Extern case conference

Wisarut Chaichan 580710229

Identification data

- ผู้ป่วยชายไทย อายุ 56 ปี
- Hospital number: 1611539
- สิทธิ์การรักษา: บัตรประกันสุขภาพบัตรทอง
- แหล่งที่มาของข้อมูล: ผู้ป่วยและเวชระเบียนทางการแพทย์
- ความน่าเชื่อถือ: สูง
- ER visit date 15/2/64

Chief complaint

ปวดท้อง 2 วัน ก่อนมาโรงพยาบาล

Vital signs
at รพช

BP 127/100

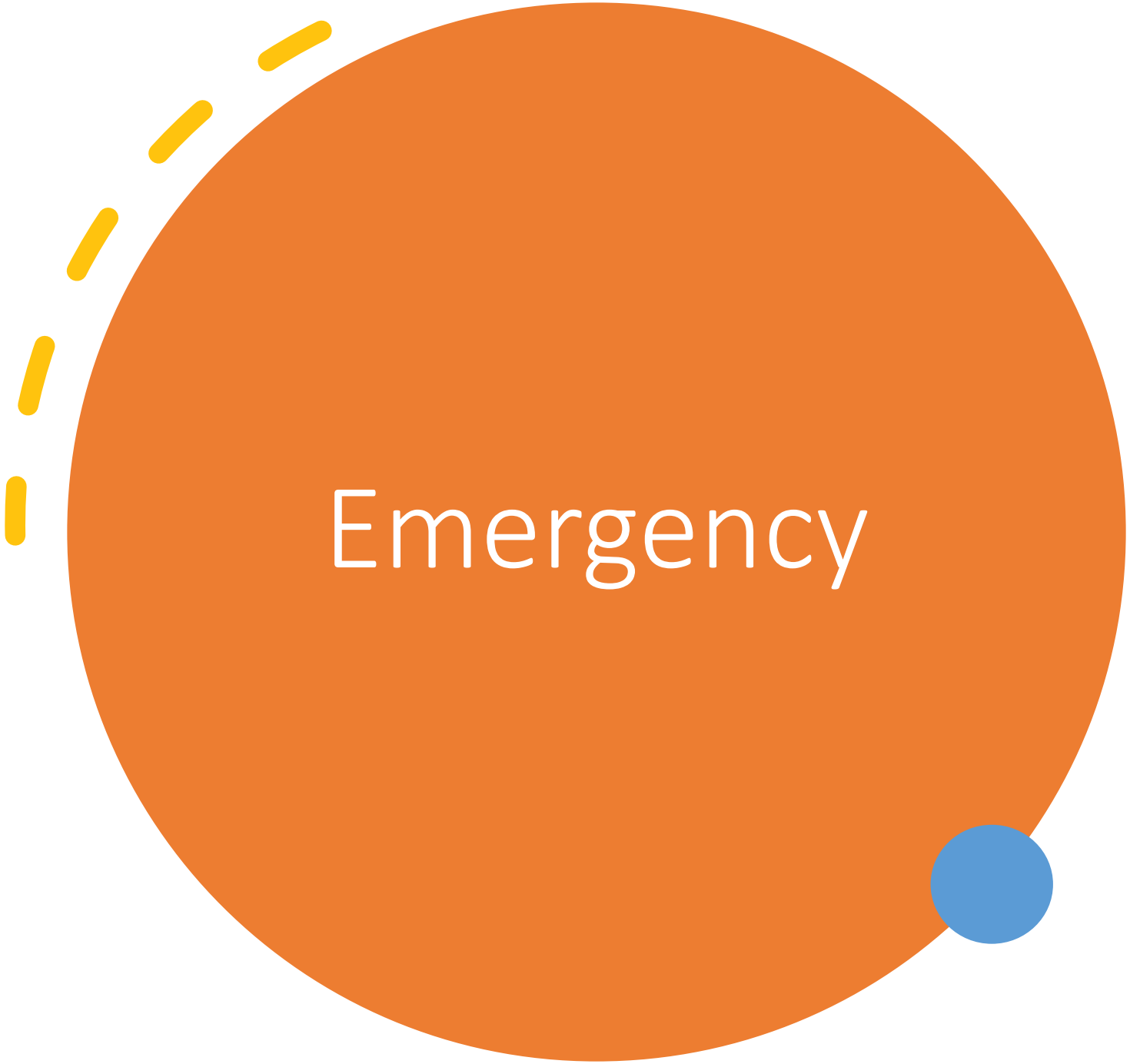
PR 90

RR 30

BT 36

SpO2 100

Triage level


Emergency

Primary survey

Airway

- Can talk, no stridor, no hoarseness

Airway

- Interpretation: no signs of airway obstruction
- Management: not necessary

Breathing

- Tachypnea, rapid shallow breathing pattern
- Symmetrical chest movement, no paradoxical chest movement
- Clear and equal breath sound both lungs

