CASE REPORT

AKI-LEPTOSPIROSIS

- A 47-year-old Caucasian man, from a rural area, presented to the emergency department with:
- Nausea
- Vomiting
- Fever
- Bloody diarrhoea
- Headache
- Dry cough
- Shortness of breath
- Myalgia
- Decreased urine output (dark urine)

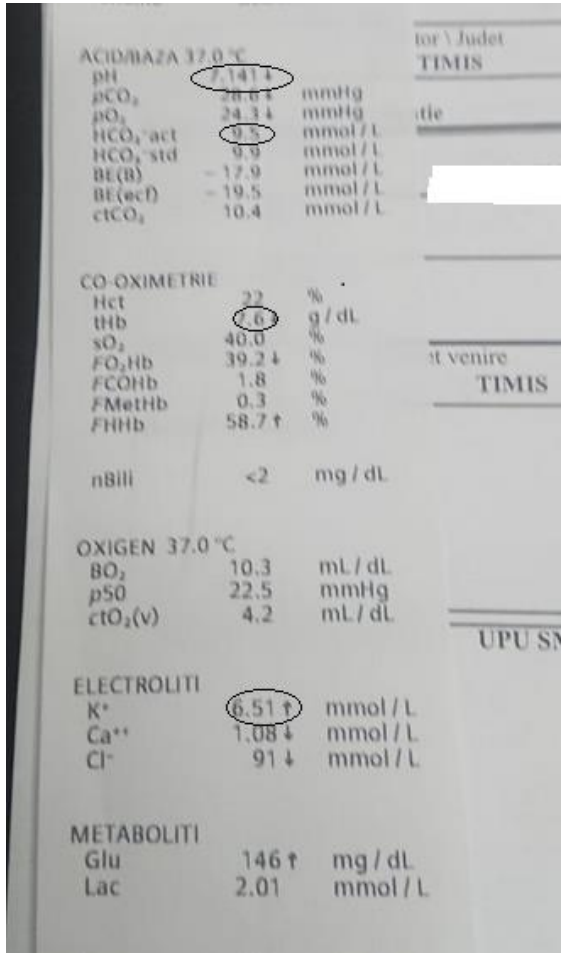
for five days

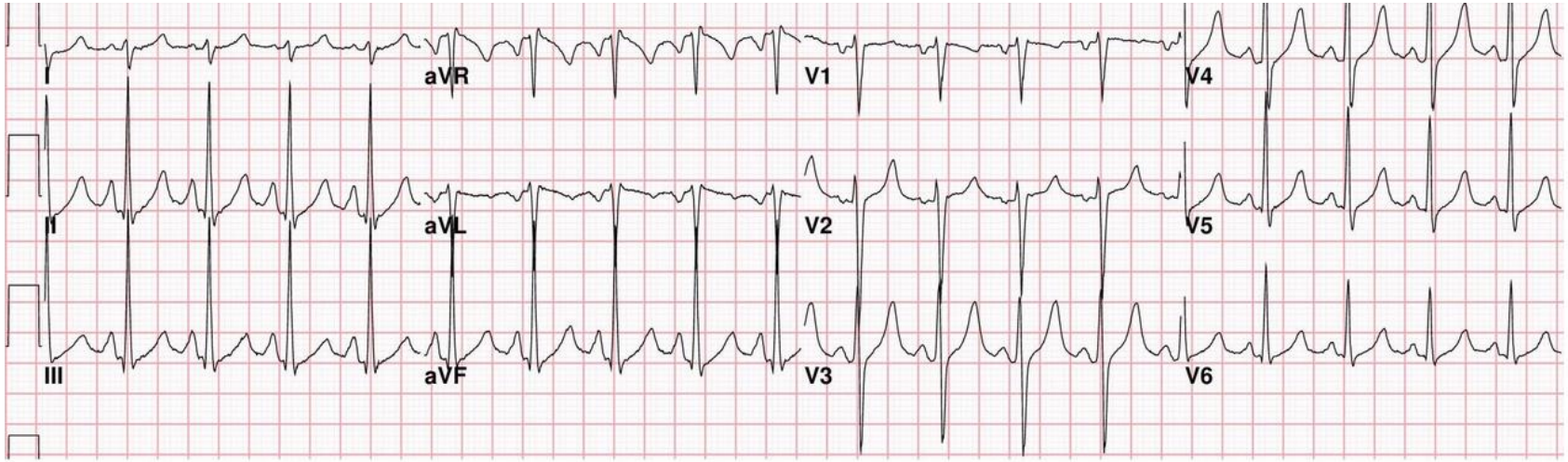
He presented to his primary care physician on the third day of illness and was treated with supportive measures for presumed viral gastroenteritis. His nausea and diarrhoea improved, but he continued to have poor oral intake, oliguria, and increased weakness.

