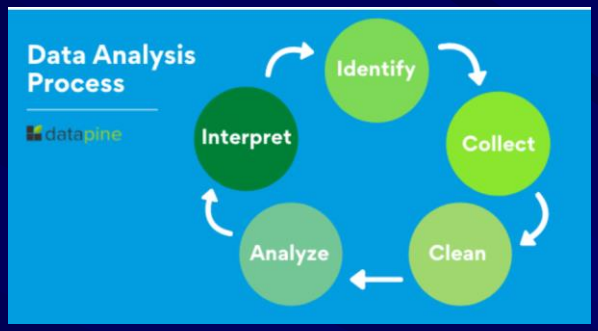
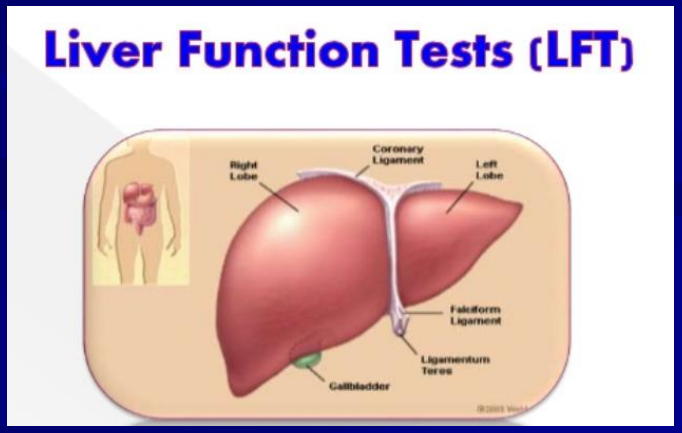


Clinical application of Laboratory data-part 2, (2024)

善用臨床檢驗-實務訓練

Cheng-Yi WANG

2024.05.10



臨床檢驗解析之步驟

- 1. 這是什麼變化?哪一個器官受損
- 是否嚴重--會不會是器官衰竭的現象
- 2. 什麼原因造成? 疾病?藥物? 檢驗誤差?
- 正常的變異 (normal variation) or ?
- 3. 怎麼辦? Management– How to treat it.
- 4. 如何評估是否改善? Assessment
- parameters

Laboratory tests之價值

- Diagnostic—直接指出疾病
- AC sugar : 200，HbA1c: 7.7-→DM
- Severity ---看出嚴重性,
- CRP>12, Bilirubin > 5 mg/dl, HB :<8 gm/dl
- 某一些疾病/狀態之可能性
- CEA>5,→ Cancer or false positive
- MCV<80-→ microcytic change-→**Fe. Deficiency or chronic blood loss**
- 特殊應用
- BUN/ Cr. >30 indicated bleeding in UGI tract

檢驗的思考羅輯

- 1.事前思考:我為何作這個檢驗:**目的**在診斷還是排除一個疾病.
- 2.**期待值**是什麼:
- 正常值,還是不正常?
- 3. **結果**是什麼?
- 期待值或非期待值-→
- 4. 那代表何種意義.
- **診斷確立**,嚴重度知,
- 還是**意料之外**,另有問題

善用臨床檢驗-實務訓練

- 下提出5個案例 分別是各個器官系統發生的問題.依據我們提出的解析步驟來分析
- 1.acute liver necrosis
- 2.acute pancreatitis
- 3.Cancer ? Or others—false positive or
- true positive
- 4.Chest pain , AMI or ?
- 5.Thromboembolic phenomenon.

Case 1, acute liver necrosis

- Case 1, a previously healthy, 34-year-old Chinese female was admitted to the emergency department (ED) with the symptoms of refractory nausea, vomiting, watery diarrhea, and cold sweats, which had lasted for 3 days after **eating wild mushrooms**. She did not present with fever, hematemesis, syncope, or disturbance of consciousness. The patient's blood pressure was 80/60 mmHg at the time of referral to the ED, with other vital signs and physical examination results appearing benign overall. The leftover samples of the mushrooms she had ingested were sent to the Shanghai Academy of Agricultural Sciences, China, and identified through gross morphology to be Lepiota brunneoincarnata Chod. et Mart. (Genus: Lepiota, Species: L. brunneoincarnata).

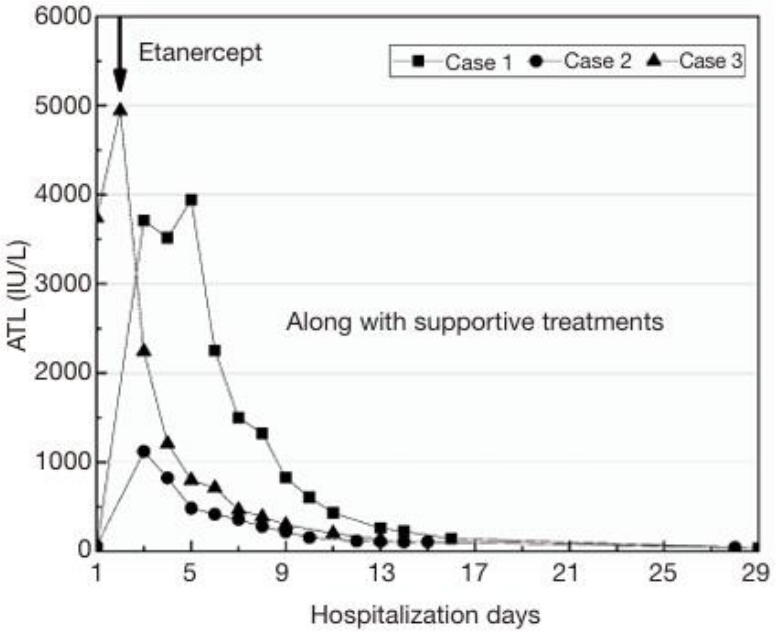


Figure 1 The variation tendency of alt of the three cases. ALT, alanine aminotransferase, normal range, 0–75 IU/L.

Case 2, Case 2 was a 50-year-old Chinese male without prior known disease. And he was the husband of the patient from case 1. He had ingested the same mushrooms and displayed exactly the same gastrointestinal symptoms as his wife. On admission, there were no abnormalities in his vital signs or physical examination results. From his lab results, the patient was determined to have a lower peak value of ALT than his wife. Based on his condition, he was given an injection of 25 mg etanercept on days 3, 6, and 9 of hospitalization, coupled with supportive treatment similar to that of his wife. He was discharged on day 15, as the level of ALT was approximately normal. He had completely recovered 2 weeks later (Figure 1).

Case 3, acute liver necrosis due to Acetaminophen (25-year-old female)

Case 1

- The patient's lab tests showed a gradual increase in the indices of alanine aminotransferase (ALT), aspartate transaminase, total bilirubin, and prothrombin time. Considering her progressive and aggravated clinical symptoms and laboratory parameters, the patient was diagnosed with acute mushroom intoxication and was considered to be at short-term risk of ALF; emergent hemodialysis (HD) was prepared for treatment. According to the recommendations of a consultant from the Digestive Department (DD),

Course and treatment case 1

- The patient received treatment with 3 etanercept injections (50 mg on day 2, and 25 mg on days 8 and 11 of hospitalization), as well as traditional supportive treatment including methylprednisolone, γ-immunoglobulin, reduced glutathione (GSH), polyene phospholipids, and coenzyme complex. On day 5 of admission, the patient's ALT level began to decrease rapidly, and her clinical manifestations improved. She was discharged on day 16 with almost normal laboratory results.

An outpatient follow-up visit 2 weeks later showed that the patient's liver enzyme measurements had fully recovered (Figure 1).

- Cases 1 and 2 were poisoned by α-Amanitin, which is known to be contained in Lepiota brunneoincarnata Chod et Mart. TNF-α is known to aggravate α-Amanitin's hepatotoxicity through interacting with its reactive oxygen species (ROS) and shortening its latency period of hepatotoxicity (3). The toxicity mechanism of case 3 was hepatotoxicity by a reactive metabolite of APAP. Treatments for poison-induced liver injury include gastric decontamination, extracorporeal treatment, **drug therapy**, and in severe cases, eventual liver transplantation (LT). The death rate remains high, despite this range of treatment options (3).

(3) Garcia J, Costa VM, Carvalho A, et al. Amanita phalloides poisoning: Mechanisms of toxicity and treatment. Food Chem Toxicol 2015;86:41-55.

- Acute hepatocyte injury brings about mitochondrial injury and the release of multiple damage-associated molecular patterns, including high-mobility group box 1 protein (HMGB1). HMGB1 subsequently binds to toll like receptor 4 (TLR4) on Kupffer cells (KCs) and activates the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) signal transduction pathway. This leads to the activation of KCs and secretion of pro-inflammatory cytokines and ROS, resulting in the apoptosis and necrosis of hepatocytes (4,5). TNF-α functions as a homotrimer that binds to p55 TNF receptors (TNF-R1) and p75 TNF receptors (TNF-R2). TNF-α secreted by KCs is the key to the cytokine net and is closely related to ALF aggravation (5,6). First of all, TNF-α can trigger the apoptosis program by promoting TNF-R1 on hepatocytes to express TNF receptor-associated Fas-associated death domain (FADD), and activating the effector caspase-3. Meanwhile, TNF-α gives rise to hepatic hypoxic ischemic injury and destroys hepatic microcirculation. TNF-α upregulates the expression of intercellular and vascular cell adhesion molecule 1 (VCAM-1), causing the swelling and activation of endothelial cells, aggregation of sinusoidal platelets, and invasion of peripheral immune cells. Above all, several downstream transcriptional factors, including NF-κB, are activated by TNF-α, which contributes to the activation of KCs and neutrophil aggregation. Infiltrated neutrophils release ROS and proteases, leading to the apoptosis and necrosis of hepatocytes, which starts the inflammatory cascade reaction

TNF-alpha blockers

- Over the last decades, TNF-α blockers, including etanercept, infliximab, adalimumab, certolizumab pegol, and golimumab, have been approved for the treatment of autoimmune diseases including inflammatory bowel disease, rheumatologic, and dermatological disorders. Also, it has been reported that TNF-α blockers can be used for treating severe stages of hepatic diseases such as non alcoholic fatty liver disease, alcoholic hepatitis, autoimmune hepatitis, and primary biliary cholangitis (7). Sun et al. suggested that TNF-α blocker therapy may down-regulate the level of pro-inflammatory cytokines and up-regulate anti-inflammatory cytokines (8). Guo et al. showed that TNF-α blockers could protect against lipopolysaccharide (LPS)-induced ALI by inhibiting the bioactivity of TNF-α and enhancing anti-oxidation (9), while a Canadian study on azoxymethane-induced ALF indicated that systemic block of TNF-α attenuated inflammation and delayed the progression of ALF, which could provide a novel therapeutic approach for patients awaiting LT (2). Further, a Chinese randomized controlled trial on rats with APAP-induced ALI concluded that TNF-α blockers could ameliorate APAP induced hepatic damage (10). Xu et al. found that TNF-α blockers were considered to have a potential therapeutic value in acute-on-chronic liver failure (ACLF) through improving the survival rate of rats with ACLF induced by D-galactosamine and LPS (11).

10. Zhang L, Zhan J, Wu BH, et al. The role of TNF-α in acetaminophen-induced liver injury in rats. Chinese Journal of Gastroenterology and Hepatology 2014;23:1181-3.
11 Xu Y, Wang H, Bao S, et al. Amelioration of liver injury by continuously targeted intervention against TNFRp55 in rats with acute-on-chronic liver failure. PLoS One 2013;8:e68757.

Case 2, Hepatitis E and alcoholic liver disease

- **A patient male in his 70s** who consumed alcohol experienced abdominal distention, loss of appetite, epigastric pain and dark urine (jaundice). After 5 days, he visited the local clinic near his residence, and the worsening of his liver function was observed by the obtained test results. The following day, he was referred and admitted to Nihon University Itabashi Hospital (Tokyo, Japan). Due to the patient's history of cerebral infarction, hypertension, diabetes mellitus and hyperuricemia, he regularly visited the local clinic. Aspirin, valsartan, amlodipine besylate, furosemide, sitagliptin phosphate hydrate, ipragliflozin L-proline, febuxostat and magnesium oxide were prescribed. He had also undergone surgery for his springer finger, and cefaclor, loxoprofen sodium salt and lebamipide were prescribed. **He began to consume alcohol at 12 years of age. He had also consumed horse sashimi 1 month prior. He had no history of transfusion, tattooing, drug abuse or drug allergies, and had not recently traveled abroad. He had no family history of liver disease.**

長久的飲酒史要想?

alcoholic liver disease-
→ cirrhosis

請分析 Laboratory data

- The height of the patient was 161 cm and his body weight was 69 kg. His blood pressure, pulse rate and body temperature were 157/93 mmHg, 71/min and 36.1˚C, respectively.
- A physical examination revealed that he was he had hepatic encephalopathy grade 2. and he had icterus and abdominal distension was also observed. .