Breathing

- Interpretation: dyspnea
- Management: on O2 mask with bag 10 LPM -> RR 36 SpO2 100 -> on ETT

Circulation

- BP 127/100, PR 90
- Pulses 2+ all, CRT < 2sec

Circulation

- Interpretation: normal
- Management: not necessary

Disability

- E4V5M6, pupils 2 mm RTL BE

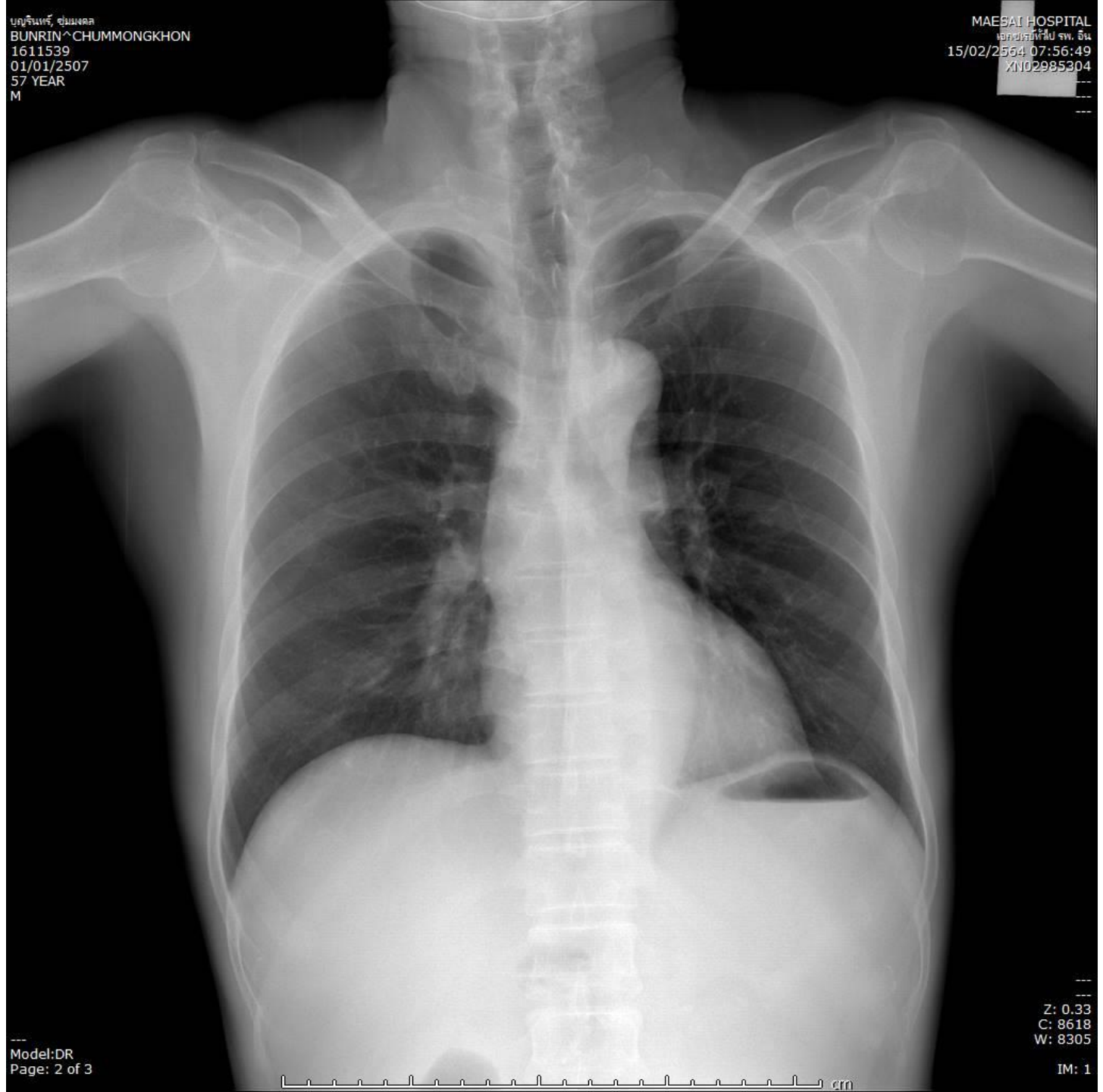
Disability

- Interpretation: normal
- Management: not necessary

Adjunct to primary survey

Adjunct to primary survey

- Monitor vital signs,SpO2
- Acute abdomen series
- ABG
- NG tube
- NSS 1000 ml IV drip 100 ml/hr
- EKG 12 leads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01/01/2507