Physical examination:

- temperature - 38.4°c;
- dry mucous membranes, icteric sclera, jaundice
- abdominal tenderness
- blood pressure (BP)- 100/50 mmHg and HR-A 110 bpm
- respiratory rate- 24/min, auscultation - normal breath sounds
- Urinary catheterization – **approx. 50 ml dark urine**

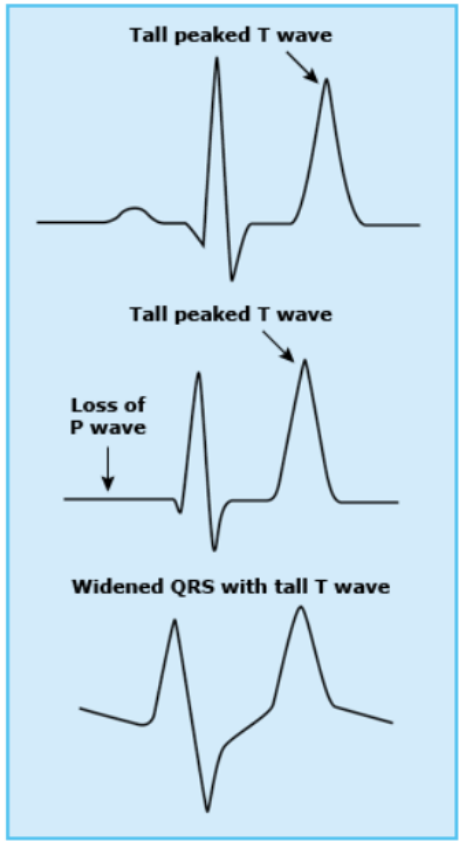
- Laboratory exam:
- ASTRUP : metabolic acidosis (NaHCO3- = 9.5 mmol/L , NV [23-27 mmol/L) and moderate hyperkalaemia (peaked T-waves on EKG)





Q1:Which are the typical electrocardiographic features of hyperkalaemia?

Typical electrocardiographic features of hyperkalemia



Serum potassium	Major change
5.5-6.5	Tall peaked T waves
6.5-7.5	Loss of P waves
7.0-8.0	Widening of QRS
8.0-10.0	Sine wave, ventricular arrhythmia, asystole

