

A case of endocarditis presenting with icterus

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Abstract: *Staphylococcus aureus* (*S. aureus*) bacteremia (SAB) may be complicated by hematogenous seeding to host tissues that the cardiac valves are the major site of metastatic infection. Herein, we presented a case of endocarditis presenting with icterus in the Capital of Iran. The case was a 36-year-old man with chief complaint of fever and shaking chills for the past week. On examination, the patient was ill, but he was not toxic. Besides the high fever, he had a pulse of 110 beats/min, blood pressure of 110/70mmHg and a respiratory rate of 20 breaths/min. His skin was not icteric and no rash and legion were found, but sclera was icteric. An abdominal examination revealed no tenderness, rigidity, ascites, organomegaly or masses. Peripheral blood smear (PBS) was examined for malaria and burrelia in three times and it was negative. On the eighth day of admission, blood culture reported and it was positive for *S. aureus*. Treatment with Vancomycin was started and Trans esophageal echocardiography (TEE) was requested. There was a response to treatment with stopping of fever, reducing the size of tricuspid vegetative mass and changing of second blood culture to negative. Treatment course was completed and the patient discharged with good condition. In conclusion, careful evaluation of prior medical records and clinical course can be very helpful. Echocardiography and serial blood cultures provide the key to diagnosis. TEE is recommended in all patients with SAB to determine whether they have *S. aureus* infective endocarditis.

Keywords: Endocarditis, Icterus, Case report, *Staphylococcus aureus*.

INTRODUCTION

Despite advances in antimicrobial therapy and intensive care support, *Staphylococcus aureus* (*S. aureus*) continues to cause significant morbidity and mortality [1]. *S. aureus* is a frequent cause of nosocomial and community-acquired bacteremia. *S. aureus* bacteremia (SAB) may be complicated by hematogenous seeding to host tissues that the cardiac valves are the major site of metastatic infection. The prevalence of *S. aureus* infective endocarditis (SAIE) among patients with SAB varies from 13% to 25% [2]. Factors significantly associated with SAIE are unknown origin of SAB, a valvular prosthesis, a pacemaker, persistent fever, and persistent bacteremia [3]. Complications of intravascular catheter-associated SAB are common (range, 11–53%) and can occur in almost any body site [4]. Herein, we presented a case of endocarditis presenting with icterus in the Capital of Iran.

CASE REPORT

In Sep 2012, a 36-year-old man referred to the emergency room with chief complaint of fever and shaking chills for the past week. His fever has been as high as 102.2°F (39°C) and was repeated two to three

times a day. His fever has not responded to antimicrobial treatment such as penicillin. He complained also from myalgia, night sweat and headache. In past medical history, 5 weeks ago the patient took a trip to Tanzania and Kenya to see wildlife. He was vaccinated against yellow fever and rewarded mefloquine as malaria prophylaxis one week before and four weeks after the trip. He hasn't had any sexual affairs and no history of swimming over there. He had dental implantation 4 months ago and anal fissure surgery one year ago.

On examination, the patient was ill, but he was not toxic. Besides the high fever, he had a pulse of 110 beats/min, blood pressure of 110/70mmHg and a respiratory rate of 20 breaths/min. His skin was not icteric and no rash and legion were found. His conjunctiva was not pale and also sclerae was icteric. No cervical, axillary, or inguinal lymph node enlargement was noted. Normal heart sounds without evidence of cardiac murmur or friction rub was found. Primary laboratory data were performed in Table 1 and 2. Examination of the chest was normal and sounds throughout both lung fields was clear (Figure 1). An abdominal examination revealed no tenderness, rigidity,

ascites, organomegaly or masses. No lower extremity edema was found and dorsalis pedis, posterior tibialis

and radial pulses were felt intact and also cyanosis and clubbing were not seen.

Table 1: Primary laboratory data, cell blood count and differential count

Variables	Result	Unit	Reference range
Cell blood count			
WBC	6.9	$\times 10^3/\mu\text{l}$	4.4-11.2
RBC	4.8	$\times 10^6/\mu\text{l}$	4.4-5.9
Hb	13.5	g/dl	13-18
HCT	39	%	40-52
MCV	80.6	fl	80-96
MCH	27.9	pg	27.5-33.2
MCHC	34.6	g/dl	31.5-35.5
PLT	85	$\times 10^3/\mu\text{l}$	150-450
RDW	13.1	%	11-16
Differential count			
NEUT	65	%	45-70
LYMP	16	%	30-50
MONO	5	%	1-7
EOS	1	%	0.5-5
BAND	13	%	-

Table 2: Primary laboratory data, biochemistry and urine

Variables	Result	Unit	Reference range
Blood biochemistry			
Bilirubin (total)	3.7	mg/dl	0.1-1.2
Bilirubin (direct)	2.1	mg/dl	<0.3
AST	141	IU/l	<45
ALT	152	IU/l	<45
ALKP	210	IU/l	64-306
BUN	12	mg/dl	10-55
Cr	0.9	mg/dl	0.6-1.5
Na	129	meq/l	135-145
K	4.1	meq/l	3-5.5
BS	83	mg/dl	<140
Urine analysis			
Color	Yellow		
App	Semi clear		
PH	5		
SG	1010		
Protein	Trace		
Glucose	Negative		
Ketones	Negative		
Bilirubin	Negative		
Urobilinogen	Negative		
Blood	1+		
Nitrite	Negative		
Ascorbic acid	Negative		
Microscopic			
WBC	2-5	Cells/LPF	
RBC	10-25	Cells/LPF	



Fig 1: Chest, X-ray

The patient was treated with the anti-malaria agent, Artesunate for three days. Fever was continued intermittently and thrombocytopenia was detected, yet. The patient was treated with anti malaria for the second round and the same time ceftriaxone was added to

antimicrobial agents. On the fifth day of admission, total bilirubin was 1.5mg/dl with direct component of 0.75mg/dl, AST: 76IU/l, ALT: 85 IU/l, Hb: 12.4g/dl and PLT: $54 \times 10^3/\mu\text{l}$. The additional lab tests were performed in **Table 3**.