Item	Values
AST	**708 IU/l**
ALT	**1,067 IU/l**
LDH	**410 IU/l**
ALP	**491 IU/l**
γ-GTP	**443 IU/l**
CPK	66 U/ml
T. Bil	**6.6 mg/dl**
D. Bil	**5.3 mg/dl**
TP	6.5 g/dl
Albumin	3.4 g/dl
BUN	**48.7 mg/dl**
Creatinine	**2.2 mg/dl**
eGFR	**24.2 ml/min/1.73 m^2**
CRP	**3.4 mg/dl**

Lab., data interpretation (LFT)

- 1. severe liver cell necrosis,
- ALT>AST:>500
- 2. Hyperbilirubinemia (Total Bilirubin:6.6)
- 3. Abnormal GGT and ALP
- 4. Albumin : borderline
- A/G: 3.4/3.1
- 4.CRP: 3.4 abnormal

找原因以及risk factors

- PT, INR 94%, 1.04
- **HbA1c : 8.6 %**
- Ac sugar :161
- Blood Ammonia : 43
- Platelet : 136k

anti-HIV	Negative
HBsAg	Negative
anti-HBc	**Positive**
anti-HBc IgM	Negative
anti-HCV	Negative
anti-HAV IgM	Negative
anti-HEV IgA	**Positive**
HEV RNA	**Positive**
ANA	Negative
AMA M2	Negative
IgG	1,391 mg/dl
IgA	336 mg/dl
IgM	143 mg/dl

Hepatitis E

Table II

Changes in biochemical and virological parameters of the patient following admission.

Day	AST (IU/l)	ALT (IU/l)	T. Bil (mg/dl)	Cre (mg/dl)	Anti-HEV IgG	COI	Anti-HEV IgM	COI	Anti-HEV IgA	COI	HEV RNA
0	708	1,067	6.6	2.2	1.724	+	2.728	+	1.963	+	+
1	533	849	6.2	1.9	1.766	+	2.517	+	2.135	+	+
5	145	315	10.3	1.4							
7	181	270	15.4	1.3							
8	192	247	15.6	1.2							
11	273	294	22.7	1.3							
13	287	320	25.9	1.4	2.680	+	2.099	+	2.279	+	+
14	246	285	24.2	1.4	2.617	+	1.901	+	2.264	+	+
15	221	277	26.3	1.4	2.630	+	1.838	+	2.218	+	+
29	69	51	13.1	1.4	2.562	+	1.720	+	2.061	+	+
32	58	49	9.3	1.3	2.582	+	1.595	+	1.848	+	+
34	62	54	8.2	1.4	2.628	+	1.577	+	1.840	+	-
36	56	52	7.1	1.4	2.562	+	1.420	+	1.771	+	-
39	60	61	6.5	1.4	2.514	+	1.508	+	1.845	+	-
43	41	48	4.3	1.3	2.814	+	1.453	+	1.672	+	-
48	51	48	3.7	1.3	2.812	+	1.456	+	1.564	+	-
61	37	37	2.3	1.3	2.751	+	1.592	+	1.265	+	-
83	30	32	1.4	1.2	2.770	+	1.026	+	0.992	+	-

住院後-第12週肝功能及E肝之變化

如何解讀?

Due to his alcohol consumption shortly prior, indications for liver transplantation were not assessed. As ribavirin could not be used due to renal dysfunction (21), only conservative treatment was administered. However, his abnormal liver function tests gradually improved, although his HEV RNA was detectable by the highly sensitive nested reverse transcription-polymerase chain reaction with primers targeting the ORF2/ORF3 overlapping region (22) until day 32 after admission. The peak alanine aminotransferase (ALT) and total bilirubin levels were 1,067 IU/l and 26.3 mg/dl, respectively. He was ultimately discharged, and he left the hospital on foot on day 43

Hepatitis E＋alcoholic cirrhosis

Clinical course

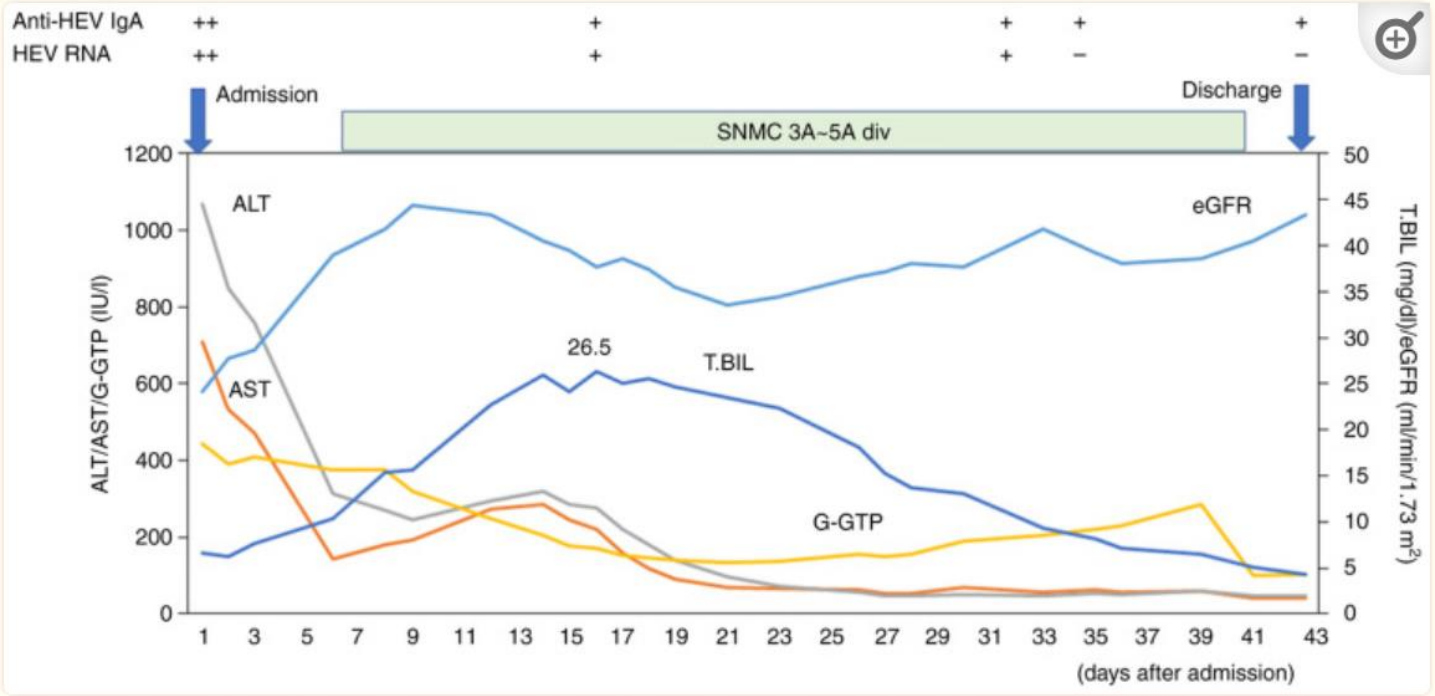


Figure 2

Clinical course of the patient in the present study. AST, aspartate aminotransferase; ALT, alanine aminotransferase; γ-GTP, γ-glutamyl transpeptidase; T. Bil, total bilirubin; eGFR, estimated glomerular filtration rate; anti-HEV IgA, anti-hepatitis E virus antibody immunoglobulin A; SNMC, Stronger Neo-Minophagen C with glycyrrhizin-containing preparation.

[1] Tatsuo Kanda et al **Severe hepatitis E virus genotype 3b in a patient with alcohol-associated liver disease: A case report** Med Int (Lond). 2024 Mar 7;4(3):22 (L1529).

Source of hepatitis E?

馬肉生魚片嘛

- 1. The peak ALT level was 1,067 IU/l in the present case. High ALT levels may provide an indication for the diagnosis of acute HEV infection (21,23). Barbosa *et al* (21) also used ribavirin in 3 patients; however, in the present study, ribavirin could not be used due to renal dysfunction. {pregnancy, severe anemia or renal dysfunction prohibit the use of ribavirin. pregnancy, }
- 2, HEV-infected patients with cirrhosis with or without HBV infection may develop ACLF, which is associated with a high mortality rate (~70%) (25-27).

- 25. Frias M, López-López P, Rivero A, Rivero-Juarez A. Role of hepatitis E virus infection in acute-on-chronic liver failure. *Biomed Res Int.* 2018;2018(9098535) doi: 10.1155/2018/9098535.
- 26. Choi JW, Son HJ, Lee SS, Jeon H, Cho JK, Kim HJ, Cha RR, Lee JM, Kim HJ, Jung WT, Lee OJ. Acute hepatitis E virus superinfection increases mortality in patients with cirrhosis. *BMC Infect Dis.* 2022;22(62) doi: 10.1186/s12879-022-07050-w.
- 27. Zhao H, Ye W, Yu X, Hu J, Zhang X, Yang M, Sheng J, Shi Y. Hepatitis E virus superinfection impairs long-term outcome in hospitalized patients with hepatitis B virus-related decompensated liver cirrhosis. *Ann Hepatol.* 2023;28(100878) doi: 10.1016/j.aohep.2022.100878.

- 3. The patient in the present study had consumed horse sashimi approximately 1 month prior to the onset of his symptoms, such as abdominal distention, loss of appetite, epigastric pain and dark urine (jaundice). It has been reported that anti-HEV IgG antibody and/or HEV RNA are positive in workhorses or horses in Egypt (28), China (29), the Netherlands (30), Bulgaria (31) and Germany (32). However, since whether horses play a role in the transmission of HEV remains unknown,

Hepatitis E in Taiwan

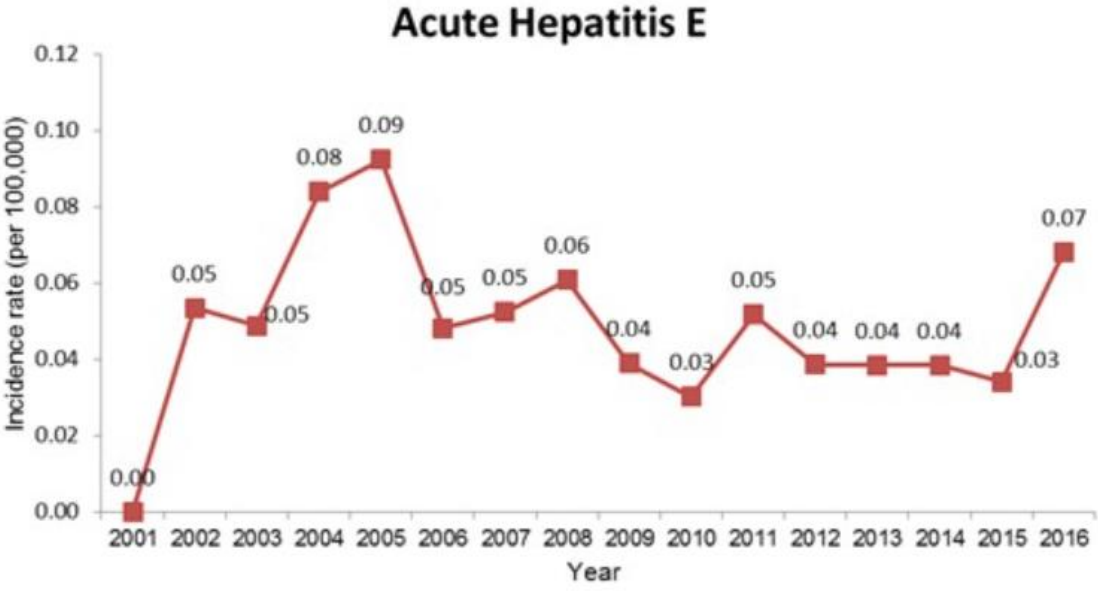


Figure5: Incidence Rate of Acute Hepatitis E in Taiwan (2001-2016)

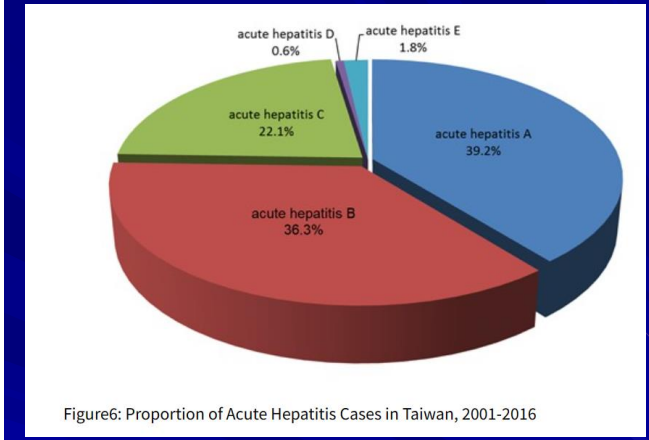


Figure6: Proportion of Acute Hepatitis Cases in Taiwan, 2001-2016

Acute Hepatitis - Taiwan Centers for Disease Control

衛生福利部疾病管制署
https://www.cdc.gov.tw/En/Category/ListContent/bg...

