## **Case Report:**

# An interesting case of wasp sting presenting as acute kidney injury and kounis syndrome

Dr. VIGNESH .P, \*Dr. PRADEEP SHENOY, Dr. JANARDHAN KAMATH

Department of General Medicine and Nephrology, K.S.Hegde Medical Academy, Deralakatte, Mangalore Corresponding author\*

#### **ABSTRACT**

Wasp stings are common occupational health hazard among farmers in tropical countries. Wasp sting venom has a cocktail of different active substances and enzymes causing local and systemic effects(including acute kidney injury) which needs immediate and aggressive treatment. Being a highly vascularised organ, the kidney is vulnerable to various environmental toxins. Wasp sting(especially multiple sites) are known to cause acute kidney injury and kounis syndrome. Early diagnosis and aggressive treatment may prevent worsening of kidney injury and need for dialysis. Also need to be vigilant about kounis syndrome, especially when risk factors for coronary artery disease exist and treat it promptly.

### INTRODUCTION

Wasp stings are common occupational health hazard among farmers in tropical countries. Wasp sting venom has a cocktail of different active substances and enzymes causing local and systemic effects(including acute kidney injury) which needs immediate and aggressive treatment. Being a highly vascularised organ, the kidney is vulnerable to various environmental toxins.<sup>(1)</sup>

## **CASE REPORT**

A 45 year old male, farmer, with no known comorbidities presented to our hospital with history of multiple wasp stings—generalised ,more in both upper and lower limbs associated with pain at sting sites and reduced urine output since 1 day. Also gives history of associated chest discomfort with giddiness and fall(one episode) after wasp sting. No history of chest pain, breathlessness, palpitations or other cardio respiratory complains

On examination, he was conscious, oriented, obeying commands.

Vitals-BP-166/90mmhg, heart rate-64bpm, oxygen saturation-98% on room air, respiratory rate-17cpm, JVP-normal, no pedal edema

General physical examination revealed multiple wasp sting wounds(hyperpigmented plaque with central necrosis, as shown in figure 2) distributed over bilateral upper and lower limbs, abdomen, total of 15 in number, size varying from  $0.5 \times 0.5 \text{cm}^2$  to  $1 \times 0.8 \text{cm}^2$ , no active discharge/bleeding.

Systemic examination(cardiovascular, respiratory ,per abdomen and central nervous system) were normal.



Figure 1: Showing an adult wasp(left) and its nest with colony(right)



Figure 2: Showing patient who is victim of wasp sting, with sting wounds over right arm(top left and right), left arm(bottom left), left foot(bottom right)

## LABORATORY RESULTS

Hb-16.5g%, TC-12,400 cells.mm<sup>3</sup>, DC-88%neutrophils, 12%lymphocytes, platelet count-3.48 lakh cells/mm<sup>3</sup>.

Urea-43.7, creat-1.74, sodium-135, potassium-4.22, venous bicarbonate-16.0(at admission)

LFT-total protein:5.9(g/dl), bilirubin(mg/dl)-1.17(total), 0.26(direct), 0.91(indirect), SGOT-50U/L, SGPT-20U/L, ALP-76, alb-3.7(g/dl), glob-2.20(g/dl).

Urine routine-Ph 6.0, protein trace, 2-4 epithelial cells, 6-8 RBC, 4-6 wbc, granular casts present.

CPK-1333(U/L)(increased to 4020 on day 2), CKMB-83.4(IU/L) Trop T normal .Serum LDH-378(U/lt), HBA1c-5.6% .

USG abdomen and pelvis- Right Kidney- 9.6X4cm²,Left Kidney- 9.9x4.8cm²,mild hepatomegaly(16.6cm) , 2DECHO –no RWMA, concentric LVH, LVEF 60%. Coronary angiogram(done on day 4)-normal epicardial coronaries.

D A Y	URE A (mg/dl )	CREATI NINE (mg/dl)	SODIU M (mmol/ L)	POTASS IUM(mm ol/L)	INTAKE/O UTPUT(in ml/day)
1	43.7	1.74	135	4.22	825/320
2	52.2	1.55	139	4.27	1183/1300
3	67.1	1.52	137	3.86	2498/1705
4	46.3	1.24	141	3.49	3306/2720
5	34.5	1.18	140	3.45	2450/3675
6	26.3	1.01	140	3.23	1400/1630
7	35.9	1.14	136	3.33	1400/1695
8	38.9	1.08	132	3.45	1706/1400
9	31.5	0.98	138	3.87	2500/2100

Table 1: showing serial monitoring of renal function test and intake output charting

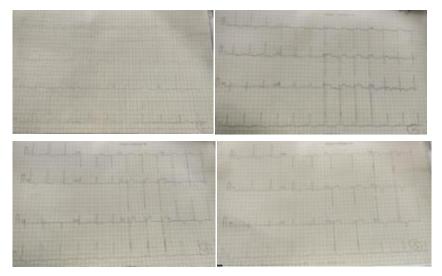


Figure 3: serial ECGs showing ST segment elevation in V2 to V4 and LVH with strain features. Day 1(top left), Day 2(top right), day 3 (bottom left), and day 4(bottom right)

Day 1 ecg showed significant ST segment elevation(3mm) in lead v2to v4 and LVH with strain.

Days 2, 3 and 4 showed resolving ST elevation(<3mm) compared to day1 in same corresponding leads



Figure 4: day 5 ECG showing insignificant ST elevation(1mm) in v2,v3

## **COURSE IN HOSPITAL:**

On evaluation lab reports were suggestive of acute kidney injury(AKI) with evidence of intravascular hemolysis and rhabdomyolysis, most probable cause of AKI being ATN(acute tubular necrosis).

