LOGO

Case Presentation

Intern：劉人銘 2008/10/30

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Identifying Data

- Patient's Name: 林鄭月霞
- ID: 8173682
- Age: 75 year-old
- Gender: Female
- Occupation: housewife
- Admission Date: '08/10/24 ~

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Chief Complaint

- Progressive shortness of breath in these two weeks

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Present Illness

- CKD for about 10 years, type 2 DM was diagnosed for 6 years medication with Glucobat(50mg) 1# BID
- ESRD for 1.5 years, refused dialysis

Follow at our OPD
Elevated Creatinine was noted

97/10 ~ She suffered from progressive shortness of breath, orthopnea, hardly sleep, leg edema and decrease in urine output in these two weeks.

97/10/24 **due to severe SOB, ESRD with uremic status was impressed, she was admitted for hemodialysis and further management**

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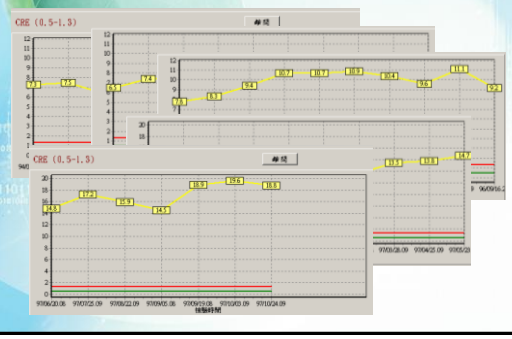

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Past History

- Allergy History: denied
- Past Medical History
  - Hypertension for years
  - chronic renal disease, stage 5, for 1.5 years
  - type 2 diabetes mellitus for 6 years under OHA control with Glucobay(50mg) 1# BID
- Social and Personal History
  - Smoking: denied
  - Drinking: denied
  - Betel nut: denied
  - Marital status: married
  - Occupation: housewife
- Family History: unremarkable

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Review of systems:

- **General :**
  - Weakness(+) Fatigue(+) Anorexia(+)
  - Fever(-)
- **Head** Dizziness(+)
- **Respiratory :** Wheezing(+)
- **Cardiovascular**
  - Dyspnea(+)
  - Leg edema(+):
  - Chest distress (+)

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Physical Examination

- **Height 159(cm) / weight 51(kg),**
- **General appearance: acute-ill appearance**
- **Mental status (Alert)**
- **Vital signs (T36.4 P92 R24 , BP162/100)**
- **Oxgenation status (N/S with O2 3 l/min)**
- **HEENT:**
  - **Neck: estimated JVP 15 cm H2O at 30 degree**
- **Chest: normal expansion**
  - **Auscultation: Wheezing(+) Rales(+)**
- **Extremities: Pitting edema(grade 1)**

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Laboratory data-10/24

項目名稱	報告值	單位	備註
R.B.C (4000-11000)	6680	/mm^3	
R.B.C (400-500)	241	X10^4/	
Hb. (12-16)	6.7	gm/dl	
Ht. (36-46)	20.0	%	
MCV (84-100)	83.0	fl	
MCH (28-32)	27.8	Pg	
MCHC (32-36)	33.5	g/dl	
Platelet (150000-487000		/mm^3	

項目名稱	報告值	單位	備註
Seg (40-75)	92.4Rechecked	%	H
Lymho (20-45)	5.1	%	
Mono (2-10)	2.1	%	
Eos (1-6)	0.2	%	
Bas (0-1)	0.2	%	
Pro-Time (8.0-12.0	10.7	"	
Pro-time INR	1.08		
A.P.T.T. (25.4-36.3	33.4	"	

項目名稱	報告值	單位	備註
K (3.0-5.0)	4.2	mmol/l	
CRE (0.5-1.3)	18.8	mg/dl	H
ALB (3.5-5.1)	3.8	g/dl	

項目名稱	報告值	單位	備註
BUN (6-22)	174	mg/dl	H
NA (138-146)	130	mmol/l	L
ALT(GPT) (3-37)	22	IU/l	
AST(GOT) (13-38)	23	IU/l	
P (2.5-4.7)	10.0	mg/dl	H
TG (15-190)	109	mg/dl	
CHO (110-200)	122	mg/dl	
UA (2.1-7.1)	8.4	mg/dl	H
CA (8.4-10.4)	8.5	mg/dl	
Mg (Magnesium) (1.	3.3	mg/dl	H
C.R.P (< 0.4)	3.01	mg/dl	