Accessed on 2024.04.30

1

Cite

Share

Hepatitis E virus infection in 6-month-old pigs in **Taiwan**.

Liao MH, Wu FT, Bai H, Doan YH, Yang JY, Takeda N, Muramatsu M, Li TC.

Sci Rep. 2020 Oct 9;10(1):16869. doi: 10.1038/s41598-020-74034-8.

PMID: 33037297 Free PMC article.

Hepatitis E virus (HEV) is the causative agent of acute **hepatitis E**. ...However, there is limited information on HEV infection status among pigs in **Taiwan**, especially pigs in the stage before transportation to the slaughterhouse. ...

2

Cite

Avian **hepatitis E** virus in chickens, **Taiwan**, 2013.

Hsu IW, Tsai HJ.

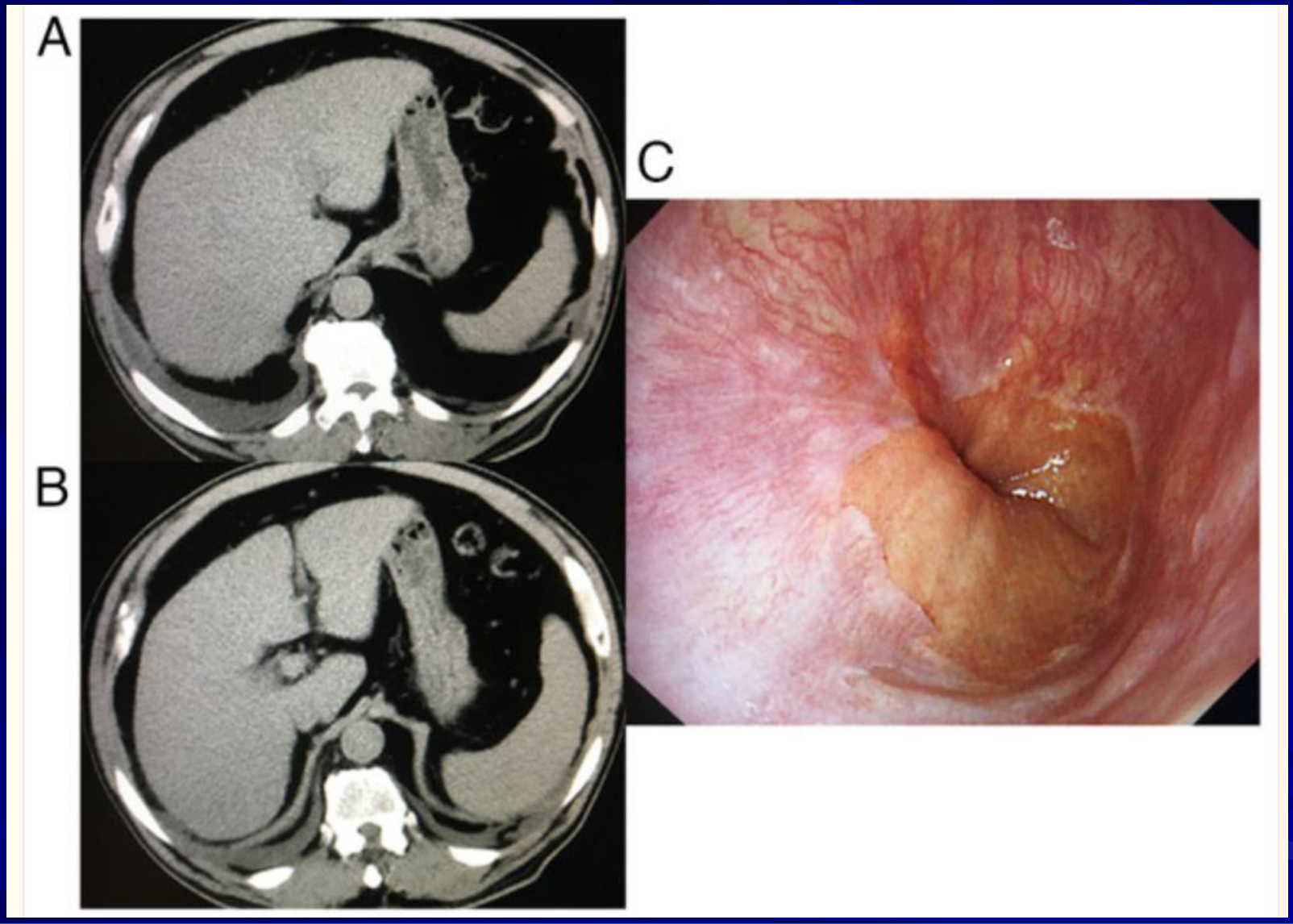
Emerg Infect Dis. 2014 Jan;20(1):149-51. doi: 10.3201/eid2001.131224.

PMID: 24378180 Free PMC article.

首次在臺灣大鼠中檢測到戊型肝炎病毒

- 臺灣流行病學簡報 **38, 2022**
- DOI： 10.6525/TEB.202201_38（2）.0001蔣派山， 黃偉倫 ， 鐘漢軒， Jyh-Yuan Yang， Hwa-Jen Teng*
- 2022 Vol.38 NO.2

戊型肝炎病毒（HEV）可分為四種（甲型、乙型、丙型和丁型）。這四個物種的寄主範圍是不同的。甲型戊型肝炎病毒可感染人類和其他哺乳動物，包括家豬、山羊和野豬、鹿和駱駝。其他 3 種物種 HEV-B、HEV-C 和 HEV-D 分別感染鳥類、嗇齒動物和蝙蝠物種。然而，最近有大鼠戊型肝炎病毒（戊型肝炎病毒C/基因型1，HEV-C1）感染人類的報導，其中包括一例在2019年患病前曾到訪臺灣的病例。為了調查 HEV-C1 是否在大鼠種群中傳播並導致臺灣人類感染，重新檢測了 50 份來自 HEV-A 陰性結果的 HEV 疑似患者的急性期人血清樣本，以進行 HEV-C 的回顧性審查。此外，從國際機場或港口捕獲的3隻Rattus tanezumi（以前稱為Rattus rattus）和47隻R. norvegicus中收集大鼠血清。通過半巢RT-PCR在人和大鼠血清樣本中鑒定 HEV-C RNA。還測試了大鼠血清的抗大鼠HEV抗體。在人類或 R. tanezumi 樣本中均未檢測到 HEV-C RNA，但在兩種 R. norvegicus 血清中鑒定出病毒 RNA。此外，2 種大鼠 HEV 菌株在 RNA 聚合酶基因中共用相同的部分序列。在血清學檢查中，在52%（26/50）的被困野生大鼠中檢測到抗HEV抗體。本研究記錄了臺灣首次發現 HEV-C1。本研究中觀察到的大鼠 HEV-C1 序列之間的血清陽性率和高度同源性可能是由於某些嗇齒動物種群中的病毒傳播造成的。臺灣原住民感染的風險不容忽視，因為已經在當地嗇齒動物種群中檢測到具有人畜共患潛力的HEV-C1。



Abdominal CT scan and upper gastrointestinal endoscopic images. (A and B) Abdominal CT scan indicating signs of liver cirrhosis with paraumbilical vein dilatation, mild splenomegaly and right pleural effusion. (C) An upper gastrointestinal endoscopic examination did not reveal any esophageal varices.

Case 2

- T he 24-year-old South-Asian female, primigravida, presented at **31 weeks of gestation with epigastric pain for 6 hours**. The pain was sharp, radiating to the back and was worse on lying supine. Pain was associated with fever, nausea, and vomiting for the same duration. The patient denied any history of recent infection, or of consuming any new medication or herbal drugs. The patient was only on iron and folic acid supplements during the course of her pregnancy. The patient was a nonsmoker and had never consumed alcohol. Her past medical, surgical, and family history were not significant. The patient was a housewife and comes from a middle-class family.

  Her obstetric follow-up was uneventful so far**.**

- **The patient received the first dose of the Pfizer vaccine 1 week prior to the onset of symptoms.** On general examination, the patient was conscious, oriented, and alert. Heart rate of 106 beats per minute, blood pressure of 140/90 mmHg, body temperature of 101˚F, respiratory rate of 22 breaths per minute, and oxygen saturation of 98% in room air were recorded. On abdominal examination, no obvious skin rashes were vis ualized in the abdomen. There was abdominal distension with gravid uterus and severe epigastric tenderness. The cardiovascular, respiratory, and neurological examinations were unremarkable.

- The initial blood investigations revealed a total leukocyte count of 17 × 109/L,
- serum lipase of 4376 U/L,
- serum amylase of 83 U/L.

要如何判斷?

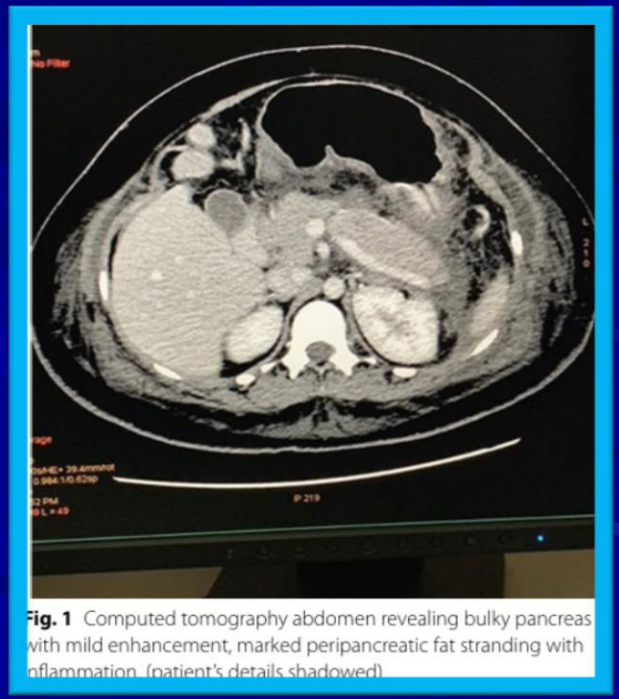
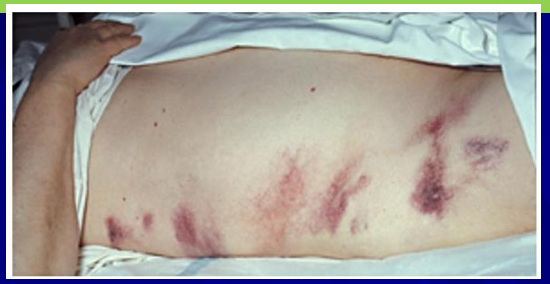


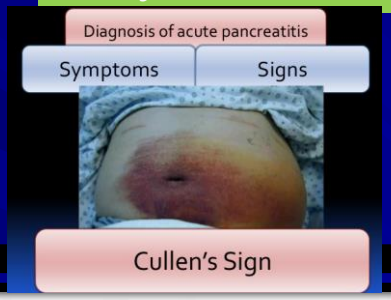
Fig. 1 Computed tomography abdomen revealing bulky pancreas with mild enhancement, marked peripancreatic fat stranding with inflammation. (patient's details shadowed)

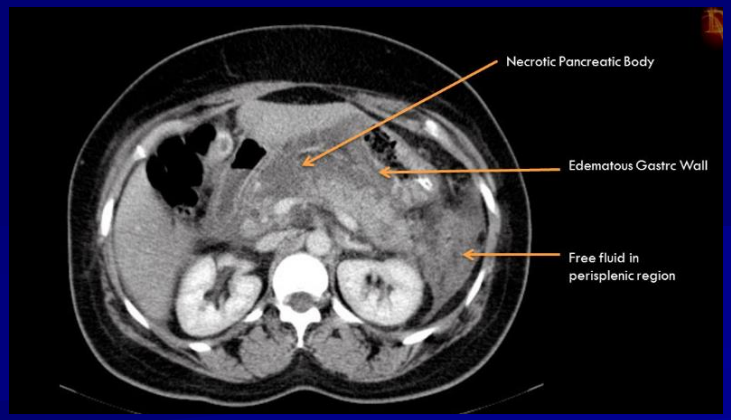
急性胰臟炎之診斷:.診斷依據

- 1, 診斷急性胰臟炎必需符合以下三個條件之二:
  - (1) 典型的上腹痛—向前灣屈較輕,躺平加重、會痛到背後
  - (2) **Serum amylase** 及 **lipase** 明顯升高, 即正常值之3倍以上.
    - **Amylase > 500 .**
    - **Lipase :>200**
  - (3) **Images : US, CT, MR**可見胰臟腫大,出水或有壞死或一般X光上出現片段性小腸腫大**(sentinel loop).**



Grey Turner's sign





Acute Pancreatitis - Diagnosis and Classification

- Diagnosis (two of three features)
  - Abdominal pain
  - Serum lipase or amylase ≥ 3 times upper limit of normal
  - Characteristic findings on imaging study (CT, MRI or ultrasound)
- Revised Atlanta Classification of Acute Pancreatitis (2012)*
  - **Categories**: interstitial edematous and necrotizing
  - **Mild**: no organ failure, no local or systemic complications, and generally resolves within 1 week
  - **Moderate**: transient organ failure, local complications, or exacerbation of co-morbid disease
  - **Severe**: persistent organ failure (>48 hours)
    - Mortality higher (~ 30%) in patients with severe AP
    - Severe disease accounts for ~15-25% of presentations

Course and treatment

- A real-time reverse transcriptase polymerase-chain-reaction (rRT-PCR) for SARS-CoV-2 screening was done prior to admission and was negative. On the second day of admission, **the patient had a spontaneous vaginal delivery and the baby was shifted to the neonatal ICU in a stable condition**. In view of ongoing upper abdomen pain, a computed tomography (CT) scan of the abdomen was performed, which revealed bulky pancreas with mild enhancement and marked peripancreatic fat stranding with
- inflammation, suggestive of acute interstitial edematous pancreatitis (Fig. 1). Over the next 48 hours, her pain settled and slowly oral intake was encouraged. Blood cultures taken at admission were sterile. The pancreatic enzymes on day 4 of admission were in the normal range. Her diet was changed to regular after the improvement of symptoms, and the patient was discharged in a stable condition on day 7. She was reviewed at 1 month and again after 6 months in the clinic and has remained stable since then.