Patient was treated with aggressive IV fluids and symptomatic support. Strict intake/output charting and vitals were monitored. Subsequently renal parameters improved and normalized on day 4 of hospital stay(as shown in table 1). ECG at admission showed ST segment elevation in lead v2 to v4 with elevation in CPK and CKMB levels. Patient was started on antiplatelets with heparin. Troponin T was normal. 2D echo did not show and evidence of RWMA at admission. He was taken up for coronary angiogram once creatinine normalised and total intake output was satisfactory (day 4 of hospital stay).

Coronary angiogram was normal. Serial daily ECG monitoring showed normalising of ST segment changes from day 1 to day 5 of hospital stay(as shown in figure 3 and figure 4), hence antiplatelets and heparin were stopped after angiogram.

Patient was monitored in the wards with continuing I.V fluid therapy after coronary angiogram. His vitals were stable, output improved and was discharged on day 10.

#### **DIAGNOSIS:**

- 1. ACUTE KIDNEY INJURY-?ACUTE TUBULAR NECROSIS, SECONDARY TO WASP STING WITH INTRAVASCULAR HEMOLYSIS AND RHABDOMYOLYSIS.
- 2. WASP STING INDUCED TYPE 1 KOUNIS SYNDROME
- 3. SYSTEMIC HYPERTENSION

#### DISCUSSION

Wasps are a group of stinging insects belong to family vespoidea under order Hymenoptera. Further divided into social wasps and solitary wasps. Social wasps - yellow jacket and hornet, they are aggressive, live in colonies with their nests found in tree stumps ,mammal burrows, fixed to side of a shed or house. Solitary wasps-mud wasp, act as predators, feed on smaller insects and their nests are found in crevices of windows. Wasp sting venom contents: phospholipase A2 and hyaluronidase, mellitin and chemotactic peptides, amines such as histamine, serotonin and catecholamines, and others such as mastoparan, kinins, apamine, acetylcholine, antigen 5 and neurotoxic cynines<sup>(1)</sup>. Phospholipase A, mellitin and apamin-cause hemolysis<sup>(2)</sup>.

Local effect- inflammation/urticarial rash. Systemic effect-commonly seen in multiple stings, causes intravascular hemolysis, rhabdomyolysis, AKI, and hepatitis.. Less commonly- cardiac (myocarditis/myocardial infarction), neurological(acute encephalopathy/stroke)<sup>(1),(3)</sup>complications, systemic allergic reactions and Anaphylactic shock.

AKI in wasp sting occurs due to two types of renal pathologies-ATN or AIN(acute interstitial nephritis). ATN-due to intravascular hemolysis, rhabdomyolysis following which pigment nephropathy occurs caused by precipitation of hemoglobin and myoglobin casts in renal tubules. Myoglobin is freely filtered in glomeruli, when it is present in high concentration, along with dehydration and acidic urine, it precipitates in the proximal tubule to form obstructive pigment casts. AIN-due to direct nephrotoxic effect from hypersensitivity to venom. (3) Histopathology-features of AIN and/or ATN with pigment casts (1)(2).

Treatment of AKI in wasp sting- aggressive i.v fluids and forced alkaline dieresis. May need steroids(in case of AIN). Those who survive, there is usually complete recovery without residual renal impairment<sup>(2)</sup>. Kounis syndrome is the occurrence of acute coronary syndrome in conditions involving mast cell activation like allergy/hypersensitivity/anaphylactoid reactions. Mediated by histamine, neutral proteases, cytokines and chemokines<sup>(4,5)</sup>.

3 variants/types of kounis syndrome-type 1-in patients with normal coronary arteries without any predisposing factors for coronary artery disease, causing coronary artery spasm without raised cardiac enzymes and troponins or coronary artery spasm leading to myocardial infarction with raised cardiac enzymes and troponins. Type 2-patients who have a pre-existing atheromatous disease developing an acute MI due to coronary spasm/plaque

erosion/rupture. Type 3 variant-patient with coronary artery stent(especially drug eluting)developing a thrombosis in the stent due to allergic reaction<sup>(6)</sup>.

Treatment of kounis syndrome-treating the allergic event with steroids and antihistamine(type 1 variant). Nitrates, calcium channel blockers and anti platelets with heparin maybe needed for type 2 and 3 variants. Complications of kounis syndrome-myocardial infarction, arrhythmias(more commonly seen in type 2 and 3 variants)<sup>(7,8,9)</sup>.

In our patient, after complete evaluation, a wasp sting induced AKI with intravascular hemolysis and rhabdomyolysis was made(other causes of AKI were ruled out). Patient responded well to aggressive IV fluid and symptomatic therapy. In view of ECG changes, was suspected to have MI, but trop t was negative, 2d echo didn't show any evidence of RWMA(regional wall motion abnormality), CAG was also normal(done on day 4 when creatinine was normal). ECG changes of ST elevation returned to normal without any thrombolysis/reperfusion therapy, hence, the ECG findings and clinical scenario could be due to acute coronary spasm secondary to allergic/anaphylactic reaction to wasp sting-a diagnosis of wasp sting induced type 1 kounis syndrome was made.

#### **CONCLUSION**

Wasp sting(especially multiple sites) are known to cause acute kidney injury and kounis syndrome. Early diagnosis and aggressive treatment may prevent worsening of kidney injury and need for dialysis. Also need to be vigilant about kounis syndrome, especially when risk factors for coronary artery disease exist and treat it promptly.

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