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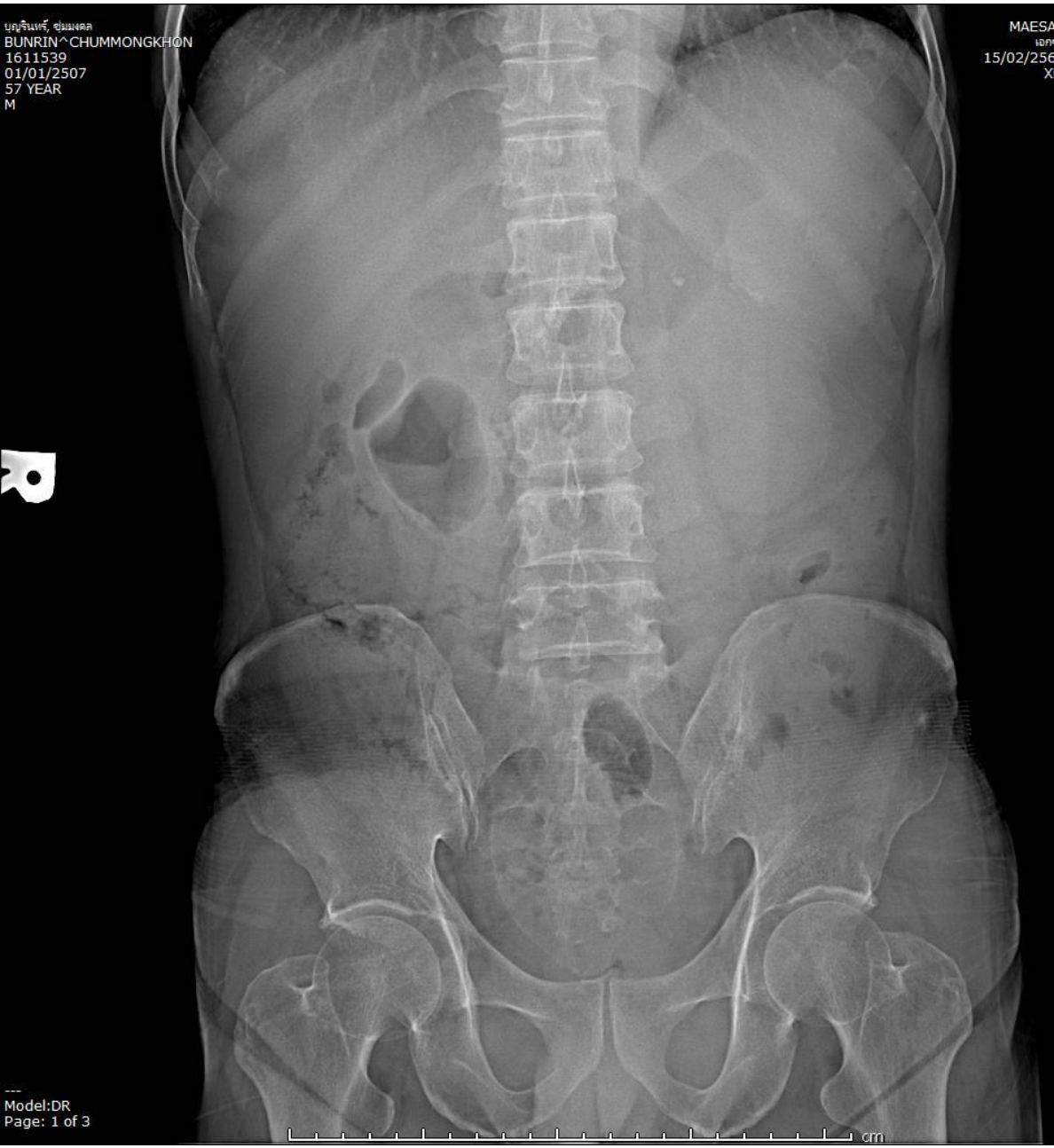
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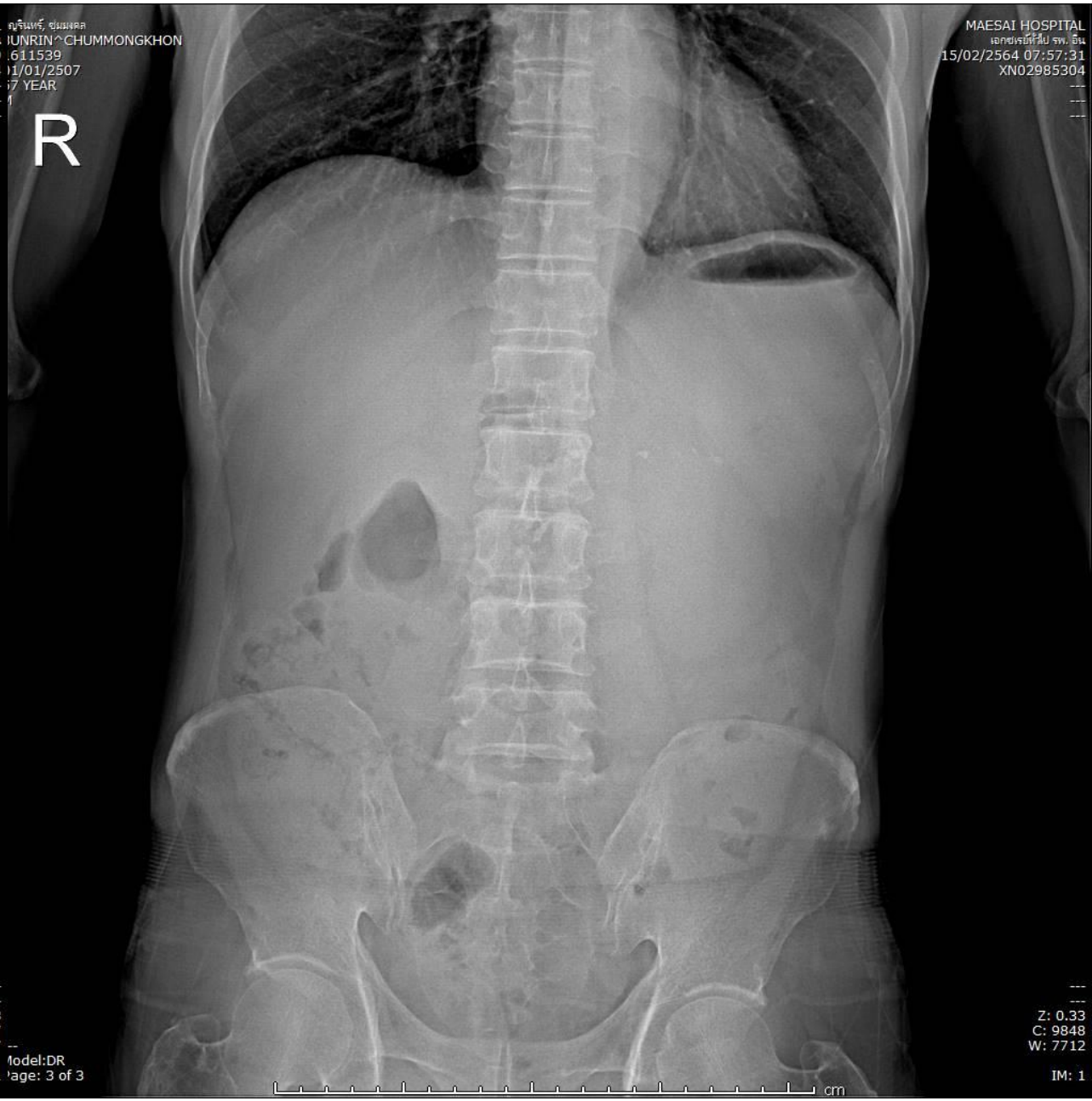
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age: 3 of 3
cm