Assessment parameters of pancreatitis

- Abdominal symptoms---(+)→(-) V
- Lipase: 4376 --→ normal V
- Calcium : unknown ?
- CRP: NA ?
- Amylase 83 (normal) useless

Etiology

- Covid 19 vaccination one week before symptom onset.
- Check reports about pancreatitis associated with pregnancy
- Check report of covid vaccination associated with acute pancreatitis

Etiology work up.

- 1. Detailed evaluation of all possible etiologies for pancreatitis did not reveal any specific etiology. Her liver func tion tests, calcium, and triglycerides were normal since admission (Table1). Her antinuclear antibody(ANA) was negative. Her ultrasound done at admission and a magnetic resonance cholangiopancreatography (MRCP) at 3 weeks did not show any gallstones.
- 2. Her pancreatic duct was normal and there was no abnormal pancreaticobiliary union.
- 3. Acute pancreatitis has been linked to various vaccines in the past including inactivated influenza, Mumps–Mea sles–Rubella (MMR), and Human Papilloma virus .
- 4. **A few cases of acute pancreatitis have also been reported after the Pfizer vaccine.**
- Prakash et al. reported a case of acute pancreatitis in a 96-year-old patient that devel oped a few days after administering the first dose of the Pfizer vaccine.
- Cieślewicz et al. published a report of acute pancreatitis in a healthy 29-year-old healthcare worker who developed symptoms 20 hours after getting the first dose Pfizer vaccine.
- Acute pancreatitis has also been reported in SARS-CoV-2 infection itself [de Sa TC et al , 2021)

1. Parkash O, Sharko A, Farooqi A, Ying GW, Sura P. Acute pancreatitis: a possible side effect of COVID-19 vaccine. Cureus. 2021;
2. Cieślewicz A, Dudek M, Krela-Kaźmierczak I, Jabłecka A, Lesiak M, Korzeniowska K. Pancreatic injury after COVID-19 vaccine—a case report. Vaccines. 2021;2021(9):576.
3. de Sá TC, Soares C, Rocha M. Acute pancreatitis and COVID-19: a literature review. World J Gastrointest Surg. 2021;13:574.

- The mechanism of vaccine-related pancreatic injury is still unclear. Bogdanos *et al.* [10] proposed the molecular mimicry theory, which states that amino acid sequence similarities between viral and self-antigens can result in an autoimmune reaction. Vojdani *et al.* [11] found that autoantibodies against SARS-CoV-2 spike protein and nucleoprotein show cross-reactivity against many human tissue antigens.
- Using the Naranjo scale, pancreatitis caused by the Pfizer vaccine in this patient scored 5 “probable” [12].
- The pregnancy was not assumed to be the cause of pancreatitis as an association between pregnancy and pancreatitis is very rare [13]. Eddy *et al.* [14] found that acute pancreatitis occurs in approximately 3 out of 10,000 pregnancies, while Sun *et al.* [15] demonstrated an increase in the incidence of preterm delivery in pregnant women with pancreatitis, as was seen in the present case report.

Acute pancreatitis associated with pregnancy ?

- 1. O'Heney JL, Barnett RE, MacSwan RM, Rasheed A. Acute and chronic pancreatitis in pregnancy. *Obstet Gynaecol.* 2021;**23**:89–93.
- 2. Eddy JJ, Gideonsen MD, Song JY, Grobman WA, O'Halloran P. Pancreatitis in pregnancy. *Obstet Gynecol.* 2008;**112**:1075–1081.
- 3. Sun L, Li W, Geng Y, Shen BO, Li J. Acute pancreatitis in pregnancy. *Acta Obstet Gynecol Scand.* 2011;**90**:671–676. doi: 10.1111/j.1600-0412.2011.01072.x.
- 4. Sudarshan Dash [1],et al .
- **Complications of Hypertriglyceridemia in Pregnancy and Its Impact on Neonates: a Hospital-Based Study From Odisha.**
- Cureus. 2022 Aug 25;14(8):e28399. *6/150 cases, dead.
- 5. Qiuxiang Xu [1], Sumei Wang [1], Zhenyu Zhang [2]
- **A 23-year, single-center, retrospective analysis of 36 cases of acute pancreatitis in pregnancy** Int J Gynaecol Obstet. 2015 Aug;130(2):123-6.
- 36 cases/no maternal death, severe in Hypertriglyceridemia

1. Genetic Factors **Associated** With Adverse **Pregnancy** Outcomes in Chronic **Pancreatitis**.
Wu D, Clin Transl Gastroenterol. 2024 Apr 1;15(4):e00691

2. When **pregnancy-associated** hypertriglyceridemia goes above and beyond the risk of **pancreatitis**.
Barac.Intern Emerg Med. 2024 Mar;19(2):477-481. doi: 10.1007/s11739-023-03378-6. Epub 2023 Jul 19.PMID: 37468772 No abstract available.

3. Dynamic nomogram for predicting infected pancreatic necrosis in female patients of childbearing age with hypertriglyceridemia-induced **acute pancreatitis**.
H.Dig Liver Dis. 2024 Feb;56(2):297-304. doi: 10.1016/j.dld.2023.07.034. Epub 2023 Aug 14.PMID: 37586905

4. EMS Diabetic Protocols For Treat and Release.
Schwerin DL, Svancarek B.2023 Jul 17. In: StatPearls [Internet]. Treasure Island (FL): StatPearls
Gestational Diabetes Mellitus (GDM) Hyperglycemia first detected during **pregnancy** is classified as gestational diabetes mellitus (GDM). Although it can occur anytime during **pregnancy**, GDM generally affects pregnant women during the second and third trimesters. Accor …

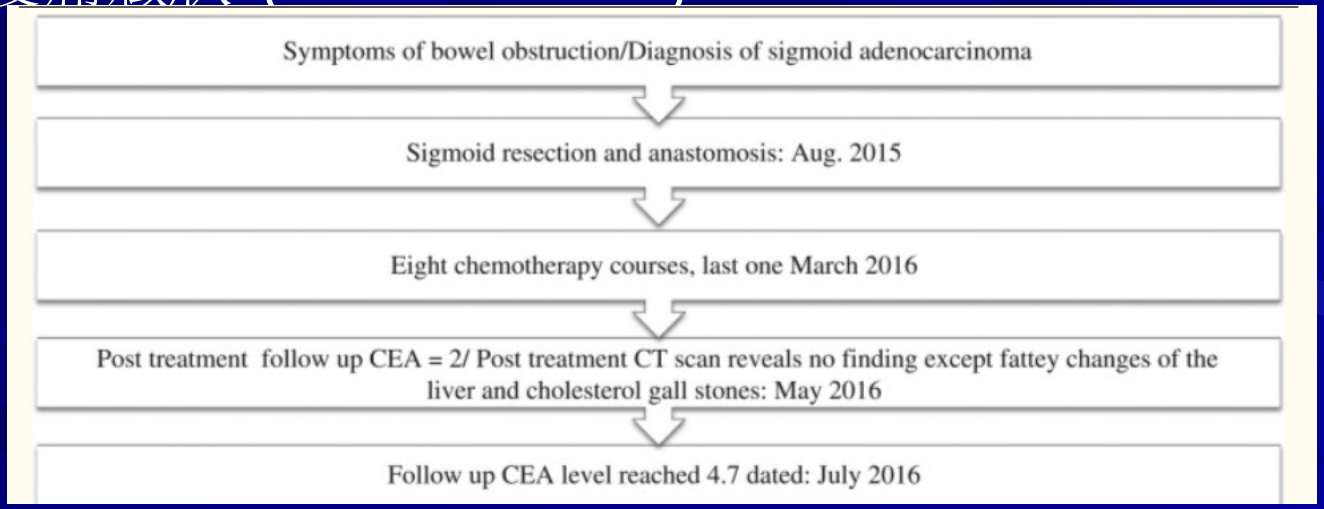
5. Challenges in the management of hypercalcemia in **pregnancy** - Case report of two cases.
Lim SH, Lim W, Thain SPT.Case Rep Womens Health. 2024 Feb 4;41:e00586. doi: 10.1016/j.crwh.2024.e00586. eCollection 2024 Mar.PMID: 38356696 **Free PMC article.**
It is **associated** with maternal complications such as urolithiasis, **pancreatitis**, renal insufficiency and preeclampsia, fetal complications such as growth restriction and intrauterine fetal demise, and neonatal complications such as neonatal hypocalcemia, tetany and …

Source of information

- Rajib Kumar Dey [1], Hemamala Ilango [2], Subash Bhatta [3], et al
- **Acute pancreatitis in pregnancy following COVID-19 vaccine: a case report** (馬爾地夫) Med Case Rep. 2022 Sep 29;16(1):354.

Case 3, elevated CEA during follow up of resected CRC.

- 一名 53 歲男性，有繼發於乙狀結腸腫塊的大腸梗阻病史，約 12 個月前接受了緊急乙狀結腸切除術和結腸吻合術。組織病理學檢查顯示乙狀結腸腺癌。因此，他接受了八個療程的輔助化療。治療結束時腫瘤標誌物在正常範圍內。監測期間，癌性抗原（CEA）水準略有升高，並伴有新的上腹痛癥狀. (CEA:2.0-→4.7)



CRC 手術後接受化療8個月療程. 2016 ,05 CEA : 2.0, 2016.07 增加為4.7, 你要怎麼辦?

Elevated CEA→1. cancer recurrence
2. false positive
3. Laboratory error

如何進行? 確定CEA增加的原因?

CEA: abnormal
in cancer patients

- 728 cases of CRC
  Memorial Sloan Kettering who underwent resection for stage I, II, or III colorectal cancer between 2003 and 2012, and who had an increase in CEA level above the normal after a normal perioperative CEA level.
- 358 had a **false-positive** elevation of CEA level,
- 335 had a true-positive elevation indicative of **recurrent** [colorectal cancer],
- 35 had a true-positive elevation indicative of the development of a **new**, [non–colorectal cancer] malignancy,"

- **1. no evidence of cancer on either imaging studies** or other diagnostic procedures,
- **2. follow –up** of (1) **at least 1 year** since the first abnormal CEA or
- (2) abnormal CEA elevations followed by **spontaneous normalization,**
- **(3)** with at least **2 consecutive subsequent normal CEA** measurements in the absence of a therapeutic intervention,

- @@@ 247 patients with 2 or more confirmed false-positive CEA level elevations, only 5 (2%) had measurements greater than 15 ng/mL, and no confirmed elevation greater than 35 ng/mL was a false-positive,
- FALSE (+) 很少 >15,
- False (+) 不會超過 35

Same conclusion in 1977, Special lecture
False (+) often around 5-10, rarely > 15,
IT was definitely abnormal when CEA>20

