Original Research Paper

Volume-8 | Issue-8 | August-2018 | PRINT ISSN No 2249-555X

Medicine

SEVERE HYPERKALEMIA DURING RADIOFREQUENCY ABLATION OF HEPATOCELLULAR CARCINOMA

Dr. Prakash Lisa (Anaesthesia Consultant), Department of Anaesthesiology, Noble hospital, Pune.

Dr. Adnanali Sarkar * (Anaesthesia Consultant), Department of Anaesthesiology, Noble Hospital, Pune.
*Corresponding Author

ABSTRACT Tumor lysis syndrome (TLS) is characterized by hyperuricemia, hyperphosphatemia, hypocalcemia, and hyperkalemia resulting from rapid tumor cell death. We report a 47 yr old male, K/C/O Liver Cirrhosis & Diabetes Mellitus (DM) , Hepatitis B carrier, recently diagnosed as early stage of Hepatocellular Carcinoma(HCC) was posted for radio frequency ablation (RFA) under General Anaesthesia(GA). The patient's renal function tests and serum potassium level were all within normal limits. At the end of the procedure, the patient developed sudden ventricular tachycardia. Immidiate blood tests showed serum and plasma potassium level more than 7 mEq/L, but no other abnormalities. The thermal destruction of large tumors during radiofrequency ablation may be associated with extensive cell breakdown and transcellular shift of potassium.

KEYWORDS : Tumor lysis syndrome, Hepatocellular caricinoma, Hyperkalemia.

Introduction:

Tumor lysis syndrome is a common complication resulting from treatment of blood-borne malignancies such as acute lymphocytic leukemia and high-grade non-Hodgkin's lymphoma[1]. Tumor lysis syndrome is characterized by hyperuricemia, hyperphosphatemia, hypocalcemia, and hyperkalemia resulting from rapid tumor cell death. Fewer than 50 cases of tumor lysis syndrome related to treatment of solid organ tumors have been reported because these cancers are inherently resistant to cytotoxic therapies [1–3]. Two of these cases followed hepatic arterial chemoembolization of hepatocellular carcinoma (HCC) and represent the only episodes of tumor lysis syndrome from treatment of this malignancy described, to our knowledge [4]. TLS has been reported in several different solid tumor types, including hepatocellular carcinoma (HCC) and cholangiocarcinoma.There are 13 published reports of primary hepatic malignancy presenting with TLS and acute kidney injury[9-20]. We report a case of tumor lysis syndrome after radiofrequency ablation of HCC.

Case report

A 47 yr old male, K/C/O Liver Cirrhosis & Diabetes mellitus(DM) on regular treatment, Hepatitis B carrier, recently diagnosed as early stage of Hepatocellular Carcinoma (HCC). Patient was on Metformin, Tenofovir, and Spironolactone and was posted for radio frequency ablation (RFA) under General Anaesthesia(GA).

Preanaesthesia check-up was done, pulse- 72/ min, BP- 130/80 mm Hg, Respiratory system, Cardiovascular system and Central nervous system- normal findings .

Laboratory Investigation- Hb- 11.2 gm%, platelets- 90,000/cumm, PT- 20 sec, INR- 1.61, Sr electrolytes- Na/k/cl- 138/4.1/108(meq/L), urea/ creatinine- 20/0.73.blood suagr level (BSL)- 209. LFT – total bilirubin-0.62 D/I- 0.2/0.42, ALP- 270. ECG/ Chest x ray- WNL . CT Abdomen- cirrhotic liver with focal lesion in segment V , s/o of HCC. Pre- procedure 3 FFPs were given to correct deranged coagulation profile. Advised for adequate reservation of Platelets , FFP and PCVs.

In CT scan room Anesthesia equipments and machine , monitors, suction and all anesthetic and emergency drugs were kept and checked prior to procedure . Monitors were attached to the patient. Pulse- 80/min with regular rhythm, BP- 160/90 mm of Hg, SpO2- 100% on Air .

Pre oxygenation with 100% O2 was done. Premedication with inj. Glycopyrrolate 0.2mg + inj. Emeset 4 mg + inj. Midazolam 1mg+ inj. Fentanyl 75mcg+ inj Pantoprazole 40 mg+ inj. Supacef 1.5 gm. Induction with inj. Propofol 80 mg with cont. Oxygenation followed by inj. Atracurim 35 mg, manually ventilated for 3minutes. Patient was intubated with 8.5 cuffed ETT , AEBE, & fixed, throat packing was done, patient made prone, air entry rechecked, vitals stable through out the induction. . RF procedure was done under CT guidence, 3 overlapping ablation of 3cm each duration of procedure was 2hrs, patient was manually ventilated through out the procedure and maintained with Oxygen and inj. Atracurium and intermittent inj. Propofol.Vitals were stable through out the procedure. At the end of procedure, patient made supine ,throat pack was removed, patient was showing spont attempts of breathing and reversal inj. Glycopyrrolate 0.4mg and inj. Neostigmine 2.5 mg was given slowly. Oral suctioning was done, patient was spontaneous regularly breathing.

