Acta Biomed 2023; Vol. 94, Supplement 1: e2023224 DOI: 10.23750/abm.v94iS1.14917

Case report

Reversible supraventricular tachycardia and left bundle branch block in a marathon runner with exertional heat stroke in the Po Valley

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Abstract. We report the case of a 52-year-old marathon runner admitted to our emergency department for exertional heat stroke (EHS). The electrocardiogram (ECG) showed a supraventricular tachycardia, probably an atrial flutter with a 2:1 block, conducted with a left bundle branch block. After 10 minutes of aggressive fluid management and rapid external cooling, the ECG returned to normal. As the high-sensitivity cardiac troponin I was elevated, coronary angiography and an electrophysiological study were performed, revealing normal coronary arteries, and excluding inducible arrhythmias. As reported in the current literature, our findings confirm that the electrocardiographic changes and elevation of cardiac markers in EHS do not reflect cardiac ischemia, but rather a myocardial injury due to the pathophysiological response to dehydration and hyperthermia, which markedly impaired stroke volume and cardiac output. EHS is a life-threatening condition with a complex pathophysiology caused by thermoregulatory failure. Diagnosis is not always straightforward, but early recognition and timely management (the "golden hour") with rapid cooling and intravenous fluids are crucial to prevent irreversible and fatal organ damage. EHS is defined by a rectal temperature > 40.5 °C with symptoms or signs of neurological dysfunction, such as confusion, drowsiness, or seizures, which can rapidly worsen with delirium, coma, and cardiac arrest. With this case report, we want to remind emergency physicians that early diagnosis and appropriate management of EHS can avoid death and inappropriate treatment. (www.actabiomedica.it)

Key words: heat stroke, critical care, left bundle branch block, tachycardia, myocardial injury, ultrasound

Case presentation

A 52-year-old male marathon runner was found collapsed at the sixteenth kilometre of a half marathon (21 km) on a hot spring day in Piacenza (Po Valley, Italy). He was brought to our emergency department (ED) by the Emergency Medical Service. He was confused, disoriented, repetitive, and amnesic. He appeared exhausted, pale, dehydrated, and sweating profusely. He denied chest pain, shortness of breath, and cramps, but he complained of severe asthenia, headache, nausea, and several episodes of vomiting gastric juice (one at the time of his collapse, one during the ambulance ride, and one when arriving in the ED). His medical history was not noteworthy. He denied any allergies or medications. He reported having never drunk water while running the marathon and having been fasting since the night before.

At the arrival in the ED the recorded tympanic temperature was 38.7 °C, blood pressure 90/60 mmHg,

heart rate 140 bpm, pulse oximetry 98% at room ambient, and respiratory rate 25/min. An electrocardiogram (ECG) showed a supraventricular tachycardia, probably an atrial flutter with a 2:1 block (resulting in a ventricular rate of ~150 bpm), conducted with a left bundle branch block (figure 1). A point-of-care ultrasound revealed a hyperkinetic cardiac pattern with a fully collapsed inferior vena cava, a significantly smaller right ventricular diameter, and a normal left ventricular wall, and excluded pericardial effusion, paradoxical movements of the interventricular septum, an intimal flap, and dilatation of the aortic root. Lung ultrasound showed a normal A pattern without pleural effusion. Abdominal US was normal.

Based on these data, exertional heatstroke (EHS) with myocardial injury was diagnosed, and the patient was started on aggressive fluid management (3500 mL crystalloids in 3 hours) and rapid external cooling with ice packs all over his body, particularly under the groin, axillae, and back of the neck. After 10 minutes, the body temperature was 36.5 °C, the ECG returned to normal (figure 2), and the patient started to feel better, with the exception of the amnesic deficit and persistent nausea treated with metoclopramide (10 mg iv) and levosulpiride (50 mg iv).

The venous blood gas analysis showed a slight respiratory alkalosis with pH 7.43 (normal value 7.32–7.42), pCO2 24.4 mmHg (normal value 41–51), pO2 59 mmHg (normal value 20–40), a significantly increased value of lactate of 77 mg/dL (normal value 5–15), acute renal damage with creatinine 1.6 mg/dL (normal value 0.6–1.20), HCO3 19.4 (normal value 22–26), base excess -8.1, normal electrolytes and glucose.