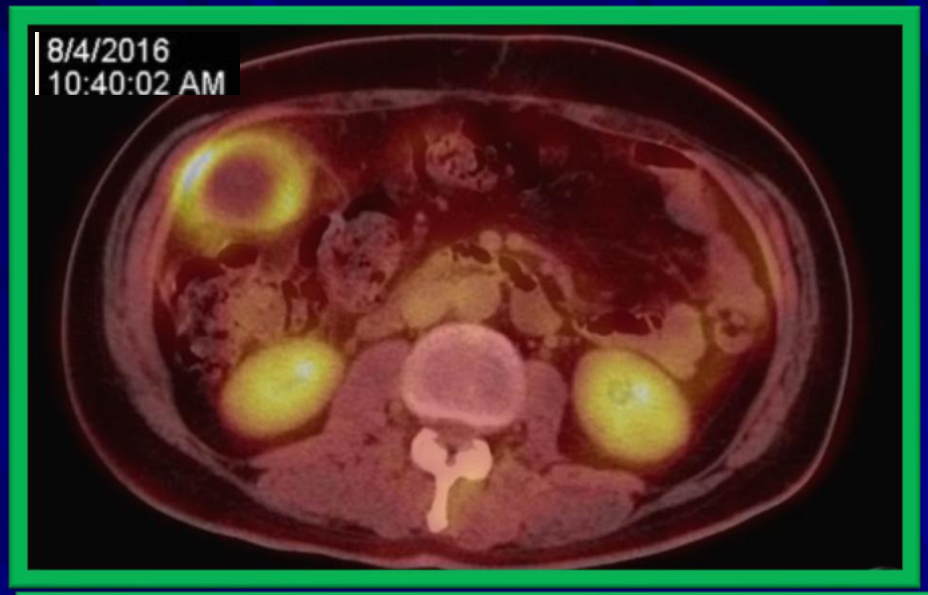
Elevated CEA→1. cancer recurrence
2. false positive
3. Laboratory error

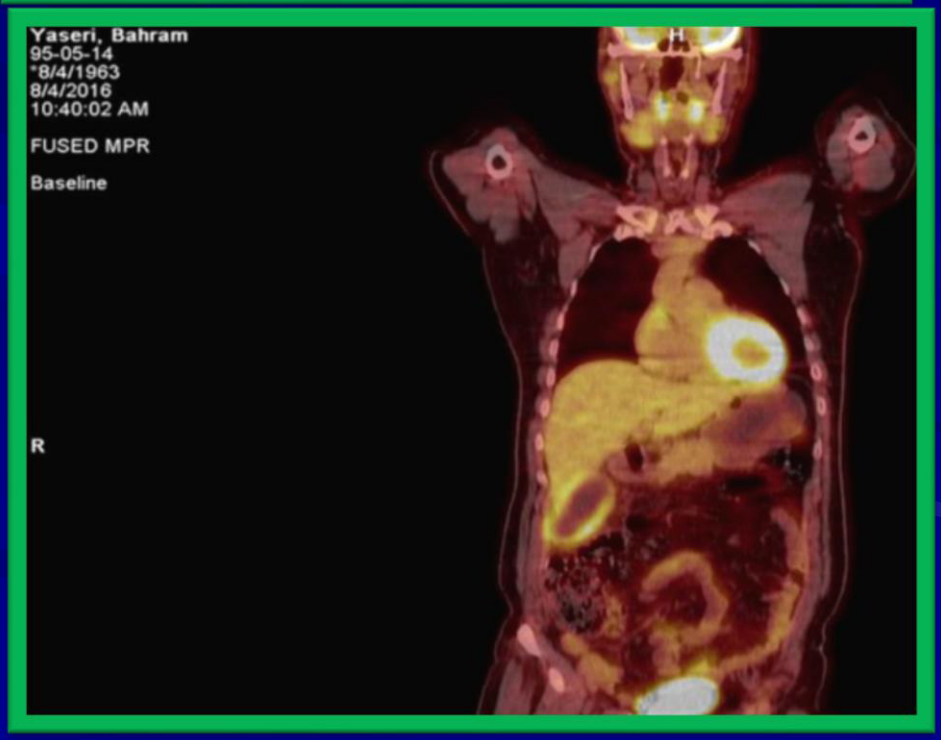
如何進行? 確定CEA增加的原因?

- Recheck CEA(RIA)
- Colonoscopy check.
- Abdominal CT for metastases
- PET scan
- Recheck history on f smoking or exposure to second smoker
- Wait and see.

PETCT

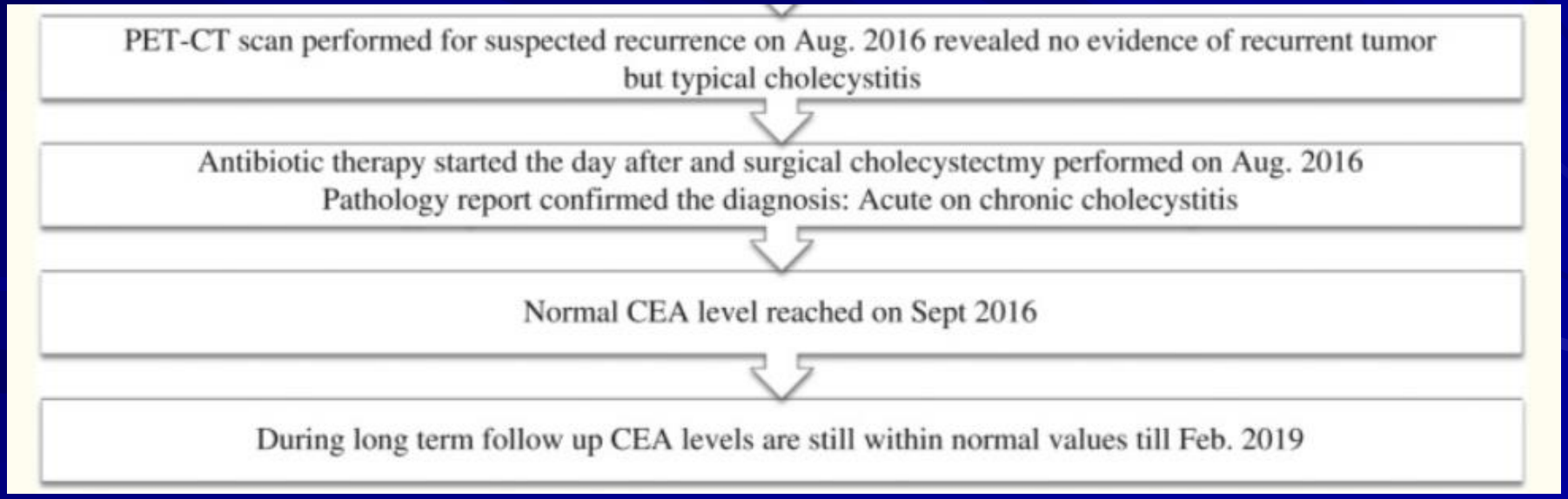
- FDG PET-CT with (a) trans-axial and (b) coronal views. PET-CT scan performed to evaluate a patient with colon cancer interfacing mild rise in blood CEA level. One hour after the intravenous administration of 11 mCi of ^{18} F-FDG, PET-CT images were obtained. A strikingly enlarged gallbladder shows intense radiotracer uptake in the gallbladder wall with a central photopenic area representing a rim like pattern. Metabolically active wall proposes the possibility of cholecystitis (acute/chronic).





Elevated CEA→1. cancer recurrence
2. false positive
3. Laboratory error

如何進行? 確定CEA增加的原因?



Increased serum CEA in biliary diseases.

- Increased serum CEA level, secondary to biliary tract diseases, such as obstruction, inflammation and epithelial malignancies of bile ducts has been already reported [1-3]. Elevated tumor markers, either CEA or $CA_{19\text{-}9}$, are not specific in gallbladder malignancies and have both been identified in xanthogranulomatous cholecystitis (XGC) as well as gallbladder cancer (GC) [1].
- Also extrahepatic biliary tract obstruction including cholangitis and common bile duct (CBD) stone are accused to cause high CEA values, even more than cholecystitis and gallbladder stone. Serum CEA level returned to normal after relief of the obstruction [2].

1. Suzuki H, Wada S, Araki K, Kubo N, Watanabe A, Tsukagoshi M, et al.. Xanthogranulomatous cholecystitis: difficulty in differentiating from gallbladder cancer. *World J Gastroenterol* 2015;21:10166–73.

2. Lurie BB, Loewenstein MS, Zamcheck N. Elevated carcinoembryonic antigen levels and biliary tract obstruction. *JAMA* 1975;233:326–30.

3. Sawada S, Shimada Y, Sekine S, Shibuya K, Yoshioka I, Matsui K, et al.. Expression of GLUT-1 and GLUT-3 in xanthogranulomatous cholecystitis induced a positive result on ^{18} F-FDG PET: report of a case. *Int Surg* 2013;98:372–8.

Case 4

History of present illness

The patient presented with cough that had started 1 wk earlier; he was treated with azithromycin (500 mg, qd, i.v.) for a common cold at a private clinic. His symptoms were alleviated after treatment. However, 2 d later, the patient experienced chest pain radiating to the right arm while walking in a cold wind. He was evaluated at another hospital, where **his troponin T level was found to be 34.34 pg/mL** (normal range: 3-14 pg/mL). He also exhibited increased white blood cell and platelet counts and an increased neutrophil percentage. His ECG was normal. Echocardiography showed that the left ventricular ejection fraction (LVEF) was 59%, with mild regional abnormalities in the ventricular and apical septa and a dilated left atrium. The patient's troponin T was 685.30 pg/mL and **the creatine kinase isozyme (CK-MB) level was 11.85 ng/**mL (normal range: 0.1-4.94 ng/mL) after 8 h. Based on these findings, the patient was admitted to our hospital.

- A 42-year-old man presented with cough that started 1 wk earlier and chest pain that begun 2 d earlier.

- Relatively young man.
- URI history one week before
- Sudden onset of chest pain-2 days ago
- ECG: normal.
- Initial troponin T level was found to be 34.34 pg/mL at A hospital
- Follow up troponin T was 685.30 pg/mL after 8 h at B hospital
- creatine kinase isozyme (CK-MB) level was 11.85 ng/mL (normal range: 0.1-4.94 ng/mL)

是不是A MI, 下一步要怎麼辦？

Chest X-ray
WBC, CRP, neutrophil counting
Follow up ECG 3 hours later
Echocardiogram
Coronary angiogram

The patient’s initial symptom was chest pain; biomarkers such as TnI and CK-MB were elevated and the ECG were abnormal. :ST-segment elevation and T-wave inversion

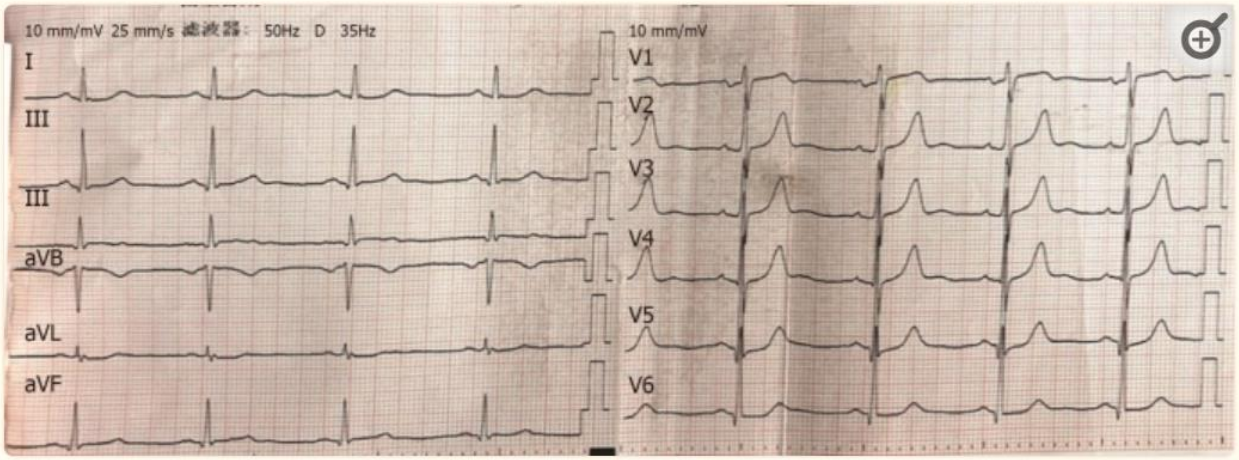


Figure 1

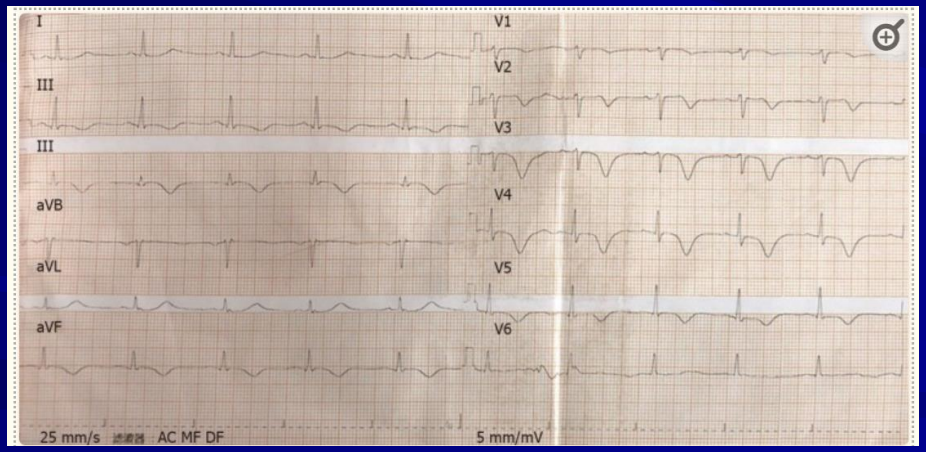
Electrocardiogram showing V2-5 T-wave towering after the chest pain.