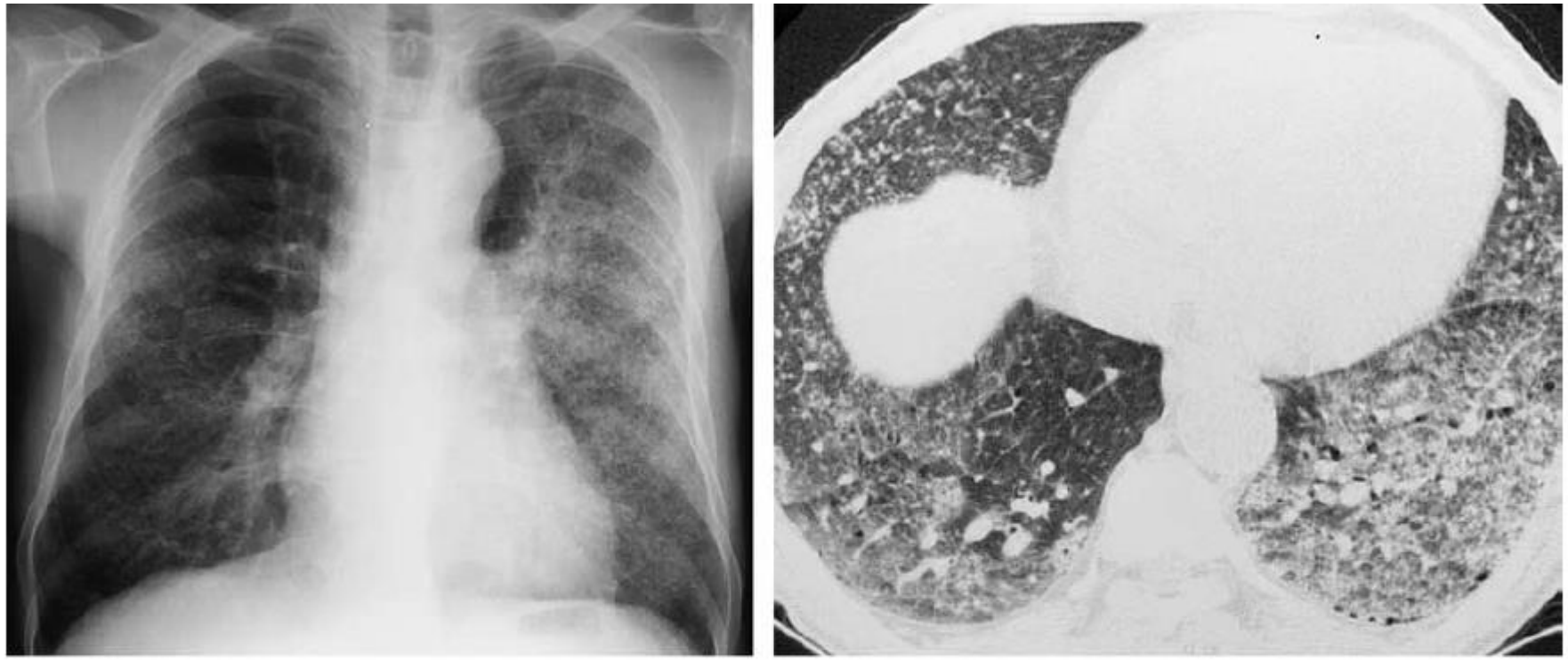
Q2. Which is the therapy of hyperkalaemia?

- 1 **Antagonists of potassium impact upon membranes**
- ☺ Calcium gluconate 10% i.v. (! NO if patients is digitised)
- 2 Drive extracellular potassium into the cells
- ☺ Insulin and glucose
- ☺ Sodium bicarbonate (NaHCO3 8.4%), primarily if metabolic acidosis
- Beta-2-adrenergic agonists (i.e. Ventolin)
- 3 Removal of potassium from the body
- ☺ Loop or thiazide diuretics (Furosemidum i.v.) !!!if patients are dehydrated FIRST replete hypovolemia with isotonic saline
- Cation exchange resin (sodium polystyrene sulfonate -Resonium)
- Dialysis, preferably hemodialysis if severe
- *! ☺ these therapies were applied in the Emergency Unit in this patient*

Parameter	Value	Normal range	Parameter	Value	Normal range
WBC	26.700/mmc	5000 – 9000	Glycaemia	80 mg/dl	65 -115
Ne	92%	52 – 74%	ASAT	650 U/L	2 -37
Hb	7.8 g/dl	11.5 – 15.5	ALAT	800	0 -41
Ht	28%	35 – 45%	BT	8 mg/dl	0.1 - 1
PLT	31.000/mmc	150.000-400.000	BD	4 mg/dl	0 - 0.4
Fibrinogen	100 mg/dl	170-420	LDH	2100 U/L	313 - 618
INR	1.4	0.8 – 1.07	CK	1500 U/L	30 -170
CRP	270 mg/L	< 3	CK-MB	14 U/L	0 -16
Procalcitonin	>10 ng/ml	<0.5	Na	130 mmol/L	136-145
D-Dimers	1800 microg/ml	<0.5	K	6.6 mmol/L	3.5 - 5
Creat	9.5 mg/dl	0.5 – 1.1	Peripheral blood smear : 1% schistocytes		
BUN	350 mg/dl	10 - 40	Urinalysis	Proteinuria + Blood + Leucocituria + Nitriti - neg	
Serum albumin	1.8 g/dl		Rapid test for Clostridium Difficile Toxin A & B (stool) - NEGATIVE		