On ECG ventricular tachycardia (VT) was seen just prior to extubation HR increased 90- 140/min with BP 160/70mm Hg, SpO2 -92%. Extubation withheld. CODE BLUE was announced, carotid and femoral pulsation was present along with VT on ECG and shock was given with 200J in 2 episodes along with inj. Calcium gluconate 10ml and inj. Soda bicarbonate 25ml. Junctional Rhythm was seen with bradycardia 40/min, patient became pulseless CPR started inj Atropine 0.6 mg was given and rhythm change to broad QRS with tall T wave , BP- 130/90 , SPO2- 99%, and patient was shifted ICU for further management with manual ventilation.

In ICU- There was an episode of pulseless VT, repeat DC shock 200 J, rhythm revived to sinus tachycardia. Immediate ABG showed- PH- 7.0, PCO2-71, PO2- 84, HCO3-19.6, SPO2-90, K- 7 ,RBS- 430 mg/dl, Cardiac refference was done , which showed global hypokinesia with EF- 30%.

Post resuscitation vitals- pulse- 130/min, BP- 103/60 mmofHg, SPO2-98%. Correction of potassium was started with G.I Drip along with Nor-Adrenaline infusion to maintain haemodynamic stability. Pt was on ventilatory support for 4 days and extubated on day 5 & shifted out to ward on next day. Discharged 3 days after extubation .

Table- Summary of investigation reports:

Date	Pre procedure	Post procedure	Post procedure day1	Day2	Day3	Day4	Day5
Hb (g%)	11.2		13.3	10.3	9.0	9.4	
Platelets(cu mm)	90,000		1.02	73,000	45,000	58,000	
Na(meq/L)	138	136,129,135	140	141	146	145	144
K(meq/L)	4.1	>7,>6,4.6	4.5	3.9	2.9,3.1	3.4	3.3
Cl(meq/L)	105	100,100,103	103	108	107	107	108
Ca(mg/dl)		10.1	8.7				
PO4(mg/dl)		3.3	3.4				
Mg(meq/L)		2.1	1.8				
LDH(U/L)		1127	982				
Urea(mg/dl)			37		51		
Creat(mg/dl)	0.73		1.02		0.91		
ABG-PH		7.0,7.26,7.44	7.38	7.43	7.42	7.47	
PCO2(mm Hg)		71,45,21.8	33	38	37	30.3	

HCO3(meq/L)		19.6,19.8,21.7	19.5	25.2	24	29.1	

Discussion

The tumor lysis syndrome (TLS), characterized by hyperkalemia, hyperuricemia,hyperphosphatemia and secondary hypocalcemia, is a consequence of treatment induced or spontaneous tumor cell death. A variety of therapeutic techniques can lead to tumor lysis syndrome including chemotherapy, radiation therapy, immunotherapy, interferon therapy, hormonal therapy, surgery, and chemoembolization [1–4].

Serum electrolyte abnormalities are the hallmark of tumor lysis syndrome. Hyperkalemia (>5 meq/L) occurs in 93% of patients; is often the first sign of TLS. It results from potassium leakage from dying cells and can lead to greatest immediate threat cardiac dysrhythmias and arrest. Hyperphosphatemia develops in 89% of patients and also results from cell breakdown.Free phosphate binds with calcium and results in hypocalcemia, which is identified in 89% of patients with changes in mental status, tetany, and seizures. Hyperuricemia occurs in 98% of patients and also results from cell death[5]. In our case other electrolytes abnormality couldnot found except Hyperkalemia.

The major causes of TLS in HCC are either spontaneous i.e. when cancer cells die without preceding chemotherapy, embolization, or radiation therapy, or secondary to cancer targeted specific treatment e.g. following transarterial chemoembolisation / radiofrequency ablation or on low dose of steroids or thalidomide .

In our case the post procedure blood tests showed serum and plasma potassium level > 7 mEq/L, but no other abnormalities. The thermal destruction of tumors during radiofrequency ablation may be associated with extensive cell breakdown and transcellular shift of potassium. We theorize, that during the prolonged ablation, the contents of the dying tumor cells may have intravasated. Such intravasation may have contributed to the severe electrolytic disturbances and explain the unusual development of tumor lysis syndrome from treatment of a relatively small solid mass. The differential diagnosis in our patient could be hemolysis leading to renal insufficiency. However, our patient did not have a decrease in hemoglobin immediate after the procedure, it happened 3 days after the procedure.

Management of tumor lysis syndrome is principally managing the symptoms with hydration to maintain urine flow and allopurinol to neutralize the uric acid salts [1]. Prophylactic treatment with prehydration and allopurinol is not routinely recommended in patients with solid tumors because of the rarity of tumor lysis syndrome in this setting [2]. Our ability to hydrate Our patient was limited by diminished ejection fraction(30%) and chances of fluid overload. So we corrected his hyperkalemia with Glucose –Insulin drip and started Noradrenaline infusion to maintain haemodyanamic parameters.