The patient had a 2-year history of hypertension and a smoking history for 20 years. Personal and family history : No specific personal or family history of disease was recorded.

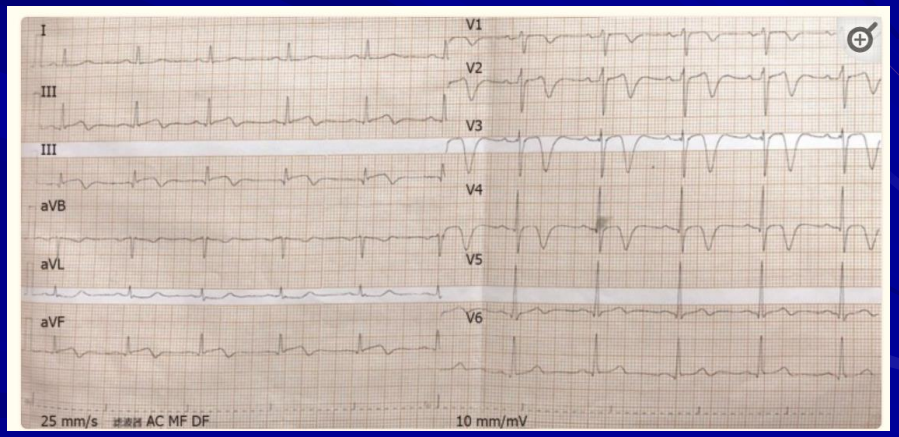
Physical examination upon admission : The patient's temperature was 37.8 ˚C, his body mass was 78 kg, his blood pressure was 164/114 mmHg, and there were no positive signs on a physical examination.

- Laboratory testing showed the following on admission:
- serum TnI, 2.57 ng/mL (normal range: 0-0.034 ng/mL);
- hypersensitive C-reactive protein, 4.95 mg/L (normal range: 0-2.87 mg/L);
- erythrocyte sedimentation rate, 45 mm/h;
- brain natriuretic peptide, 168 pg/mL;
- white blood cell count, 13.30 $\times 10^9$/L; neutrophil percentage, 0.735; and platelet count, 312 $\times 10^9$/L.
- His **liver function**, kidney function, thyroid function, D-dimer, electrolytes, blood lipids, and blood glucose levels showed no significant abnormalities;
- rubella virus immunoglobulin G (IgG) and immunoglobulin M (IgM) antibody levels were elevated on admission.
- **The patient's chest radiograph and D-dimer level were normal, thus excluding pulmonary embolism.**

- The patient's ECG (Figure (Figure2)2) showed II, III, and aVF T-wave inversions, and a wide range of precordial segment T-wave inversions that exhibited dynamic changes compared with the previous ECG. Electrocardiography (Figure (Figure3)3) revealed the abovementioned segment T-wave inversion after 20 h.

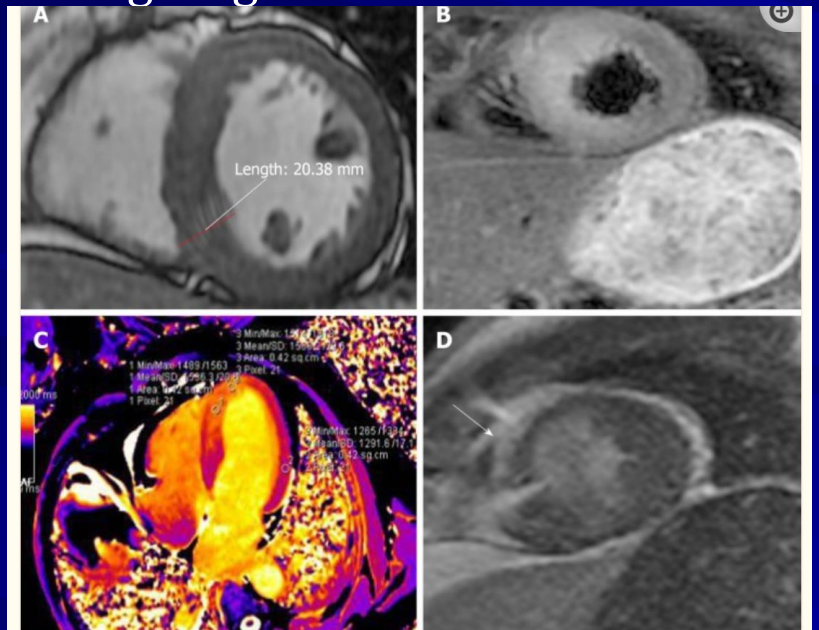


ECG on admission： II, III, and avF T-wave inversion and V2-5 T-wave inversion after admission



ECG 20 hours after admission: T wave inversion

- Coronary angiography was performed on day 5 after admission. There was **no obvious coronary stenosis** during the operation. Cine CMR imaging showed wall motion abnormalities (mid septal, apical septal, and apical anterior), and the anterior walls were obviously thicker than the normal walls (Figure (Figure4A),4A), with the thickest part of the ventricular wall being approximately 20 mm. Fat suppressed T2-weighted imaging showed the edema of the mid septal, apical septal, and apical anterior walls (Figure (Figure4B).4B). We observed the myocardial edema more specifically on T1 mapping (Figure (Figure4C).4C). The endocardium and middle myocardium of the mid septal and apical septal walls showed patchy late gadolinium enhancement (LGE) (Figure (Figure4D).4D). The high signal area was smaller than the area of edema.



A: wall motion abnormalities (mid septal, apical septal, and apical anterior), and the anterior walls were obviously thicker than the normal walls
B: Fat suppressed T2-weighted imaging showed edema
C: myocardial edema more specifically on T1 mapping
D: The endocardium and middle myocardium of the mid septal and apical septal walls showed patchy late gadolinium enhancement (LGE)

Final diagnosis

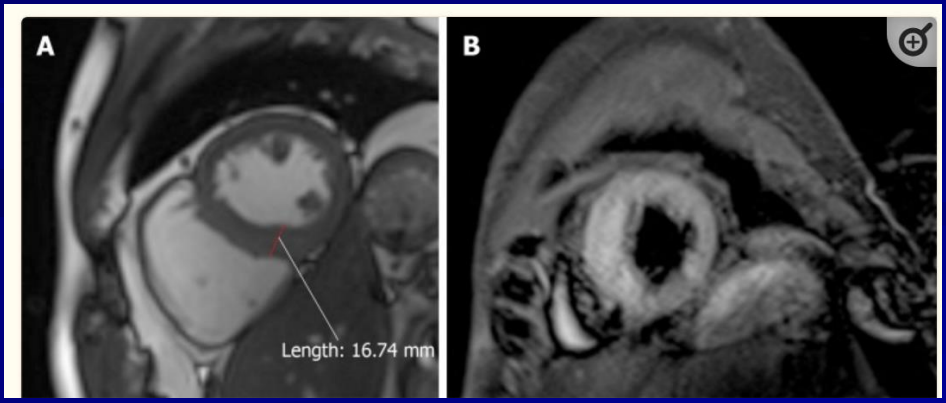
- viral myocarditis,
- Not AMI

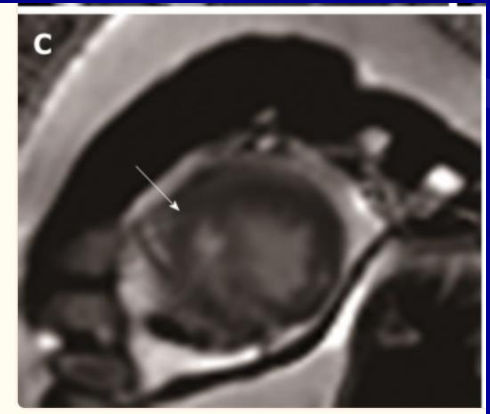
Treatment :

He was given aspirin (100 mg, qd, p.o.) and clopidogrel (75 mg, qd, p.o.) for 6 d before the CMR examination. After the CMR examination, the patient was treated using acyclovir (250 mg, q12h, i.v.) combined with levofloxacin (500 mg, qd, i.v.) for 11 d. The patient was treated with trimetazidine and creatine phosphate for 19 d during hospitalization.

Outcome: CRP and TnI : normal

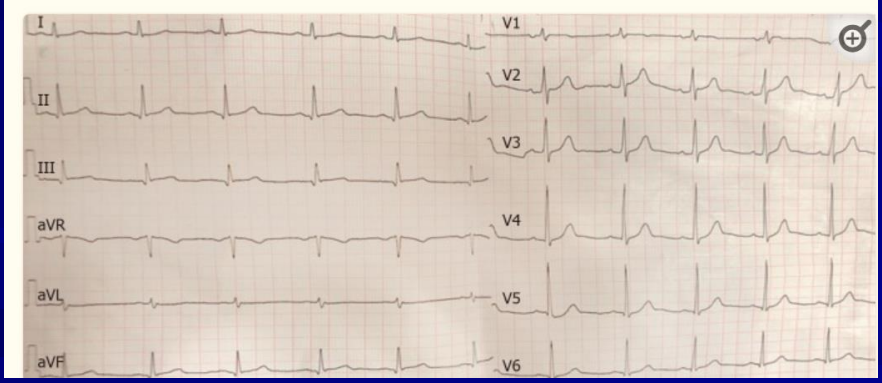
- The patient had no more chest pain and his temperature returned to normal, as did his C-reactive protein and TnI (0.027) levels; his rubella virus-specific IgG and IgM antibody levels were slightly lower than before. A review of the CMR images showed that the edema observed in the mid septal, apical septal, and apical anterior walls was less extensive than before on T2WI; the thickest part of the ventricular wall was approximately 17 mm (Figure 5A and B) and the anterior and interior enhancement moved to the middle myocardium, confirming the diagnosis of myocarditis (Figure 5C}

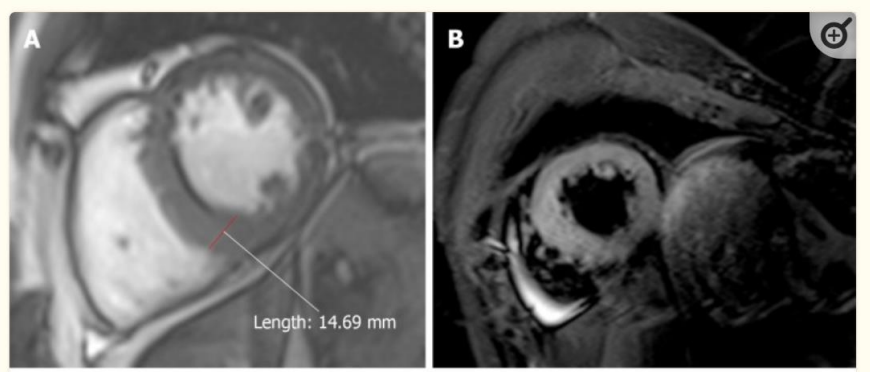




Follow up(up to 13 months)

- The patient's condition improved and he was discharged.
- Ten months later, the patient was asymptomatic and the ECG was normal (Figure 6). Ultrasound echocardiography showed normal wall motion but patchy enhancement of the ventricular septal wall.
- At his 13-mo follow-up examination, the patient remained asymptomatic; CMR showed that the edema had disappeared and the thickest part of the ventricular wall was approximately 14 mm (Figure 7A and B). LGE was distributed in the myocardium, indicating fibrosis.





- 1. Myocarditis that mimics myocardial infarction presents with elevated serum TnI, TnT, and myocardial enzyme (*e.g.*, CK-MB) levels, and electrocardiograph may reveal ST-segment elevation or depression, and/or Q- and T-wave inversions. The coronary angiogram is usually normal.
- 2. In a study that showed the patients with an initial clinical presentation of acute myocardial infarction with normal coronary angiograms, almost 77% were suspected of having myocarditis according to the imaging data.
- Sarda L, Colin P, Boccara F, Daou D, Lebtahi R, Faraggi M, Nguyen C, Cohen A, Slama MS, Steg PG, Le Guludec D. Myocarditis in patients
- with clinical presentation of myocardial infarction and normal coronary angiograms. *J Am Coll Cardiol.* 2001;37:786–792
- 3. In addition to coronary angiography, myocarditis and myocardial infarction can be identified by CMR. T1 mapping provided the best diagnostic parameters in patients who had been diagnosed with acute myocarditis during 2 wk.
- 4. Endomyocardial biopsy (EMB) is the gold standard for diagnosing myocarditis, its invasiveness and low sensitivity limit its clinical application.

Conclusion

- Myocarditis may have mimicked myocardial infarction. For a middle-aged man with myocardial infarction, the possibility of acute myocardial infarction should be ruled out first.
- It is also important to consider myocarditis, such that early CMR has a strategic role in the differential diagnosis. Traditional heart failure medications and antiviral treatments are effective for myocarditis.

