

一、The *Chicago Tribune* (芝加哥論壇報) reported “pharmacies miss half of dangerous drug combinations” using the example of combination of clarithromycin and simvastatin. Please provide the possible mechanism of this drug-drug interaction, the potential adverse effect and how we can do to prevent this happening. (8%)

二、請列出下列藥品之劑型、適用於何種毒物（或藥品）中毒時之解毒劑，及其解毒機制： [請另填寫於「試卷」本] (16%)

序號	解毒劑			
	名稱	劑型	中毒病因 (毒物或藥品)	作用機轉
1	acetylcysteine			
2	deferoxamine			
3	dimercaprol			
4	edetate calcium disodium			
5	flumazenil			
6	naloxone			
7	physostigmine			
8	phytonadione			
9	pralidoxime chloride			
10	protamine			

接背面

三、試說明下列各藥品之藥理分類及用途：

[請另填寫於「試卷」本]

(28%)

序號	藥名	藥理分類	用途
1.	amantadine		
2.	aripiprazole		
3.	bortezomib		
4.	chloral hydrate		
5.	chlorpromazine		
6.	clarithromycin		
7.	clozapine		
8.	Co-trimoxazole		
9.	dexmedetomidine		
10.	domperidone		
11.	dronedarone		
12.	escitalopram		
13.	famotidine		
14.	foscarnet		
15.	fosphenytoin		
16.	granisetron		
17.	imipramine		
18.	itraconazole		
19.	levofloxacin		
20.	metronidazole		
21.	mifepristone		
22.	pentamidine		
23.	risperidone		
24.	ritonavir		
25.	sunitinib		
26.	tacrolimus		
27.	tamoxifen		
28.	venlafaxine		

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四、Read the following abstract carefully and answer questions I ~ IV below.

Epidemiology of Acute Kidney Injury in Critically Ill Children and Young Adults

Background

The epidemiologic characteristics of children and young adults with acute kidney injury have been described in single-center and retrospective studies. We conducted a multinational, prospective study involving patients admitted to pediatric intensive care units to define the incremental risk of death and complications associated with severe acute kidney injury.

Methods

We used the Kidney Disease: Improving Global Outcomes criteria to define acute kidney injury. Severe acute kidney injury was defined as stage 2 or 3 acute kidney injury (plasma creatinine level ≥ 2 times the baseline level or urine output < 0.5 ml per kilogram of body weight per hour for ≥ 12 hours) and was assessed for the first 7 days of intensive care. All patients 3 months to 25 years of age who were admitted to 1 of 32 participating units were screened during 3 consecutive months. The primary outcome was 28-day mortality.

Results

A total of 4683 patients were evaluated; acute kidney injury developed in 1261 patients (26.9%; 95% confidence interval [CI], 25.6 to 28.2), and severe acute kidney injury developed in 543 patients (11.6%; 95% CI, 10.7 to 12.5). Severe acute kidney injury conferred an increased risk of death by day 28 after adjustment for 16 covariates (adjusted odds ratio, 1.77; 95% CI, 1.17 to 2.68); death occurred in 60 of the 543 patients (11.0%) with severe acute kidney injury versus 105 of the 4140 patients (2.5%) without severe acute kidney injury ($P < 0.001$). Severe acute kidney injury was associated with increased use of mechanical ventilation and renal-replacement therapy. A stepwise increase in 28-day mortality was associated with worsening severity of acute kidney injury ($P < 0.001$ by log-rank test). Assessment of acute kidney injury according to the plasma creatinine level alone failed to identify acute kidney injury in 67.2% of the patients with low urine output.

Conclusions

Acute kidney injury is common and is associated with poor outcomes, including increased mortality, among critically ill children and young adults.

From: *N Engl J Med* 2017;376:11–20.