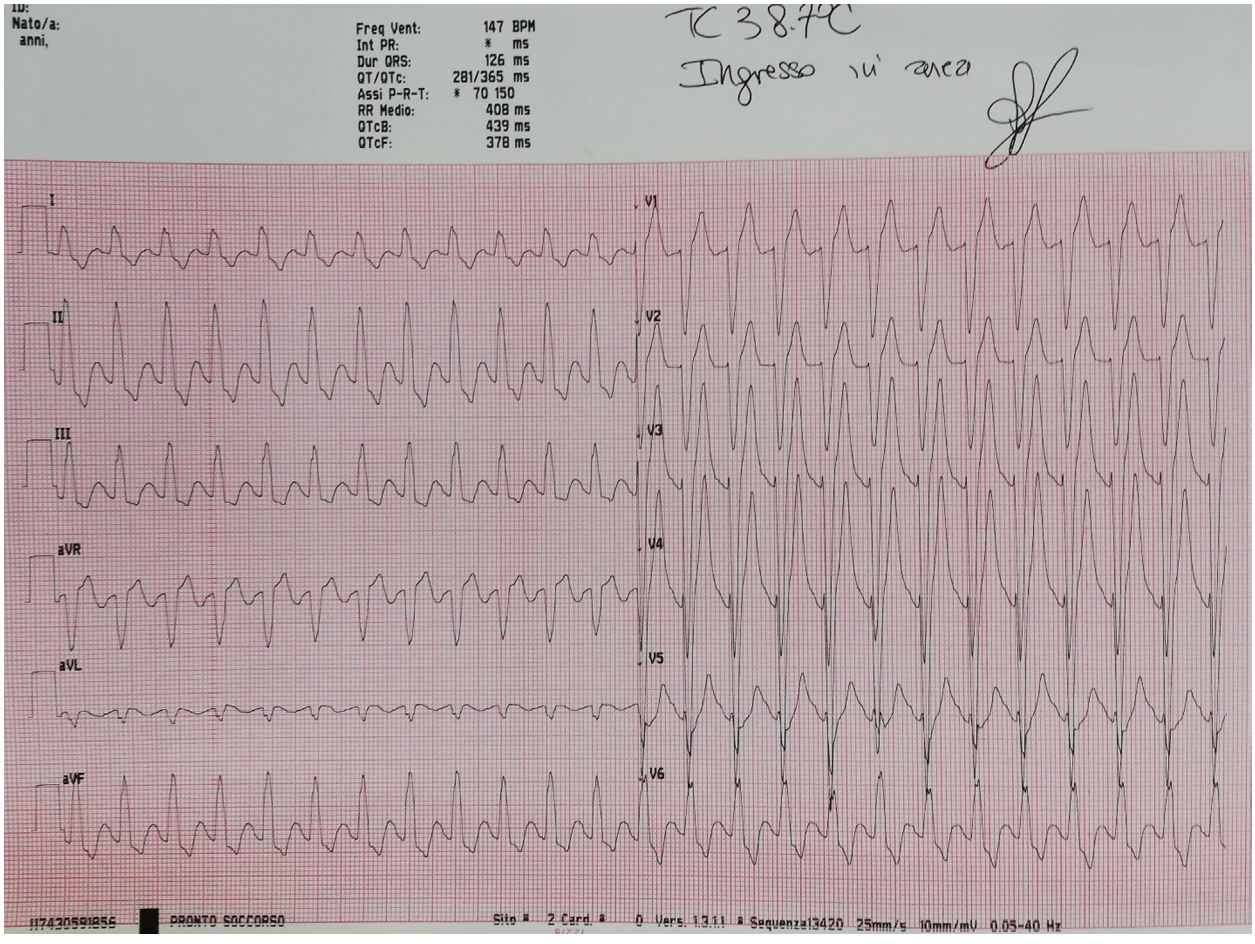

Figure 1. ECG at admission in the ED showing a supraventricular tachycardia, probably an atrial flutter with a 2:1 block (ventricular rate of 150 bpm), conducted with a left bundle branch block.

The laboratory results are reported in table 1 and showed significantly increased values of high-sensitivity cardiac troponin I (hsTnI) (220.6 ng/L, normal value < 12; Beckman coulter), a slight increase of creatine kinase (307 U/L) and transaminases (ALT 39 U/L, AST 59 U/L), and acute renal failure with creatinine of 1.77 mg/dL and eGFR of 46 mL/min/1.73 m². We repeated the hsTnI assay at 2 and 3 hours, which was 310 ng/L and 757 ng/L, respectively, while the patient was not complaining of chest pain or shortness of breath, and ECG and POCUS changes were not found. A chest X-ray and a CT scan of the brain were performed and were both normal.

The patient was admitted to the Cardiovascular Intensive Care Unit (CICU) for further evaluation and monitoring. He underwent coronary angiography, which showed a non-significant coronary artery disease, and an electrophysiological study, that excluded inducible arrhythmias. Echocardiography did not show evidence of wall motion abnormalities and confirmed a normal left ventricular ejection fraction of about 60%. He was treated with continuous hydration and careful monitoring of his electrolyte balance and urine output. HsTnI fell back to near-normal levels (27.8 ng/L) after 72 hours of admission (table 1). On day 4, the patient was discharged in good clinical condition but remained amnesic for the event. At discharge, the ECG was normal with a sinus rhythm (69 bpm).