Calcium	
Phosphorus	NORMAL
iPTH	
25-OH-Vit D	

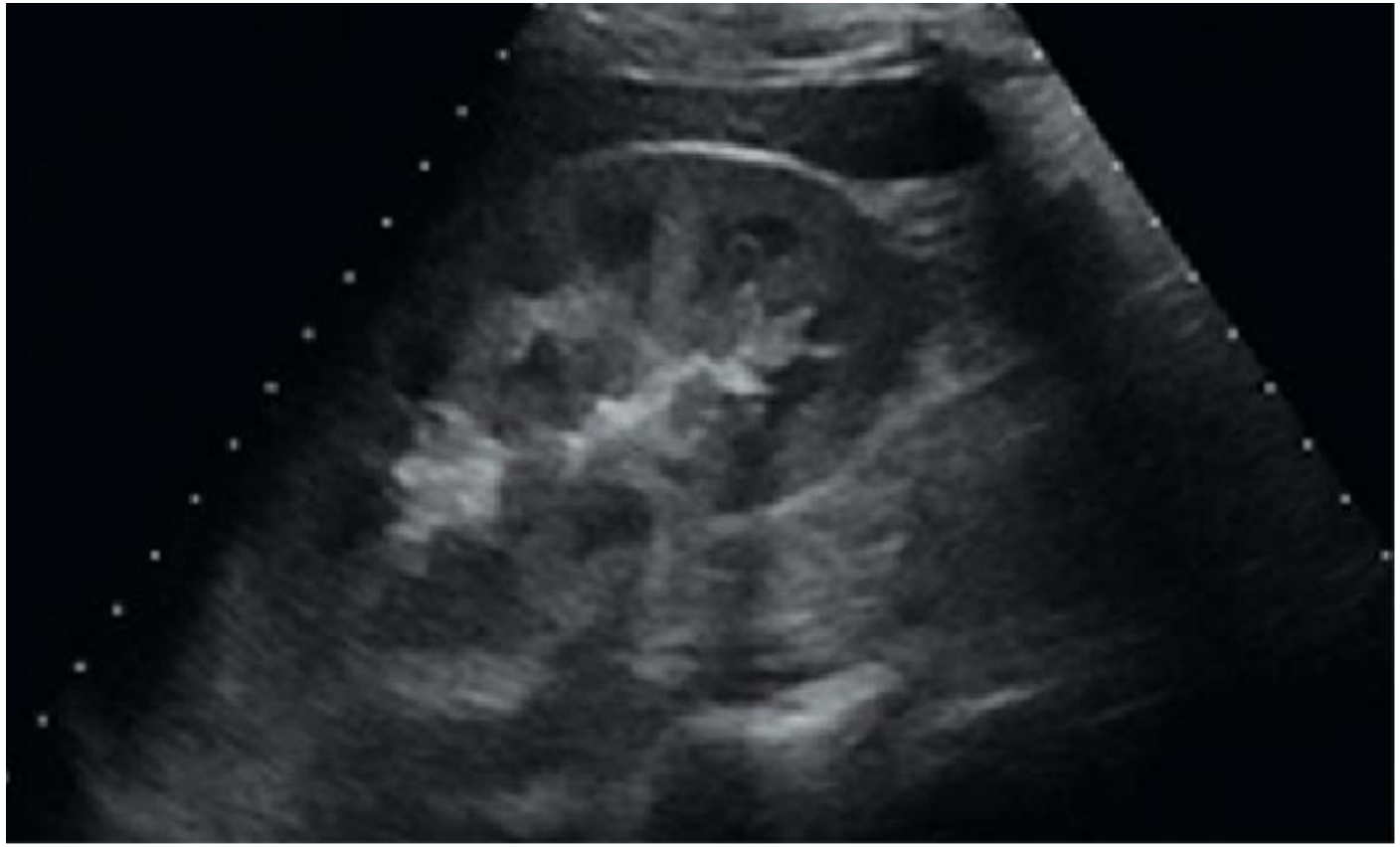
- X-Chest Rg:



a. b.