Source of information

- 1. Ya-Min Hou, Peng-Xi Han, Xia Wu et al :Myocarditis presenting as typical acute myocardial infarction: A case report and review of the literature
- World J Clin Cases. 2020 Jan 26; 8(2): 415 – 424.
- Published online 2020 Jan 26. doi: 10.12998/wjcc.v8.i2.415

2. Cardiovascular magnetic resonance (CMR) has become the primary tool for non-invasive assessment of myocardial inflammation in patients with suspected myocarditis.

. Friedrich MG, Sechtem U, Schulz-Menger J, et al International Consensus Group on Cardiovascular Magnetic Resonance in Myocarditis. Cardiovascular magnetic resonance in myocarditis: A JACC White Paper. *J Am Coll Cardiol.* 2009;53:1475–1487.

Cardiovascular magnetic resonance (CMR) has become the primary tool for noninvasive assessment of myocardial inflammation in patients with suspected myocarditis. The International Consensus Group on CMR Diagnosis of Myocarditis was founded in 2006 to achieve consensus among CMR experts and develop recommendations on the current state-of-the-art use of CMR for myocarditis. The recommendations include indications for CMR in patients with suspected myocarditis, CMR protocol standards, terminology for reporting CMR findings, and diagnostic CMR criteria for myocarditis (i.e., "Lake Louise Criteria").心血管磁共振（CMR）已成為疑似心肌炎患者無創評估心肌炎症的主要工具。CMR心肌炎診斷國際共識小組成立於2006年，旨在CMR專家達成共識，並就目前最先進的CMR治療心肌炎提出建議。這些建議包括疑似心肌炎患者的 CMR 適應證、CMR 方案標準、報告 CMR 結果的術語以及心肌炎的診斷性 CMR 標準（即“Lake Louise 標準”）。

Case 5, Thromboembolic phenomenon.

- DIC
- D-dimer
- AMI
- Stroke,
- Thrombosis of coronary arteries—causing VT and AMI

Hemogram

- Complete blood count (CBC) showed :
- white blood cell count of **79,050/µL**
- **95 % blasts.**
- hemoglobin :11.2 g/ dL
- Platelets : < 6,000

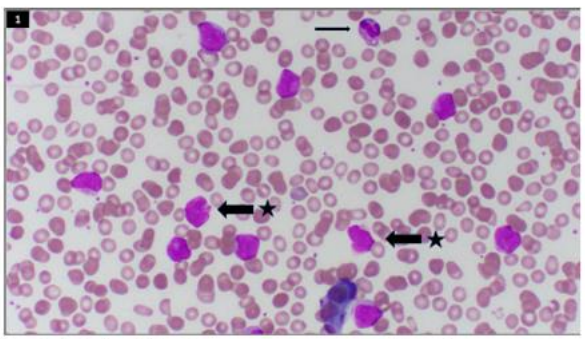


Fig. 1 Peripheral blood smear demonstrating blasts (arrows with star) and segmented neutrophil (arrow)

一名 59 歲的女性，既往無明顯病史，因嚴重疲勞、厭食和不適 2 周到primary care doctor就診，伴有寒戰、盜汗和肌痛。

Hemogram 要怎樣interpretation分析

- 一名 59 歲的女性，
- 既往無明顯病史，因嚴重疲勞、厭食和不適 2 周到primary care doctor就診她否認有任何惡性腫瘤或心臟病家族史。
- 在尿路感染治療失敗后，她第二次到初級保健醫生就診。

- 表現為：體溫 37.3 ˚C，血壓 121/74 mmHg，心率 117 次/分，呼吸頻率 18 次/分，血氧飽和度（SpO2） 92%。體格檢查顯示臉色蒼白，右腳底呼吸音減弱，左小腿壓痛無腫脹。

- 白血球細胞計數為 79,050/μL，血紅蛋白為 11.2 g/dL，血小板< 6000/μL，手動分類顯示 95% 的原始細胞胸部、腹部和盆腔計算機斷層掃描 （CT） 發現脾臟、雙側腎臟、右肺和肝臟存在右節段性肺栓塞和血栓性梗死

Bone marrow aspirate demonstrated AML by morphology (86.7%) and flow cytometry. Marrow was hypercellular (> 95%) with reduced trilineage hematopoiesis, consistent with AML

- 凝血酶原時間 19.7
- 部分凝血活酶時間 partial thromboplastin time35 秒、
- 纖維蛋白原 133 mg/dL
- D-二聚體 41.4 μg/mL

。

如一般認為APTT延長、纖維蛋白原減少、血小板計數減少，加上臨床上有出血傾向，如傷口、針孔不斷滲血或四肢指端末梢出現血栓現象，即符合DIC的定義

- 實驗室分析還顯示彌散性血管內凝血病（DIC）

泛發性血管內血液凝固症(DIC，Disseminated Intravascular Coagulation) 是醫療上的急症，其致病原因相當複雜，且死亡率非常高，對醫師而言是一種極具挑戰性的病症。 在加護病房或手術室中DIC並不少見。 但往往因延遲診斷或治療而導致組織缺氧及多種器官衰竭的嚴重後果。

DIC臨床表徵的特色與致病機轉相同，即微小管血栓與異常出血傾向。以各器官的血栓現象來講，中樞神經系統的表徵為譫妄及昏迷；肺部則出現成人呼吸窘迫症候群(ARDS)；腎臟方面為少尿、BUN上升、腎皮質壞死；腸胃道急性潰瘍；皮膚出現指端壞疽。以出血特異性而言，會有腦出血、紫斑、瘀青、抽血針孔出血、血尿、流鼻血、牙齦出血、上消化道大量出血等現象。

AML 治療

- The patient was admitted to the intensive care unit, where she was treated for AML with hydroxyurea 2 g twice daily, all-*trans* retinoic acid until acute promyelocytic leukemia was ruled out, prednisone 1 mg/kg for prevention of differentiation syndrome, allopurinol 300 mg daily, intravenous fluids, and transfusions to maintain hemoglobin > 7 M/μL, platelet > 30,000/μL, and cryoprecipitate to maintain fibrinogen > 150 mg/dL.
- She was also given fresh frozen plasma to maintain international normalized ratio (INR) < 1.5, and a heparin infusion was initiated once platelet count exceeded 30,000/μL.

Course and treatment

- On hospital day 2, the patient developed acute hypoxia with complaints of dyspnea and chest pain. She was tachypneic with respiratory rate of 27 breaths per minute, and telemetry demonstrated sinus tachycardia at 117 beats per minute . Spontaneous hemodynamically significant ventricular tachycardia (VT) occurred and self-terminated After resolution of her VT, an electrocardiogram (ECG) identified the presence of sinus tachycardia with anterolateral ST elevation consistent with acute anterolateral injury (Fig. .4). Soon after, the patient developed acute dysarthria and nonreactive pupils. In the setting of her stroke evaluation, the patient developed recurrent VT with pulseless electrical activity (PEA) (Fig. .5). Resuscitative measures were initiated. However, due to inability to restore spontaneous circulation after 15 minutes of resuscitative efforts, the patient died secondary to presumed cerebrovascular and coronary thromboses causing stroke and anterolateral infarct complicated by VT and PEA .

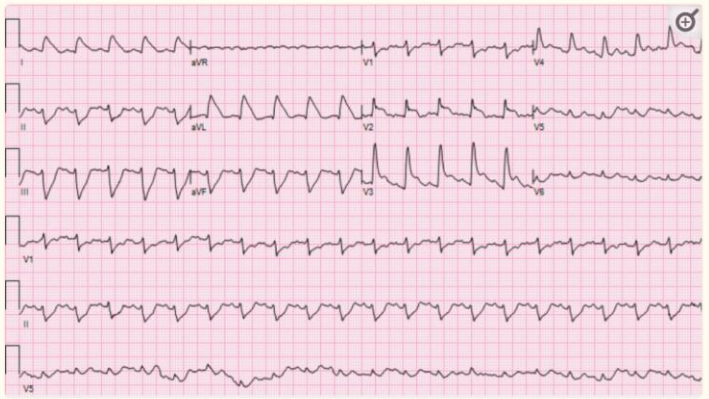


Fig. 4

ECG with acute anterolateral ST elevation myocardial injury at 07:48

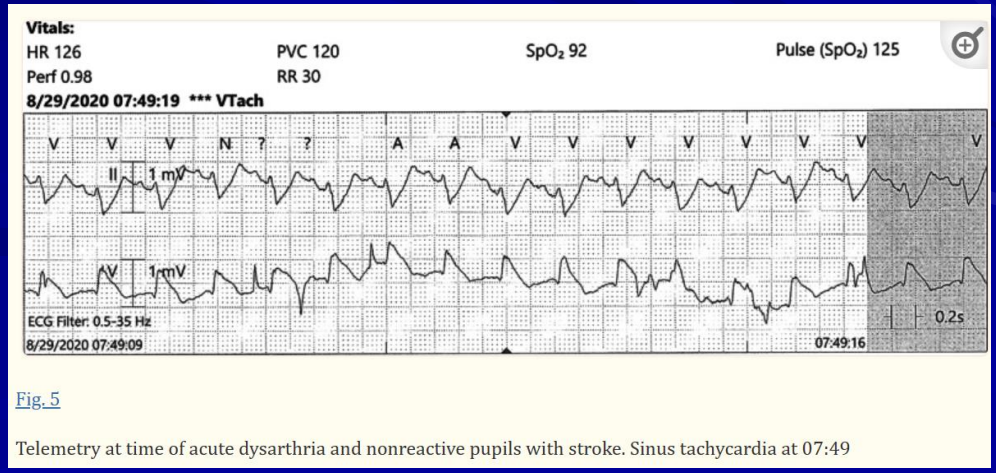


Fig. 5

Telemetry at time of acute dysarthria and nonreactive pupils with stroke. Sinus tachycardia at 07:49

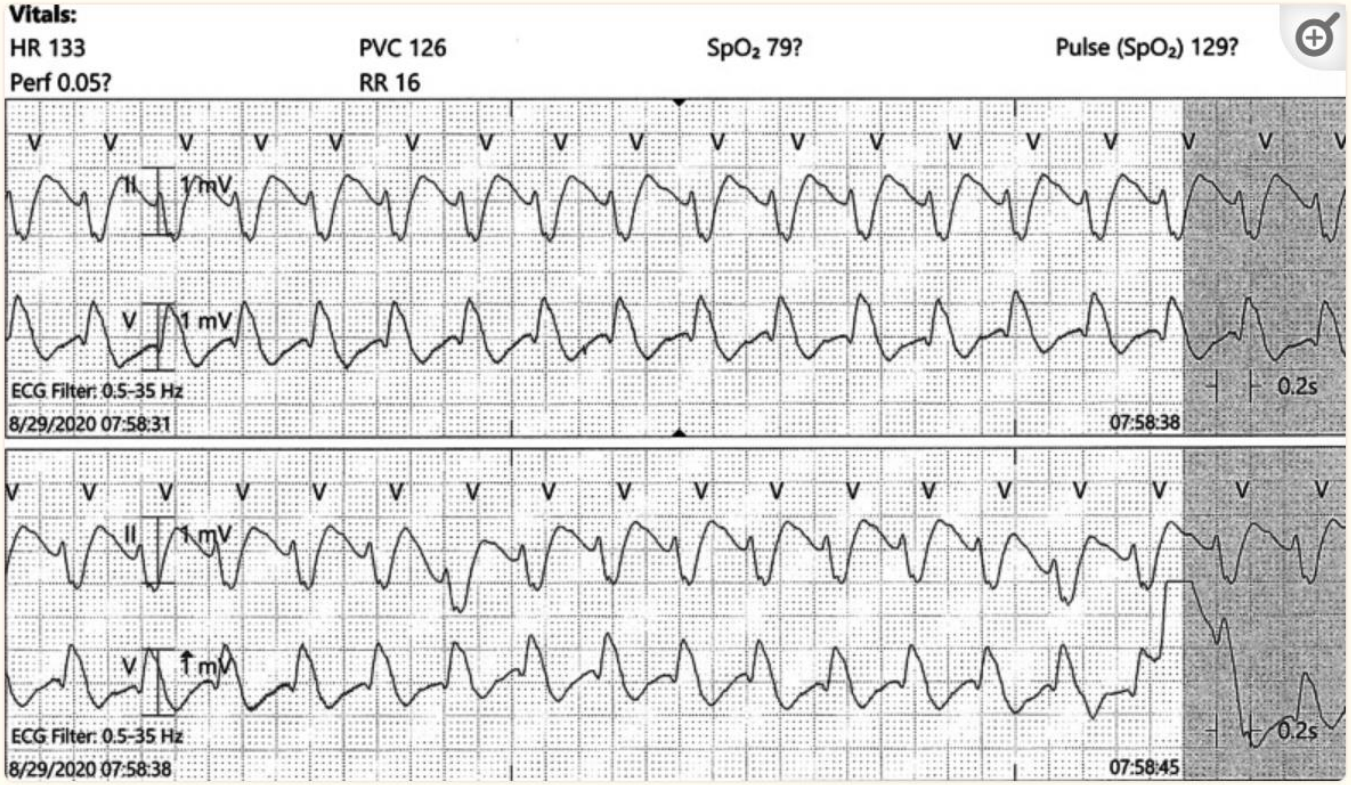


Fig. 6

Telemetry with recurrent ventricular tachycardia at onset of pulseless electrical activity arrest

Final diagnosis :

1. AML,
2. DIC
3. cerebrovascular and coronary thromboses
4. stroke and anterolateral myocardial infarct complicated by VT and PEA
5. Recurrent ventricular tachycardia

- Source of information.
- Acute myeloid leukemia causing acute thrombosis of the coronary arteries: a case report.Ferrel MN, Ryan JJ, Han *Meganne N Ferrel*[1], *John J Ryan*[2], *Frederick T Han*[3]
- J Med Case Rep. 2022 Apr 12;16(1):149. doi: 10.1186/s13256-022-03280-3.