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項目名稱	報告值	單位	備註
Ferritin (5-148)	54.1	ng/ml	
TIBC (250-450)	261	ug/dl	
Fe (70-200)	16	ug/dl	L
Intact PTH (12-65)	728	pg/ml	H

備註：未標示尿液種類

項目名稱	報告值	單位	備註
CRE-U	68.7	mg/dl	
TP-U	247.2	mg/dl	

247.2/68.7=3.6 g/day

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ABG:

- PH: 7.16 ↓
- PCO2: 25 ↓
- PO2:87
- HCO3-: 8.9 ↓
- T.CO2:18.2
- SO2:93%
- primary: metabolic acidosis

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項目名稱	報告值	單位	備註
Color	yellow		
Appearance	slight turbid		
Glucose	+		
Bilirubin (-)	-		
Ketones	Trace		
Sp.Gr.	1.013		
Occult Blood	+		
pH	6.0		
Protein	+++		
Urobilinogen (0.1-0.1		EU/dl	
Nitrite	-		
Leukocyte esterase	+		
RBC	3-4	/EPF	
WBC	25-30	/EPF	.
Ep.cell	0-2	/EPF	.
Bacteria	+/EPF		

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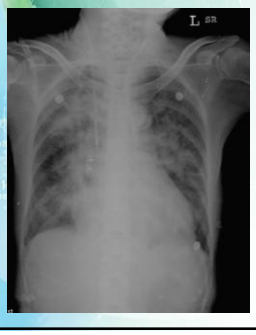
Image - CXR



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Impression

- **Impression 1**
  - ESRD with uremic status (severe anemia, platelet dysfunction, metabolic acidosis, hyperphosphatemia and secondary hyperparathyroidism)
  - Plan to do
    - check CBC/DC, PT, aPTT, BUN/Cre, Na/K/Ca/P/Mg,ABG
    - on double lumen for emergent HD
    - BT with PRBC 2U during HD*2 days
    - CaCO3 500mg TID for hyperphosphatemia
    - Rolikan for metabolic acidosis
    - consult CVS for AVF

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Impression

- **Impression 2**
  - CXR revealed pulmonary edema
  - Plan to do
    - lasix(20mg) 3amp
    - f/u CXR and observation
- **Impression 3**
  - elevated CRP and U/A revealed pyuria and bacteria, suspected **UTI**
  - Plan to do
    - U/C and empiric antibiotic used

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Impression

- **Impression 4**
  - **HTN**
  - Plan to do
    - medication with Norvasc(5mg) 1# QD
    - check BP Q6H
- **Impression 5**
  - **type 2 DM**
  - Plan to do
    - NovoNorm(1mg) 1# TID
    - sugar one touch QID

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platelet dysfunction in uremia

UPTODATE

(platelet dysfunction in uremia)

(Congenital and acquired disorders of platelet function)

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INTRODUCTION

- This impairment is multifactorial and includes defects intrinsic to the platelet as well as abnormal platelet-endothelial interaction. Uremic toxins and anemia also play a role.
- Clinical bleeding in uremia is typically cutaneous, including easy bruising and mucosal bleeding. Less frequent is epistaxis, gingival bleeding, or hematuria.

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PATHOGENESIS

- The pathogenesis of uremic bleeding has been attributed to a number of factors, most prominently platelet dysfunction. This dysfunction is due to both decreased platelet aggregation as well as impaired platelet adhesiveness. Contributing factors are both intrinsic to platelets as well as external.

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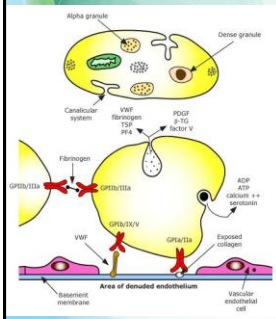

- NORMAL PLATELET FUNCTION
  - 1) adherence :via GP receptor
  - 2) activation
    - Alpha granule
    - Dense granule: ADP.serotonin
  - 3) aggregation
  - 4) interaction with coagulation factor

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PATHOGENESIS - Intrinsic factors

- **Intrinsic factors** include
  - **abnormal expression of platelet glycoproteins,**
  - **altered release of ADP and serotonin,**
  - **faulty arachidonic acid and depressed prostaglandin metabolism,**
  - **decreased platelet thromboxane A2 generation, and**
  - **abnormal platelet cytoskeletal assembly.**