- (一) This article applied the Kidney Disease: Improving Global Outcomes criteria to define acute kidney injury (AKI). Another criteria, RIFLE, is also commonly used. Explain what the acronym, RIFLE, stands for and their definitions. (10%)
- (二) Authors in this article found “assessment of acute kidney injury according to the plasma creatinine level alone failed to identify acute kidney injury in 67.2% of the patients with low urine output.” Please provide possible rationales for this phenomenon and your suggestions for clinicians to improve this problem. (6%)
- (三) Please translate the underlined sentence into Chinese. (4%)
- (四) List 4 most common causes (disease, medication, medical intervention, etc.) predisposing AKI in critically ill patients. (12%)

接背面

五、請就下列臨床案例回答提問：

A 72-year-old hypertensive woman with no renal impairment (glomerular filtration rate using the MDRD formula $> 90 \text{ mL/min/1.73 m}^2$) was admitted for pacemaker implantation for sinus node dysfunction. She had been taking apixaban 5 mg twice daily for 7 weeks for paroxysmal atrial fibrillation (CHA2DS2-VASc score: 3). She had also been taking flecainide 50 mg/day (for 7 weeks), bisoprolol 2.5 mg/day (for 7 weeks), amlodipine 10 mg/day, sulpiride 100 mg/day, and bromazepam 3 mg/day long-term. As recommended, apixaban was interrupted 3 days before pacemaker implantation and resumed at the same dose 48 h after the procedure. Four days after reintroduction of apixaban, asymptomatic elevation of aspartate aminotransferase and alanine aminotransferase was observed, reaching 3 times and 5 times the upper limit of normal, with no significant elevation of γ -glutamyltransferase, bilirubin, or alkaline phosphatase. This transaminase elevation prompted apixaban discontinuation. The levels of both enzymes normalised within 5 days of dechallenge (Fig. 1). Ten days after pacemaker

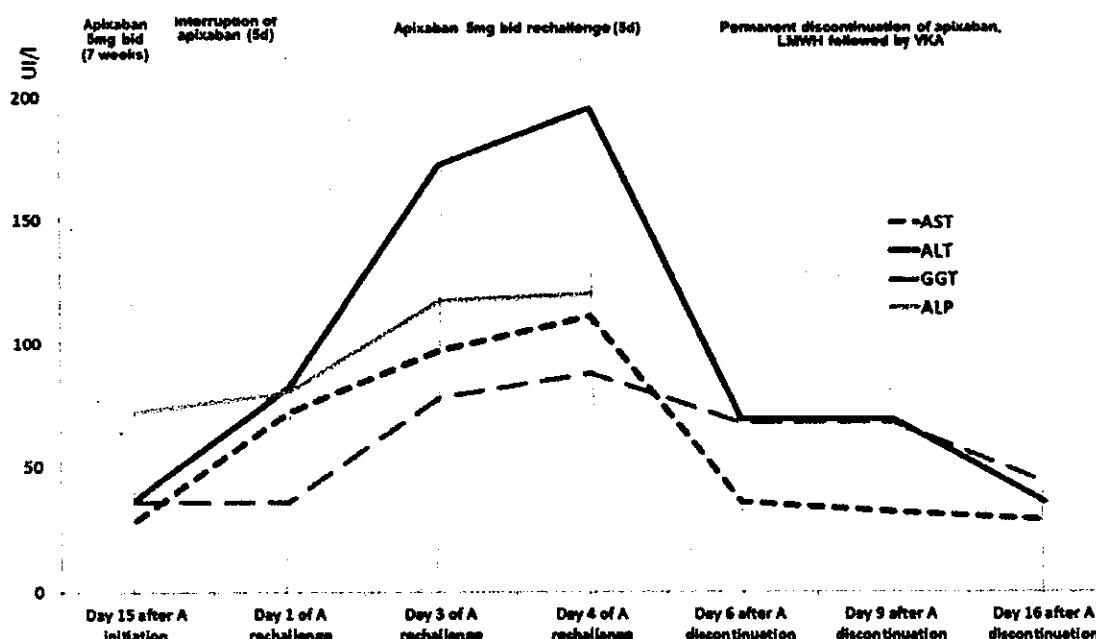


Figure 1. Effect of apixaban rechallenge and dechallenge on hepatocellular enzymes: AST and ALT peaked on day 4 after rechallenge and subsided after dechallenge. A: apixaban; VKA: vitamin K antagonist; LMWH: low-molecular-weight heparin.

implantation, low-molecular-weight heparin was introduced, followed by a vitamin K antagonist. Viral hepatitis serology was negative, and no liver abnormalities were seen on ultrasound. From: *Int J Cardiol* 2016;204:4–5.

(一) 試概要闡述內容並分析之。 (9%)

(二) 請列舉此案例之處方，並說明各藥品適應症。 (7%)