Assessment parameters
代表治療反應好或不好

Improvement　轉好

- 1. CRP　減少
- 2. WBC　return to normal
-
- 3. Amylase return to normal
- 4. Hb: 增加/貧血改善,
- 　出血停止

Downhill效果不好

- 1.CRP 增加
- 2.Leukocytosis.
  Leukemoid reaction
  WBC增加
- 3. relapse of pancreatitis
- 4. 減少, 貧血加重或繼續出血

很好的 assessment parameters

怎樣選assessment parameters

- 對疾病深入瞭解,
- 善於利用各種 lab data
- 發病日(期間,staging) 會影響改變
- Normal or abnormal.
- Individual variation個別差異/治療之影響
- 挑本**案例差異(不正常)**最明顯的項目.

那個檢查最能看出疾病之進行

- Presenting data –
- Tumor markers– CEA,
- UC :Inflammatory parameters– ESR,CRP, and WBC.
- Hypoglycemia—sugar, symptoms.
- Hypokalemia –K, ECG
- Acute necrotizing pancreatitis :Ca. CRP and lipase

仍要考慮檢驗錯誤或 false positive
Ex. CEA

- 1. CEA – 5~ 10 --→smoker ? Or other conditions
- 2. 檢驗方法也會影響結果
- EIA ---data 穩定性不夠,
- RIA : 比較可靠
- 3. 增加 -→ Lab error ?，Cancer 更厲害,
- 未控制住(recurrent)，吸菸故態復萌----
- 4. > 20: 可能已有 metastases.
- 5. 不同醫院不同的 **Lab.** 不宜直接比較
- 但仍可以參考.

好好思考、紀錄、處理

- 跟 Present illness 的關係
- 1. Diagnostic evidence
- 2. Indicates severity
- 3. Indicates therapeutic response better or worse.
- 4. **Associated diseases.**
- 5. Treatment regimen. Change or modify.
- 6 Express the opinion at recording of the lab data

41. Electrolyte abnormalities
hypokalemia,

Serum K : <3.2 m Eq/L.

Severe hypokalemia : <2.5 mEq/L.

內科學誌 2010：21：31-39

低血鉀的診斷與治療

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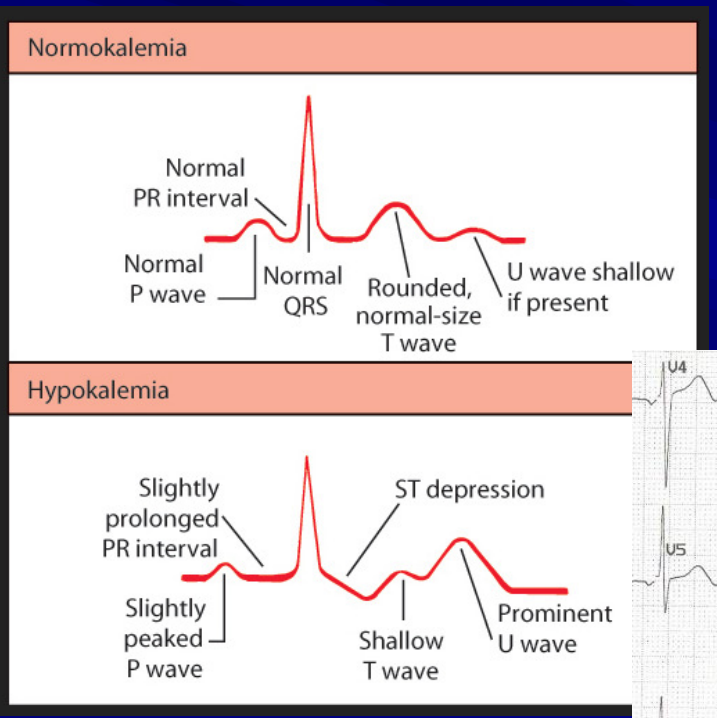
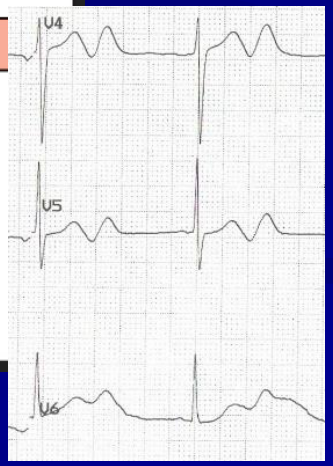




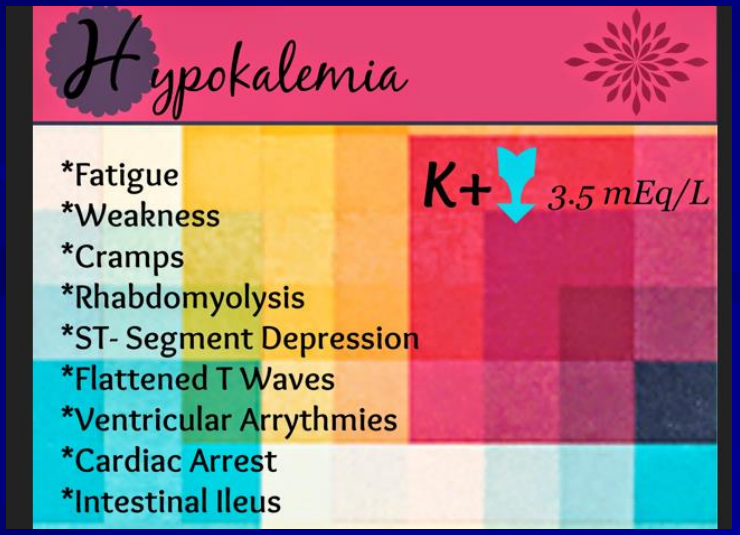
低血鉀可能會增加心臟血管疾病患者的罹病率及死亡率，導致 心律不整 (cardiac arrhythmias)、肌肉癱瘓甚至呼吸停止，因此如何快速的診斷與治療低血鉀 是本篇探討的主題。其病因可以依尿液鉀離子的排泄量 (potassium (K+) excretion)、血中酸鹼 值、血壓高低、血漿腎素活性(renin activity)、醛固酮 (aldosterone)濃度、皮質類固醇(cortisol) 濃度，及尿液中鈉離子與氯離子的濃度而進一步鑑別診斷。低尿液鉀離子排泄可為鉀離子急 性往細胞內移動，腸道鉀離子流失或之前腎臟過度排泄所引起。而高尿液鉀離子排泄則通常 合併有酸鹼異常。低血鉀的治療，主要取決於低血鉀的程度、造成低血鉀的快慢、臨床症狀、 潛在病因及其他相關的潛在危險因子。

hypokalemia

表三：低血鉀的治療

1. Medical emergency	Cardiac arrhythmia, respiratory insufficiency
2. Avoid risks of K^+ shift into cells	Do not give glucose, insulin and $NaHCO_3$
3. Magnitude of K^+ deficit	Large vs. small does of K^+
4. Route of K^+ administration	Central, peripheral or oral
5. K^+ preparations	KCl vs. $KHCO_3$ (K^+ citrate) vs. K^+ phosphate
6. Adjuncts to therapy	K^+-sparing agents, ACEI, AIIA
7. Associated settings	HPP, chronic hyponatremia, hypomagnesemia, volume depletion, severe metabolic acidosis, low muscle mass

ACEI: angiotensinogen converting enzyme inhibitor, AIIA: angiotensin II antagonist, HPP: hypokalemic periodic paralysis.



一、K^+ 補充量：在病人沒有 K^+ 往細胞內移動的狀況下，K^+ 濃度由 4 mmol/L 下降到 3 mmol/L 需要流失 350 mmol，如果降到 2 mmol/L 則需流失 750 mmol 的 K^{+1}。

二、K^+ 補充途徑：如果聽不見腸音，口服補充 K^+ 則不可行；周邊靜脈補充濃度不可超過 40 mmol/L；補充速度除非在緊急狀況下，否則不可超過 60 mmol/hour。

What are the causes of liver cell necrosis?

- Hepatitis
- Heart failure
- Drugs
- Fatty liver
- Alcohol and or other liver toxins
- Marijuana (cannabis) smoking
- leptospirosis
- ---

recognised causes of acute liver cell necrosis:

The following are recognised causes of acute liver cell necrosis:
(i) paracetamol overdose
(ii) severe heart failure
(iii) human papilloma virus
(iv) leptospirosis
(v) cannabis smoking

Sonia, 2008.

Thyroid function tests

- 國內目前常用檢查有下列五項：
- [T3 (RIA), (EIA), (CLIA)],
- [T4 (RIA), (EIA), (CLIA)],
- [FT4 (RIA, analogue method), (RIA, two-step method)], [FT4I (T4×T3 uptake)],
- [**TSH** (IRMA), (ICMA)]。

真正表現甲狀腺功能高低的是FT3及FT4。
FT4只佔 total T4的 0.03%。FT3只佔 total T3的 0.3%。
改用TSH and/or FT4 (RIA)
Sensitive TSH:
正常值約 0.4-5 μu/ml，而甲狀腺高能症應低於0.1μu/ml，TSH對FT4之變化有明顯放大反 應 (amplification)各人 TSH 對 FT4之set point 並不相同，在所謂『正常 範圍』內之FT4，有人 TSH正常，有人 TSH 已超出正常值，

Hyperthyroidism診斷

- 1.臨床上懷疑是hyperthyroidism，檢查 TSH+FT4，並保留血清，如果兩項都正常， 就排除 hyperthyroidism 之診斷，
- 如果 TSH 低，FT4 高，確定診斷為 primary hyperthyroidism；
- 如果只有 TSH 低，FT4正常，則加做T3(原來保存之血清)，如 果T3高，是T3 toxicosis。
- 如果光是TSH低而FT4及T3 正常，則是 subclinical hyperthyroidism；必要時可加做 TRH test 來確認

Hypothyroidism 之診斷

- 臨床上懷疑是hypothyroidism，檢查TSH+FT4，如果正常，就排除診斷，
- 如果 TSH高，T4 低，就是primary hypothyroidism；
- 如果是 TSH 高，而FT4正常， 則是subclinical hypothyroidism，
- 至於 hypothalamic/pituitary hypothyroidism，則是TSH低，FT4低，但少數TSH正常甚至高，是由於腦垂分泌不具生物活性 之TSH 所致，這時應對病人之症狀及其他腦垂功能一起做檢查評估。

T4 supplement therapy

- T4 supplement therapy,甲狀腺低能症接受甲狀腺素補充治療，劑量之監測，原則上TSH 單項就可以， 目標當然是TSH在正常值內。如果TSH異常，每次調整劑量後，需6至8週， 再測 TSH，直到TSH正常或接近正常為止。

Hyperthyroidism under antithyroid treatment

- antithyroid drug 治療早期(至少 2-3 個月內)要評估治療效果，**應使用FT4**，因 為此時期 hypothalamic - pituitary - thyroid axis 受先前 high T4 抑制，即使治療 後甲狀腺功能正常或過低，TSH 也不能立即恢復正常或升高。藥物治療一段時 間(2-3 個月以上)，檢查TSH+ FT4，**如果FT4降低而TSH升高表示藥物過量，** 此時可視情況(主要甲狀腺大小)來決定抗甲狀腺藥物劑量及是否添加甲狀腺 素，目標是使TSH及FT4在正常值內。

摘要(2024.05.10)

- 1. 一般常用的檢驗項目要熟悉 Normal ranges, clinical significance.思考不正常所代表的意義.與主要疾病有無關連.
- 2. 很多檢驗數據因Lab. 品質不良, Lab error大,會影響判斷
- 3. Lab data 可作疾病變動的指標(信號), 必須參考症狀及徵象之變動.
- 4. 要好好紀錄在病歷之上, 而且至少要在一週內Follow up.(主要變化時要 QD or Q2D)