SYNTHETIC CANNABINOID AND KIDNEY

Erman ÖZDEMIR[1], Pınar KUTLUTÜRK ÖZDEMIR[2], Erdogan ÖZDEMIR[3], Meral Mese[1], Ergün Parmaksız[1], Gülsah Boz[1], Okan Akyüz[1], Zerrin BICIK BAHÇEBASI[1],

[1]Dr Lutfi Kirdar Training and Research Hospital, Nephrology, Istanbul, TURKEY,

[2]Dr Lutfi Kirdar Training and Research Hospital, Internal medicine, Istanbul, TURKEY,

[3]Fırat University, School of Medicine, Internal medicine, Elazıg, TURKEY.

INTRODUCTION AND AIMS: Synthetic cannabinoid (SCs) use has been increasing in Turkey parallel to the rest of the world. It is more commonly named as bonzai, jamaica, k2 or spice and is used illegally for pleasure and hallucinogenic effects. Here we presented six patients who were followed up in Nephrology clinic with acute kidney injury (AKI) due to synthetic cannabinoid usage in last six months .

METHODS: Single centre experience; Here we presented six patients who were followed up in Nephrology clinic with acute kidney injury (AKI) due to synthetic cannabinoid usage in last six months .

RESULTS: Six male patients aged between 28-42 years were admitted to the nephrology clinic, after presenting to the ER with nausea, vomiting and abdominal pain and whose laboratory results showed high levels of urea and creatinine. Clinical and laboratory findings in all six patients are summarized in Table 1. The most evident complaints of patients were nausea and vomiting. The patients declared that nausea-vomiting started approximately 10-16 hours before coming to the ER and approximately 2-4 hours after inhalation. All 6 cases have abdominal pain starting from the epigastric region and felt more severely in both lumbar regions. Metabolic alkalosis was observed in 4, normoacidemia in 2 patients. Kidney biopsies were performed in 2 of the cases (Case 3 and 5) revealed evidence of acute tubular necrosis, focal tubular atrophy, flattened and granulated epithelium and interstitial fibrosis. On the other hand; hypertrophic and global sclerotic glomerulies were observed. Vessels were unremarkable, with no significant staining on the immunofluorescence.

CONCLUSIONS: SCs using is growing up daily, particularly in young generation and becoming a serious public health threat. In less than 6 months, a total of 6 cases were hospitalised in our clinic and unfortunately 4 of them (%66) became dependent on hemodialysis with ESRD. In most part of cases it is difficult to determine the reason for the loss of organ function and effects / side effects since additives and unknown herbal / chemical substances are being used in the preparation . Since the patients usage of SCs was not under a commercial brand or single form, the determination of the toxic substance seems very difficult. Adding to the challenge of recognizing SC intoxication is the lack of rapid laboratory tests to confirm exposure.

In the majorty of the cases (n 4) , it has been thought that metabolic alkalosis has been developed though loss of gastrointestinal (GIS) acid. It has been further observed that hypovolemia due to loss of through GIS triggers/accentuates acute kidney damage. The common character of the progressing cases to ESRD were long term and dense exposure definitions. Sclerotic glomeruli and tubular atrophy results of two cases supported this hypothesis. The main difference between AKI connected to SCs and other toxic nephropathies was that they were presented with metabolic alkalosis. We propose that both toxic and ischemic, tubular injury due to direct effect of SC (or potentially other added constituents such as heavy metals) can be responsible.

Table 1

Patient	Age (years)	Duration of SCD use (years)	Last use of SCD (hours prior to admission)	Major symptom at presentation	*SCr at admision/ peak (mg/dl)	pH/HCO^{3-} (mEq/L)	Urine microscopy (hpf)
1	28	2	12	flank /abdominal pain	4,6/ 6,2	7.38/24	20-25 RBCs, 6-8 WBCs
2	30	1	10	abdominal / back pain	8,7/ 8,7	7.48/30	10-12 RBCs, 1-2 granular casts
3	32	4	14	flank pain	5,2/ 6,1	7.40/26	5-7 RBCs
4	32	unclear	20	Nausea and vomiting	5,6/ 5,6	7.50/36	10-12 RBCs
5	37	unclear	12	Nausea and vomiting	6,2/ 8,0	7.52/40	16-18 RBCs
6	42	5	16	Nausea and vomiting	6,4/ 8,2	7.50/36	8-10 RBCs, 4-6 WBCs

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