In conclusion, tumor lysis syndrome is an uncommon complication of cytotoxic treatment of solid malignancies. Hallmarks include hyperkalemia, hyperuricemia, hypocalcemia, and hyperphosphatemia. Routine prophylaxis with allopurinol is not necessary before radiofrequency ablation for all patients with elevated lactate dehydrogenase, elevated uric acid, and azotemia; hydration should be strongly considered if risk factors are present. Hydration and allopurinol are clearly mandatory if concern regarding tumor lysis syndrome arises after radiofrequency ablation.

References

1. Baeksgaard L, Sorensen JB. Acute tumor lysis syndrome in solid tumors: a case report and review of the literature. Cancer Chemother Pharmacol 2003;51:187–192
2. Kalemkerian GP, Darwish B, Varterasian ML. Tumor lysis syndrome in small cell carcinoma and other solid tumors. Am J Med 1997; 103:363–367
3. Castro MP, VanAuken J, Spencer-Cisek P, Legha S, Sponzo RW. Acute tumor lysis syndrome associated with concurrent biochemotherapy of metastatic melanoma: a case report and review of the literature. Cancer 1999; 85:1055–1059
4. Burney IA. Acute tumor lysis syndrome after transcatheter chemoembolization of hepatocellular carcinoma. South Med J 1998; 91:467–470.
5. Shannon G. Lehner1 Jennifer E. Gould2 Wael E. A. Saad2 Daniel B. Brown2 Lehner SG, Gould Tumor Lysis Syndrome After Radiofrequency Ablation of Hepatocellular Carcinoma AJR 2005;185:1307–1309
6. Mayank Jain, Joy Varghese, Jayanthi V Tumor lysis Syndrome: Pathogenesis and Risk Factors with Special Reference to Hepatocellular Carcinoma – A review. ARC Journal of Hepatology and Gastroenterology Volume 1, Issue 1, 2016, PP 23-26.
7. PK MUTHUKUMARASWAMY Oncologic Emergencies: Tumor Lysis SyndromeMedicine Update (359-363).
8. JuliaMetznerMD. Jennifer L.EvansDO.Karen B.DominoMD. Life-threatening hyperkalemia during radiofrequency ablation of hepatocellular carcinoma, Journal of Clinical Anaesthesia Volume 22, Issue 6 . September 2010, Pages 473-476.
9. Lehner SG, Gould JE, Saad WE, Brown DB. Tumor lysis syndrome after radiofrequency ablation of hepatocellular carcinoma. AJR Am J Roentgenol 2005;185(5):1307–1309.
10. Hsieh P-M, Hung KC, Chen YS. Tumor lysis syndrome after transarterial chemoembolization of hepatocellular carcinoma: case reports and literature review.World J Gastroenterol 2009;15(37):4726–4728.
11. Burney IA. Acute tumor lysis syndrome after transcatheter chemoembolization of hepatocellular carcinoma. South Med J 1998; 91(5):467–470.
12. Vaisban E, Braester A, Mosenzon O, Kolin M, Horn Y. Spontaneous tumor lysis syndrome in solid tumors: really a rare condition, AmJ Med Sci 2003;325(1):38–40
13. Lee CC, Wu YH, Chung SH, Chen WJ. Acute tumor lysis syndrome after thalidomide therapy in advanced hepatocellular carcinoma. Oncologist 2006;11(1):87–88, author reply 89
14. Sakamoto N, Monzawa S, Nagano H, Nishizaki H, Arai Y, Sugimura K. Acute tumor lysis syndrome caused by transcatheter oily chemoembolization in a patient with a large hepatocellular carcinoma. Cardiovasc Intervent Radiol 2007;30(3):508–511
15. Shiba H, Ishida Y, Wakiyama S, Sakamoto T, Misawa T, Yanaga K. Acute tumor lysis syndrome after transarterial chemoembolization for hepatocellular carcinoma. Cancer Sci 2008;99(10): 2104–2105
16. Huang W-S, Yang CH. Sorafenib induced tumor lysis syndrome in an advanced hepatocellular carcinoma patient. World J Gastroenterol 2009;15(35):4464–4466
17. Shiozawa K,WatanabeM, Takenaka H, et al. Tumor lysis syndrome after sorafenib for hepatocellular carcinoma: a case report. Hepatogastroenterology 2010;57(101):688–690
18. Chao CT, Chiang CK. Rasburicase for huge hepatocellular carcinomawith tumor lysis syndrome: case report. Medical principles and practice: international journal of the Kuwait University. Health Science Centre 2012;21(5):498–500E
19. Nishida Y, Fujii H, Hagihara A, et al. [Tumor lysis syndrome after transarterial embolization for hepatocellular carcinoma]. Nippon Shokakibyo Gakkai Zasshi 2013;110(3):441–448
20. Ali AM, Barbaryan A, Zdunek T, Khan M, Voore P, Mirrakhimov AE. Spontaneous tumor lysis syndrome in a patient with cholangiocarcinoma. J Gastrointest Oncol 2014;5(2):E46–E49
21. Kekre N, Djordjevic B, Touchie C. Spontaneous tumour lysis syndrome. CMAJ 2012;184(8):913–916