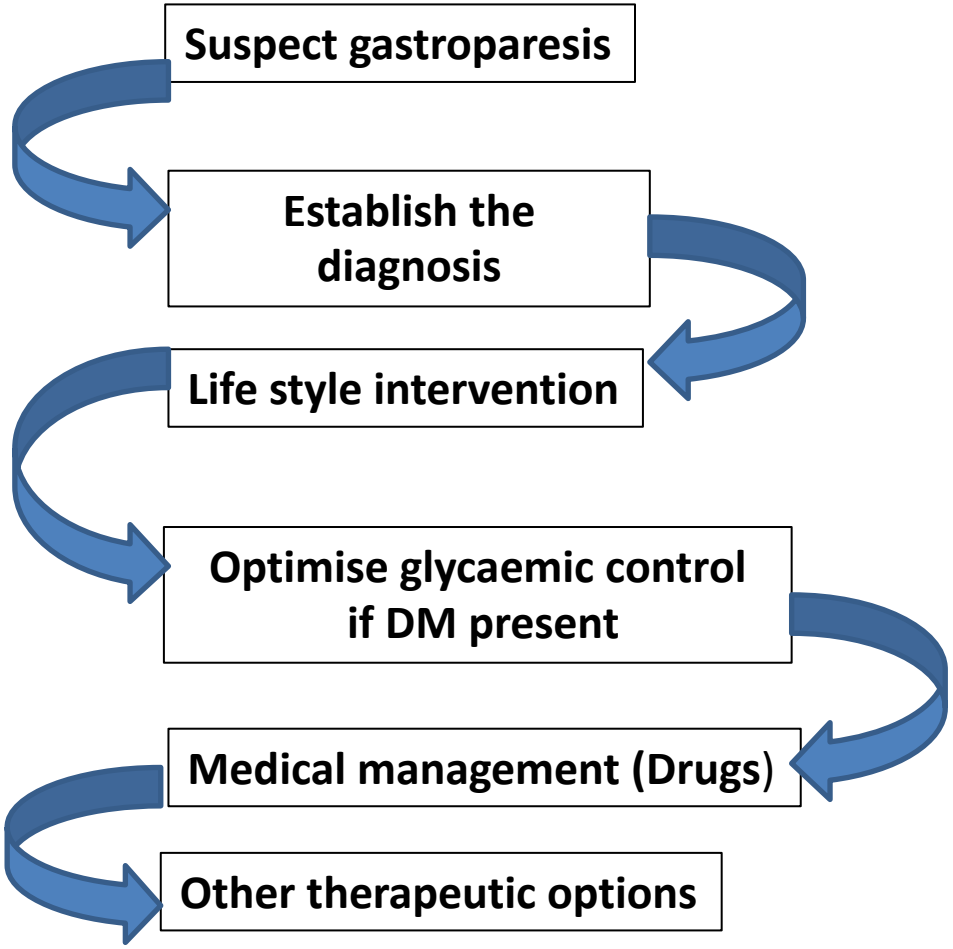
- Involves a stable nonradioactive 13C isotope bound to a digestible substance, such as octanoic acid, acetate, or *Spirulina platensis* (algae)being ingested

- After ingestion- absorbed from the small intestine, it is metabolized into 13C-CO2,which is expelled from the lungs and collected

- Rate-limiting step of this reaction is the rate of solid gastric emptying

# How do we treat gastroparesis in PD patients ?

# Approach To Treatment



# Life style intervention

- Improve Nutrition ,fluid and electrolyte status
- Multiple small meals (4-6/day)
- Low fat and fibre meals
- Avoid smoking and alcohol
- Tight glycemic control in case of DM
- Encourage high calorie liquid drinks
- Assisted nutrition may be necessary (if oral not adequate)

# Medical management

| Class of drugs | Name | Action | Side effect in ESKD |
|---|---|---|---|
| Dopamine D2 receptor antagonist | Metoclopramide<br>Domperidone | Release of Ach from enteral neurons : increased gastric contractions | Tardive dyskinesia (dose reduction to 50-75% in ESKD)<br>Lesser CNS side effects with Domperidone |
| Motilin Receptor antagonist | Erythromycin | Increased gastric contraction | Risk of tachyphylaxis with erythromycin |
| 5HT4 receptor agonists | Cisapride | Increases contraction via cholinergic pathway | Risk of arrhythmias<br>**Withdrawn from the market after FDA warning due to increased deaths due to arrhythmias** |

**Metoclopramide (10mg/2 litres bag) and erythromycin (100mg/2 litres bag) may be used intraperitoneally**

# Gastric electrical Stimulation

- Involves delivery of impulses at high frequency causing low-energy stimuli through a pacemaker stimulating gastric emptying
- Apparatus consists of a neurostimulator implanted in the skin of lower abdomen and 2 leads
- Watch out for bowel obstruction, peritoneal infection and intractable bleeding
- Contraindicated in patients with associated hernias

Majanović SK et al Perit Dial Int. 2018 Nov-Dec;38(6):458-462.

# Take home points

- Gastrointestinal symptoms including Gastroparesis is an important yet understudied and ignored complication of PD

- It may remain unreported or unnoticed but has a major impact on QOL and may lead to discontinuation of PD

**Intractable cases with Gastroparesis may need to stop PD and switch to HD either temporarily or sometimes even permanently**

- Gastroparesis needs to be specifically looked for in patients on PD

- Life style interventions and drugs may offer some relief albeit for a limited extent and period of time

- Gastric electric stimulation may be an emerging therapy that requires more in depth studies

# Thank you