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PATHOGENESIS - Extrinsic factors

- **Extrinsic factors** include
  - **the action of uremic toxins,**
  - **anemia,**
  - **increased nitric oxide (NO) production,**
  - **von Willebrand factor abnormalities,**
  - **decreased platelet production,**
  - **abnormal interactions between the platelet and the endothelium of the vessel wall.**

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Uremic toxins ★

- often beneficial effect of acute dialysis on platelet dysfunction
- Urea is probably not the major platelet toxin
- The adverse effect of uremic plasma on platelet function **cannot** be replicated by adding urea, guanidinoacetic acid, or creatinine to the plasma.
- high levels of guanidinosuccinic acid and methylguanidine have resulted in abnormal platelet function, likely through stimulation of NO production

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Anemia ★

- Anemia is a common finding in chronic renal failure and is due primarily to decreased renal erythropoietin production.
- Correction of anemia with blood transfusions or erythropoietin often improves platelet function
- rheologic factors - At a hematocrit above 30 percent, the red cells primarily occupy the center of the vessel, while the platelets are in a skimming layer at the endothelial surface. This close proximity allows the platelets to adhere to the endothelium and then form a platelet plug when there is endothelial injury.

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Nitric oxide ★

- NO is an inhibitor of platelet aggregation that is produced by endothelial cells and platelets.
- The increase in NO synthesis may be due to elevated levels of guanidinosuccinic acid, a uremic toxin that may be a precursor for nitric oxide in this setting
- the administration of an NO synthesis inhibitor normalizes the bleeding time in uremic rats

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TREATMENT

- No specific therapy is required in asymptomatic patients.
- correction of platelet dysfunction is desirable in patients who are actively bleeding or who are about to undergo a surgical procedure
- Correction of anemia
- Desmopressin (dDAVP)
- Estrogen
- Cryoprecipitate

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Correction of anemia

- Raising the hematocrit to above 25 to 30 percent will reduce the bleeding time in many patients
- The elevation in hematocrit can be achieved acutely by red cell transfusions or chronically via the administration of recombinant human erythropoietin
- **Erythropoietin**
  - Correction of anemia
  - increasing the number of GPIIb/IIIa molecules on the platelet membrane,
  - improving the defect in thrombin-induced phosphorylation of platelet proteins
  - improving platelet calcium signaling

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Desmopressin (dDAVP)

- simplest and least toxic acute treatment for platelet dysfunction
- an analog of antidiuretic hormone with little vasopressor activity
- increasing the release of factor VIII:von Willebrand factor multimers from endothelial storage sites
- at a dose of 0.3 microg/kg given in 50 mL of saline over 15 to 30 minutes.

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Estrogen

- The mechanism perhaps due to decreased generation of NO
- the administration of conjugated estrogens (0.6 mg/kg intravenously, daily for five days, 2.5 to 25 mg of Premarin orally, or 50 to 100 microg of transdermal estradiol twice weekly)
- The effect of estrogens is dose-dependent

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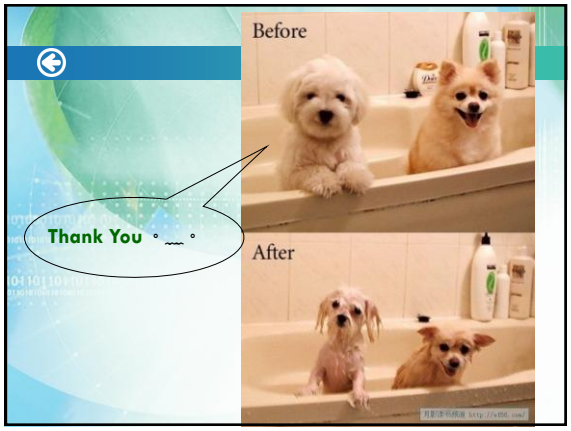
Cryoprecipitate

- **每單位約含大於45國際單位的第八凝血因子，125mg纖維蛋白原及存在於原單位約40~70%的von-Willebrand因子及20~30%的第十三因子。**
- **適應症：**
  - A型血友病(factorVIII缺少)。
  - von-Willebrand氏病。
  - 第十三因子及纖維蛋白原缺乏症。
  - 偶用於控制尿毒症病人之出血。
- **10 units intravenously every 12 to 24 hours**


Before
After
Thank You 。﹏。