chest radiograph shows fine reticulonodular opacities throughout both lungs. (b) High-resolution CT scan obtained the same day shows ground-glass attenuation, intralobular interstitial thickening,

ABDOMINAL ULTRASOUND : normal kidney size with thick hyperechogenic parenchyma and contrasting hypoechogenic pyramids, NO signs of cholecystitis or angiocholitis

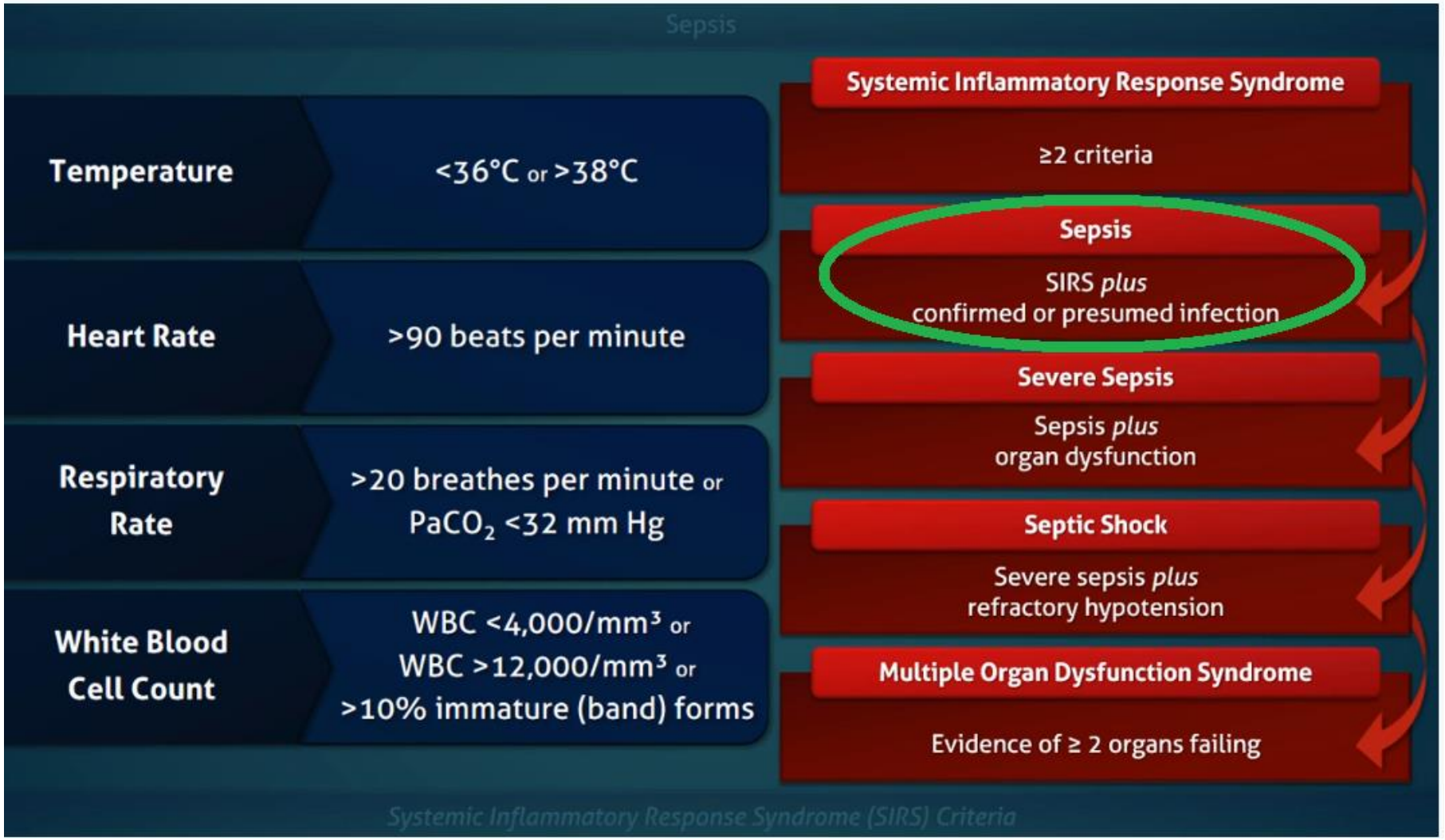


Q3. Which is the preliminary diagnosis in the Emergency Dept?