Table 3: The additional lab tests

Variables	Result
ESR	58mm/h
CRP	37mg/l
Wright	Negative
2ME	Negative
Widal	Negative
S.typhi(OD)	Negative
s typhi para A(OA)	Negative
s typhi para B(OB)	Negative
s typhi (HD)	Negative
s typhi paraA(Ha)	Negative
s typhi para B(Hb)	Negative
Anti HIV	Negative
Anti HBS	2mIU/l
Anti HBC	Negative
Anti HCV	Negative
HBS Ag	Negative

Peripheral blood smear (PBS) was examined for malaria and burrelia in three times and it was negative. Lab test for leptospira was negative. CBC was checked daily and in spite of the anemia and decreasing of Hb (13 to 10.9g/dl), schistocyte was not seen on PBS. Stool exam for occult blood was negative and repeated urine analysis was normal. At the same time LDH was 327U/l, Retic count: 1.4%, TIBC: 147 $\mu\text{g/dl}$ and Ferritin: 453ng/ml. On the seventh day of the treatment, PLT count was elevated to $120 \times 10^3/\mu\text{l}$, but the fever was continued, yet. On the eighth day of

admission, blood culture reported and it was positive for *S. aureus*. Treatment with Vancomycin was started and Trans esophageal echocardiography (TEE) was requested (**Figure 2**). TEE report on tricuspid valve showed large, highly mobile hypoechoic mass on the anterior leaflet (13 \times 18mm) compatible with vegetation with mild tricuspid regurgitation and small patent foramen ovale. Forty-eight hours later, the fever was stopped and PLT returned to normal. During hospitalization Purpura and petechiae on both legs was appeared (**Figure 3**).



Fig 2: Trans esophageal echocardiography



Fig 3: Purpura and petechiae on patient's leg

The patient had complaint of abdominal pain during treatment, but abdomino-pelvic CT scan was normal (**Figure 4**).



Fig 4: Abdominal CT scan

The second blood culture was negative and the follow-up echocardiography after two weeks showed, reduction in the size of the mass. Treatment course completed and the patient discharged with good condition.

DISCUSSION

Infectious endocarditis results from bacterial or fungal infection of the endocardial surface of the heart and associates with significant morbidity and mortality. Risk factors include the presence of a

prosthetic heart valve, structural or congenital heart disease, intravenous drug use, and a recent history of invasive procedures [5]. Endocarditis is caused by *S. aureus* presents as two distinct clinical syndromes depending on the patient population (intravenous drug user or non-addictive) and the location of infection (right-sided or left-sided). Medical management alone is often successful for this disease, but in certain subsets of patients, notably those with infection of aortic or multiple valves, early operation may be necessary [6]. Lesens et al.; [7] found that a previous valvular defect on a native valve was the single unfavorable risk factor for SAIE in patients with SAB. This case was right sided despite of no previous history of addiction, but had mild tricuspid regurgitation.

All patients with SAB should undergo a TEE irrespective of whether a risk factor for infective endocarditis (IE) has been identified or not to avoid the uncertainties that plagued previous studies [3]. TEE is recommended early in all patients with SAB [8]; its sensitivity for detecting vegetations is 75% to 95% and specificity, 85% to 98% [2]. One study [1], suggested that the presence of hyperbilirubinemia in patients with *S. aureus* sepsis may identify persons at high risk of dying from overwhelming sepsis. In patients with icterus, however, bilirubin can potentially hinder cerebral oximetry [9]. Endocarditis should be suspected in patients with unexplained fevers, night sweats, or signs of systemic illness [5]. The most commonly reported symptom for patients with SAB was fever that the mean leucocyte and platelet counts were $14.95 \times 10^9/l$ (range, 0.7–65), and $259 \times 10^9/l$ (range, 23–760), respectively. A platelet count of $<100 \times 10^9/l$ was associated with a significantly increased mortality ($P < 0.002$). Out of 113, one hundred and two patients (90%) developed one or more complications of SAB and endocarditis was diagnosed in 19 patients (17%) whom 13 died (68%, $P < 0.0001$) [10]. These complications are especially important, because they are difficult to diagnose and are often associated with poor outcome [11]. Therefore, persistent fever and persistent bacteremia appear to be the most consistent characteristics that indicate a high risk of SAIE. However, even in the absence of identifiable risk factors, the risk of SAIE remains high, up to 16% [12, 13]. Clinicians should be alert for complications in all patients with SAB.

CONCLUSIONS

Careful evaluation of prior medical records and clinical course can be very helpful. Echocardiography and serial blood cultures provide the key to diagnosis. TEE is recommended in all patients with SAB to determine whether they have SAIE.

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