RESTING ECG (3.50) FEB 15 2021 06:59

FILTER:HF,DF,MF2

STRESS TEST: NO

ID	=
AGE/SEX	= /
H./W.	= cm/ kg
B.P.	= / mmHg
H.R.	= 90/min(0.666s)
PR	= 0.188s
QRS	= 0.094s
AXIS	= 51deg
QT/QTc	= 0.368s/0.450
RV5	= 1.85mV
SV1	= 1.31mV
MEDICATION	=

****** ABNORMAL ECG ****** DOMINANT

434:ST-T abnormality (Lat)

Noise or baseline drift is present.

(V5)

Check for any measurement error.

Muscle filter may reduce QRS amplitude.

**** Unconfirmed. MD must review. ****

REVIEWED BY

1-1-2 4-1 5-2-1

· V6) -T = 0.27mV

วันที่ : 15/02/64 HN : 560254473

ชื่อ : นายบุญรินทร์ ชุ่มมงคล

เพศ : ชาย อายุ : 56 ปี 4 เดือน

ห้อง :

แพทย์ : .....

.....

10mm/mV 25mm/s

I II III aVR aVL aVF

V1 V2 V3 V4 V5 V6

II 25mm/s

V1 25mm/s

MAESAI HOSPITAL

[Kenz Cardico1210 V-3.50]

ABG

- pH 6.6
- pCO2 15.5
- pO2 138.8
- HCO3 1.7

ABG

- Impression: severe metabolic acidosis with respiratory alkalosis
- Management: 7.5% $NaHCO_3$ 200 ml IV push then 200 ml IV drip in 1 hr

Primary survey at CRH

Primary survey at CRH

- A: proper ETT, no secretion
- B: on ventilator, SpO2 100, clear and equal breath sound both lungs
- C: BP 128/77, HR 123, pulses 2+ all, CRT < 2 sec
- D: E4VtM6, pupils 2 mm RTL BE, DTX 129%

Arterial blood gas

- pH 7.24
- pCO2 23
- pO2 271
- HCO3 12.5
- BE -16.3
- O2sat 100%

Secondary survey at CRH

Secondary survey at CRH

- A: none
- M: allopurinol (100) 1x1 po pc, colchicine (0.6) 1x1 po pc, metformin (500) 1x2 po pc, enalapril (20) ½ x 2 po pc, amlodipine (5) 1x1 po pc, clopidogrel (75) 1x1 po pc
- P: U/D: DM, HT, Gout
- L: 10.00 am
- E: ปวดท้อง 1 วันก่อน ปวดที่ใต้ลิ้นปี่ อาเจียนมากกว่า 20 ครั้ง ไม่ถ่ายเหลว หายใจเร็วมากขึ้น

Event

4 วันก่อน ไม่ค่อยทานอาหาร ดื่มแต่สุรา

2 วันก่อน ปวดที่ใต้ลิ้นปี่ ปวดแน่นๆ ไม่ร้าวไปไหน อาเจียนมากกว่า 20 ครั้ง เป็นน้ำลาย ไม่ถ่ายเหลว ไม่มีไข้ ไม่มีเลือดปน หายใจเหนื่อย ไม่ไอ หยุดดื่มสุรา

วันนี้ ปวดท้องมากขึ้น ร่วมกับหายใจเหนื่อยมากขึ้น จึงมาโรงพยาบาล

Physical examination

- Vital signs: pulse rate – 77 bpm, respiratory rate - ETT, body temperature 36.6 C, blood pressure 128/77 mmHg
- General appearance: a middle age man with normal consciousness, no jaundice, no cyanosis
- HEENT: mild pale conjunctiva, no icteric sclera, retained NG tube with pale red content – 30 ml
- Skin: no rash, no petechiae
- LN: can’t be palpated
- CVS: regular rhythm, no murmur

Physical examination

- Lungs: clear and equal breath sound both lungs
- Abdomen: tenderness at epigastrium with voluntary guarding, no signs of chronic liver disease
- Extremities: no pitting edema
- Neuro: awake and aware
- PR: empty rectum, no mass

Problem lists

- A 56 years old man with DM, HT, gout present with
  - Acute epigastric pain
  - Acute dyspnea with Kussmaul breathing

Differential diagnosis

Adjunct to secondary survey

Adjunct to secondary survey

- CBC
- BUN, Cr, Electrolytes, Ca, Mg, PO4, LFT
- Blood lactate
- Serum amylase
- NG lavage 500 ml

Complete blood count

- Hb 9.5 g.dL
- Hct 29.5 %
- MCV 66.3 fl
- MCH 21.3 fl
- WBC count 7800 cell/cu.mm
- Neutrophil 78.5 %
- Lymphocyte 19.1 %
- Monocyte 2%
- Platelet 131000

Coagulogram

- PT 12.2 sec
- PTT 28.5 sec
- INR 1.09

Electrolytes

- Sodium 143 mmol/L
- Potassium 3.5 mmol/L
- Chloride 81 mmol/L
- Carbondioxide 21 mmol/L
- Calcium 6.8 mg/dl
- Magnesium 1.6 mg/dl
- Phosphorous 7.7 mg/dl
- BUN 42 mg/dl
- Creatinine 2.51 mg/dl
- Serum amylase

Liver function test

- AST 93 U/L
- ALT 34 U/L
- ALP 155 U/L
- TB 0.6 mg/dl
- DB 0.2 mg/dl
- Albumin 3.5 gm/dl
- Serum amylase 99

Management at ER

- ABG
- NG lavage 500 ml -> coffee ground
- Hct stat 29%
- 7.5% $NaHCO_3$ 100 ml IV push
- Pantoprazole 80 mg IV stat
- G/M PRC 2 u -> hold
- Ceftriaxone 2 gm IV OD
- Thiamine 100 mg IV OD
- Admit MED

Metabolic acidosis

Definition

- A pathologic process that, when unopposed, increases the concentration of hydrogen ions in the body and reduces the HCO_3 concentration.