- **Gut-derived sepsis**
- **Acute gastroenteritis**
- **Acute kidney injury AKIN 3 (AKI AKIN 3)**
- **Moderate thrombocytopenia**
- **Microangiopathic haemolytic anaemia (MAHA) * ***
- **Metabolic acidosis**
- **Moderate hyperkalaemia**

* * fragmentation of erythrocytes occurred as they passed through the thrombotic meshwork and strictured microvasculature (presence of peripheral schistocytes + elevated serum lactate dehydrogenase + markers of hemolysis (*e.g.*, indirect bilirubin and haptoglobins)

Q4. What are the criteria for the diagnosis of “Sepsis”?



Q5.Which is the AKI definition?

- Acute kidney injury (AKI), formerly called acute renal failure (ARF), is commonly defined as an abrupt decline in renal function. KDIGO defines AKI as any of the following:

- **Increase in serum creatinine by 0.3mg/dL or more within 48 hours**

or

- **Increase in serum creatinine to 1.5 times baseline or more within the last 7 days**

or

- **Urine output less than 0.5 mL/kg/h for 6 hours**

Q6. KDIGO Staging for AKI Severity?

Stage	Serum Creatinine	Urine Output
1	1.5-1.9 times baseline or ≥0.3 mg/dL increase	< 0.5 mL/kg/h for 6 h
2	2-2.9 times baseline	< 0.5 mL/kg/h for 12 h
3	3 times baseline or Increase in serum creatinine to ≥4 mg/dL or Initiation of renal replacement therapy	< 0.3 mL/kg/h for 24 h or Anuria for ≥12 h