Major causes of metabolic acidosis according to mechanism and anion gap

Mechanism of acidosis	Increased AG	Normal AG
Increased acid production	Lactic acidosis	
	Ketoacidosis	
	Diabetes mellitus	
	Starvation	
	Alcohol associated	
	Ingestions	
	Methanol	
	Ethylene glycol	
	Aspirin	
	Toluene (if early or if kidney function is impaired)	Toluene ingestion (if late and if kidney function is preserved; due to excretion of sodium and potassium hippurate in the urine)
	Diethylene glycol	
	Propylene glycol	
	D-lactic acidosis	A component of non-AG metabolic acidosis may coexist due to urinary excretion of D-lactate as Na and K salts (which represents potential HCO_3)
	Pyroglutamic acid (5-oxoproline)	

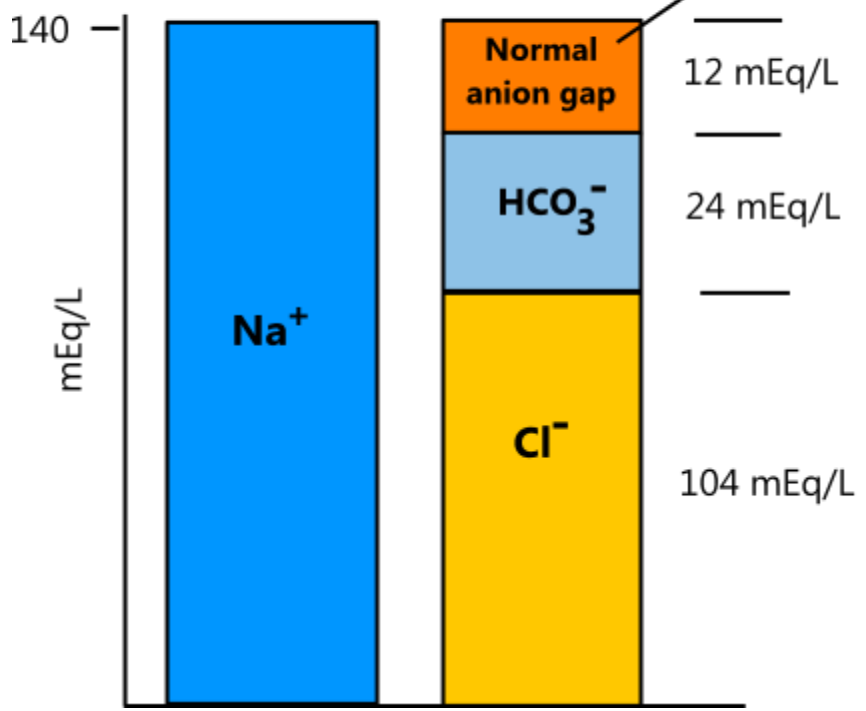
Loss of bicarbonate or bicarbonate precursors		Diarrhea or other intestinal losses (eg, tube drainage)
		Type 2 (proximal) RTA
		Posttreatment of ketoacidosis
		Carbonic anhydrase inhibitors
		Ureteral diversion (eg, ileal loop)
Decreased renal acid excretion	Severe kidney dysfunction (eGFR <15 to 20 mL/min/1.73 m^2)	Moderate kidney dysfunction (eGFR >15 to 20 mL/min/1.73 m^2)
		Type 1 (distal) RTA (hypokalemic)
		Hyperkalemic RTA
		Type 4 RTA (hypoaldosteronism)
		Voltage defect
Large volume infusion of normal saline		Diffusion acidosis

AG: anion gap; RTA: renal tubular acidosis.

The Boston formulae*			
State	Rule	Formula	Range
Metabolic acidosis	1.5+8	PCO_2 (mmHg) = 1.5*bicarbonate + 8	2
Metabolic alkalosis	0.7+20	PCO_2 (mmHg) = 0.7*bicarbonate +20	5
Acute respiratory alkalosis	2 for 10	bicarbonate (mmol/l) drops 2 mmol/l for every 10 mmHg PCO_2 drop	?
Chronic respiratory alkalosis	4 for 10	likewise, but 5 mmol/l	?
Acute respiratory acidosis	1 for 10	bicarbonate (mmol/l) increases 1 mmol/l for every 10 mmHg	?
Chronic respiratory acidosis	4 for 10	likewise, but 4 mmol/l	?