Q7. Please complete the most frequent complications associated to AKI 😂😂

Q8. Differential diagnosis AKI VS. CKD

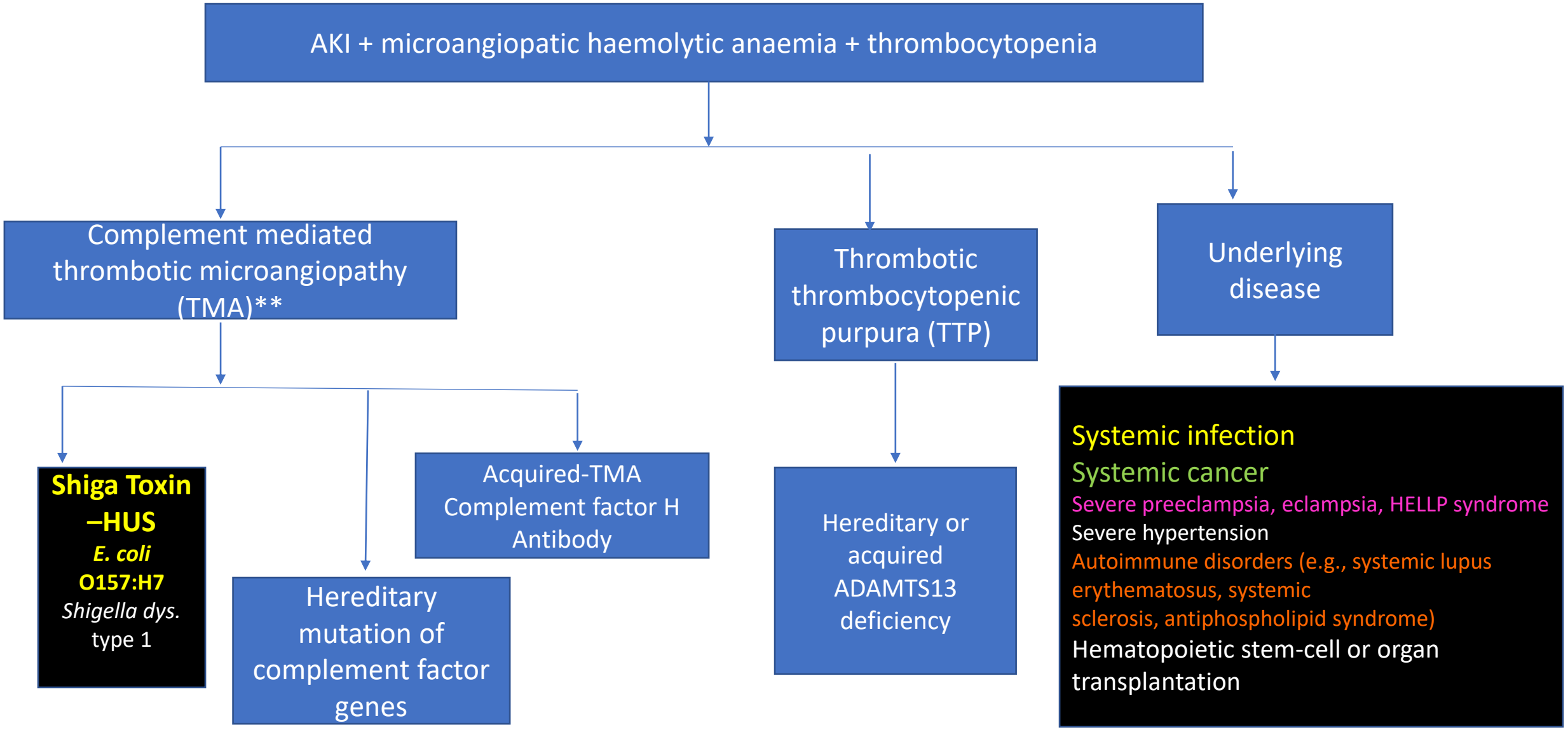
	AKI	CKD
Causative factor	- Renal hypoperfusion (dehydration, sepsis, acute myocardial infarction, etc) - Exposure to nephrotoxics (NSAIDS, AMINOGLYCISIDES)	---------
Comorbidities	CKD	Diabetes mellitus, Arterial Hypertension
Clinical signs	Tahipneea (due to metabolic acidosis)	Nictoria, Cutaneos changes: xerosis, pruritus, palour
Complications of Kidney disease	Metabolic acidosis – poor tolerated NO Mineral and Bone Disorder(MBD) related to CKD	Metabolic acidosis – well tolerated Anaemia – well tolerated + Signs of MBD-CKD (↓ Ca, ↓25-OH-D3; ↑ PTH,↑ P)
Renal Ultrasound	**normal kidney size with thick hyperechogenic parenchyma and contrasting hypoechogenic pyramids**	smaller kidney, thinning of the parenchyma and its hyperechogenicity (reflecting sclerosis and fibrosis) EXCEPTION – DIABETIC NEPHROPATHY

The patient was admitted to the Nephrology Dept.

- THERAPY:
- antimicrobial therapy -Meropenem 500mg I.V., every 12 h
- methylprednisolone 500 mg, daily was given intravenously for haemorrhagic alveolitis
- fresh frozen plasma and concentrated units PLT to correct the coagulopathy
- intravenous solutions for correction of hydro-electrolytic and acid-base disturbances
- BUT:

The patient's condition continued to deteriorate with decreasing platelet counts, worsening renal failure, and hyperbilirubinemia; **haemodialysis** was then initiated at 24 h after admission

…and now, as a veritable nephrologist, look for the AKI etiology



- LABORATORY RESULTS:

Parameter	Results	Interpretation
Two stoool culture (taken on day 1 and 2)	negative for *Escherichia coli* O157:H7, Shigella, Salmonella,	NO Shiga-toxin HUS
Complement C3	1.03 g/L (0.9-1.8 g/L)	
Complement C4	0.34 g/L (0.15-0.55)	
Anti-factor H IgG autoantibody	was not measured	
sC5b-9 (terminal compleent complex)	was not measured	
ADAMTS 13 activity	60% (NV >10%)	NO Thrombotic thrombocytopenic pur
Haptoglobin	0.01 g/L (0.3-2 g/L)	Support ongoing hemolysis
Antinuclear antibody panel	NEGATIVE	NO autoimmune disease
Anticardiolipin antibodies (IgM, IgG), Anti-beta2-GP I antibod	NORMAL VALUE	NO Antiphospholipid syndrome
Leptospira antibody	**Ig M titers of more than 1:3200 (ELISA)**	**LEPTOSPIROSIS**
HANTAVIRUS antibody	NEGATIVE	
HBs Antigen	NEGATIVE	
Hepatitis virus E Antibody	negative	
HCV antibody	NEGATIVE	
Two blood and urine cultura (taken on day 1 and 2)	negative for bacteria and fungus	

The diagnosis of the nephrologist is

Severe form of leptospirosis or Weil's disease

- Intravenous ceftriaxone 1g every 12 hours was also started; the patient's condition improved after initiation of ceftriaxone. The other antibiotic (Meropenem) was discontinued.
- Haemodialysis was discontinued when the patient started producing urine, followed by improved kidney function – at 12 day after admission

Patient's outcome

Parameter	Day 1	Day 2	Day 5	Day 10	Day 14
WBC/mmc	26.7	30	18	8900	7000
Hb (g/dl)	7.8	7	7.9	8.2	8.5
PLT (/mmc)	31	28	50	110	150
CRP (mg/L)	270	300	200	87	25
Creat (mg/dl)	9.5	11	8	7.5	3.8
Ureea (mg/dl)	350	400	280	190	85
BT (mg/dl)	8	10	5.5	4	2.3
BD (mg/dl)	4	5.6	3	1.8	0.8
ALAT (U/L)	800	750	390	200	85
LDH (U/L)	2100	2700	1800	1200	450
Albumin (g/dl)	1.8		2.1	2.5	2.8

OUTCOME AND FOLLOW-UP

- The patient was feeling better and was discharged on day 16 after his condition further stabilized.

- At his 3 mo.- follow-up visit, his serum creatinine level was 1.2 mg/dl (RFG = 71.6 ml/min/1.73m2)

Summaries about Leptospirosis

- Leptospirosis is a zoonosis caused by pathogenic spirochetes of the genus *Leptospira*
- Rodents are the most important reservoirs
- Human infection -exposure to environmental sources, such as animal urine, contaminated water or soil, or infected animal tissue; portals of entry include cuts or abraded skin, mucous membranes, or conjunctivae.
- The infection may rarely be acquired by ingestion of food contaminated with urine or via aerosols.

Summaries about Leptospirosis

- The illness generally presents with the abrupt onset of **fever, rigors, myalgias, and headache** in 75 to 100 percent of patients
- **Conjunctival suffusion, nausea, vomiting, and diarrhoea** occur in approximately 50 percent of cases
- Pulmonary involvement in leptospirosis ranges from20–70% (**pulmonary haemorrhage, is a serious complication of leptospirosis**).
- Aseptic meningitis is observed in 50 to 85 percent of patients
- **Renal failure is often nonoliguric**; supportive renal replacement therapy may be required during the acute phase; renal recovery is generally complete

Summaries about Leptospirosis

- **Diagnosis:**
- Clinical – epidemiologic exposure
- Serology — Serological tests are used most frequently for diagnosis of leptospirosis [antibodies only appear from day 5 to 7 of illness]

Treatment: For hospitalized adults with severe disease, we favour treatment with penicillin (1.5 million units intravenously [IV] every six hours), doxycycline (100 mg IV twice daily), ceftriaxone (1 to 2 g IV once daily), or cefotaxime (1 g IV every six hours)

- **! A Jarisch-Herxheimer reaction** may occur following antimicrobial therapy for leptospirosis; this is an acute inflammatory response to clearance of spirochetes from the circulation and is characterized clinically by fever, rigors, and hypotension

Thank – you and have a nice day !