The Anion Gap

- primarily composed of albumin
- also contains phosphate, sulfate, and other anions



Anion gap = Na^+ - (Cl^- + HCO_3^-)

Corrected anion gap for hypoalbuminemia =
(4 - serum albumin concentration) X 2.5 + calculated anion gap

Reference [1,2,5]

Causes of high anion gap metabolic acidosis

- Lactic acidosis
  - Type a
  - Type b
  - Type d
- Ketoacidosis
  - Diabetic
  - Alcoholic
  - starvation
- Alcohol ingestion
  - Methanol
  - Ethylene glycol
- Salicylate poisoning

Causes of normal anion gap metabolic acidosis

- Diarrhea
- Proximal RTA
- Acute or chronic kidney disease

Lactic acidosis

Lactic acidosis occurs when lactic acid production exceeds lactic acid clearance. The increase in lactate production is usually caused by impaired tissue oxygenation, either from decreased oxygen delivery or a defect in mitochondrial oxygen utilization.

Lactic acidosis is generally defined as a plasma lactate concentration greater than 4 mmol/L

Etiology of lactic acidosis

Increased lactate production
Increased pyruvate production
Enzymatic defects in glycogenolysis or gluconeogenesis (as with type 1 glycogen storage disease)
Respiratory alkalosis, including salicylate intoxication
Pheochromocytoma
Beta-agonists
Sepsis
Impaired pyruvate utilization
Decreased activity of pyruvate dehydrogenase or pyruvate carboxylase
• Congenital
• Possibly a role in diabetes mellitus, Reye syndrome
Altered redox state favoring pyruvate conversion to lactate
Enhanced metabolic rate
• Grand mal seizure
• Severe exercise
• Hypothermic shivering
• Severe asthma
Decreased oxygen delivery
• Shock
• Cardiac arrest

• Acute pulmonary edema
• Carbon monoxide poisoning
• Severe hypoxemia (PO_2 <25 to 30 mmHg)
• Pheochromocytoma
Reduced oxygen utilization
• Cyanide intoxication (decreased oxidative metabolism), which may result from cyanide poisoning or, during a fire, from smoke inhalation of vapors derived from the thermal decomposition of nitrogen-containing materials such as wool, silk, and polyurethane
• Drug-induced mitochondrial dysfunction due to zidovudine or stavudine
• Sepsis
D-lactic acidosis
Primary decrease in lactate utilization
Hypoperfusion and marked acidemia
Alcoholism
Liver disease
Mechanism uncertain
Malignancy
Diabetes mellitus, including metformin in the absence of tissue hypoxia
Acquired immunodeficiency syndrome
Hypoglycemia
Idiopathic

Although this table has been divided into either increased production or decreased utilization of lactate, there is considerable overlap among listed causes.

Type A lactic acidosis

- Most cases of lactic acidosis are due to marked tissue hypoperfusion resulting from hypovolemia, cardiac failure, sepsis, or cardiopulmonary arrest

Type B lactic acidosis

The mechanisms that may be involved in toxin-induced impairment of cellular metabolism and regional areas of ischemia.

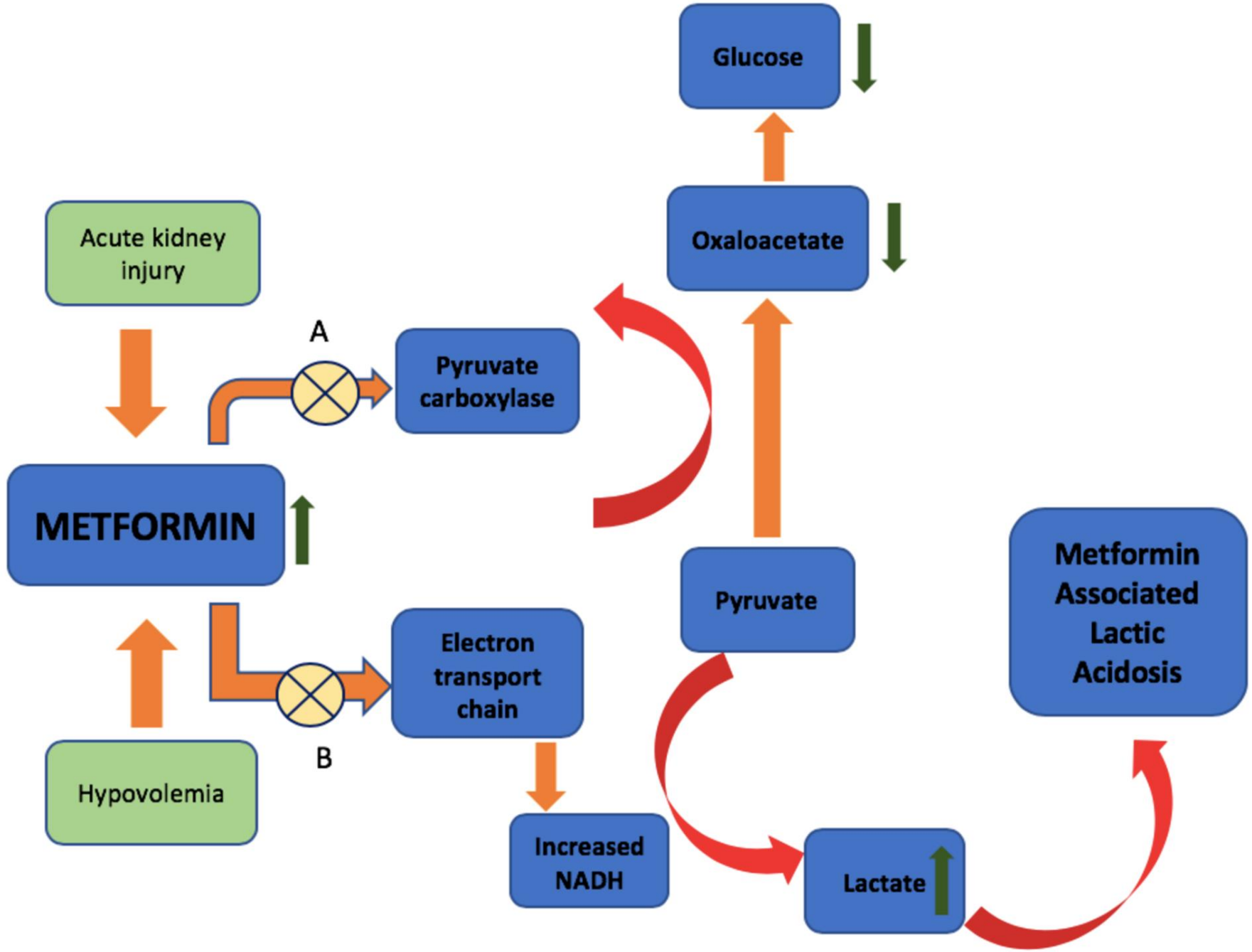
- Diabetes mellitus: Biguanide therapy (metformin)
- Malignancy: leukemia, lymphoma, and solid malignancies
- Alcoholism: hepatic dysfunction
- Beta-adrenergic agonists: IV epinephrine
- Mitochondrial dysfunction: NRTI, propofol, linezolid

Metformin associated lactic acidosis

- 10 cases per 100,000 patient years
- May occur following acute overdose
- Rarely develops in patients without comorbidities such as kidney or hepatic insufficiency or acute infection.

Risk factors

- Impaired kidney function
- Concurrent liver disease
- Active alcohol abuse
- Unstable or acute heart failure
- History of lactic acidosis during metformin therapy
- Decreased tissue perfusion or hemodynamic instability
- Hypoxic states or serious acute illness


Glucose
Oxaloacetate
Acute kidney injury
A
Pyruvate carboxylase
METFORMIN
Pyruvate
Metformin Associated Lactic Acidosis
Electron transport chain
Hypovolemia
B
Increased NADH
Lactate

Clinical presentation

- Nausea/vomiting
- Abdominal pain
- Diarrhea
- Alteration of conscious
- Shortness of breath

MANAGEMENT OF OVERDOSE OR TOXICITY

- Airway, breathing, circulation
- Gastrointestinal decontamination: activated charcoal in acute ingestion
- Lactic acidosis
  - Sodium bicarbonate: maintain the pH above 7.1 (or above 7.3 in patients with severe acute kidney injury), until the acute toxicity resolves.
  - Hemodialysis: indications
    - Serum lactate > 20 mmol/L
    - pH < 7
    - Failure to improve with $NaHCO_3$ in 2-4 hr

Disposition

- Clinical improve after observed at least 6-8 hours
- Stop using metformin

Type D lactic acidosis

- A rare form of lactic acidosis that can occur in patients with short bowel syndrome or other forms of gastrointestinal malabsorption. Causse by, abnormally large amounts of glucose and starch are metabolized (fermented) by intestinal bacteria to multiple organic acids

Thank you