Discussion

Sustained exercise for several hours is associated with heat generation, high circulating lactate concentration, and depletion of energy as adenosine triphosphate (1), which contribute to several exercise-related medical conditions (2), including exercise-associated collapse, EHS or hypothermia, and cardiovascular

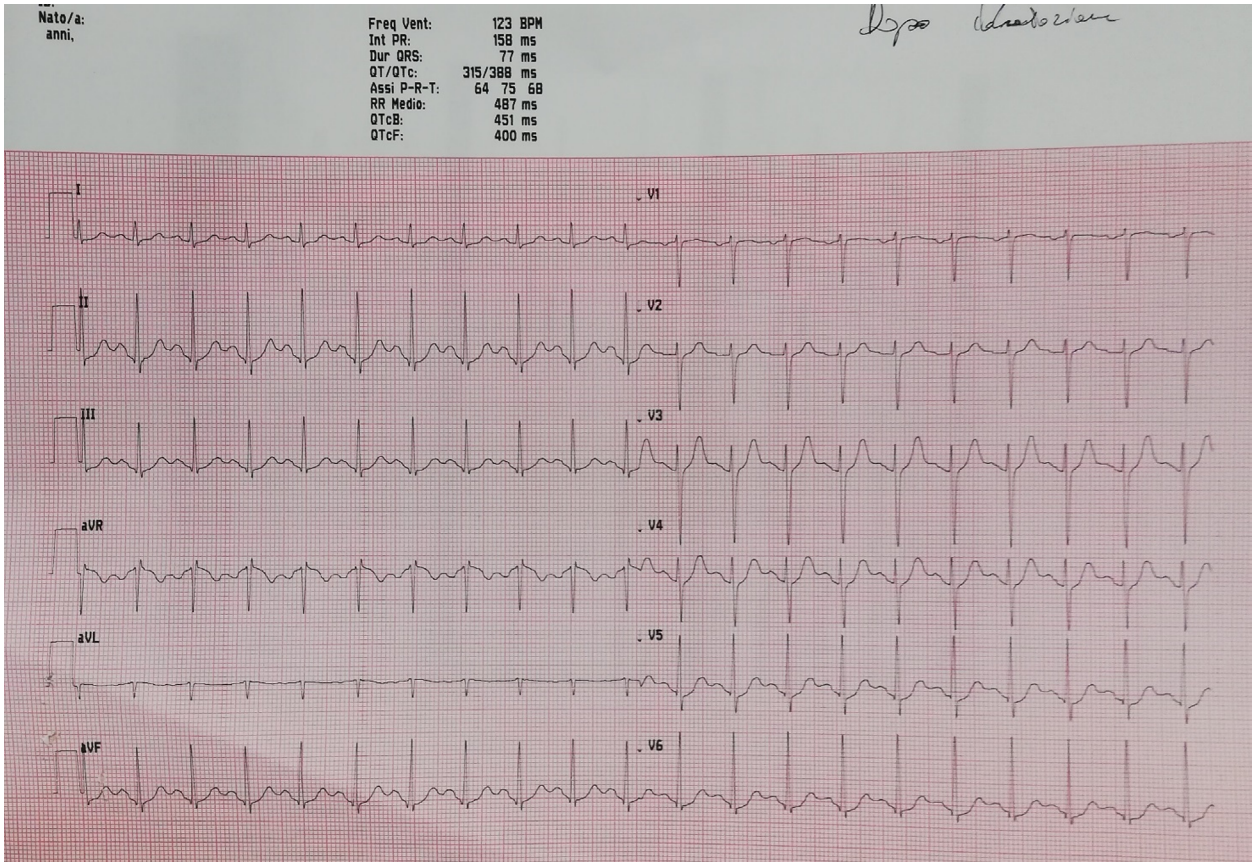

Figure 2. Repeat ECG after hydration and rapid external cooling showing a normal heart rate and the complete resolution of the left bundle branch block.

Table 1. Laboratory findings at admission in the ED, during the recovery in the CICU, and at discharge.

Variable	Reference range	At admission	In CICU	At discharge
hsTnI (ng/L)	0 – 20	**220.6**	**757**	**27.8**
CK (U/L)	0 – 172	**307**	**844**	150
Urea (mg/dL)	10 – 50	**59**	**59**	38
Creatinine (mg/dL)	0.6 – 1	**1.77**	0.84	0.87
Sodium (mEq/L)	135 – 146	144	145	138
Potassium (mEq/L)	3.6 – 5	4.2	3.8	3.8
Clorum (mEq/L)	97 – 110	106	105	102
CRP (mg/dL)	0 – 0.5	0.07	ND	**0.8**
LDH (U/L)	0 – 248	**285**	**290**	ND
AST (U/L)	10 – 37	**59**	**77**	ND
ALT (U/L)	10 –37	**39**	**65**	ND
Glucose (mg/dL)	74 – 100	**115**	83	**105**

High-sensitivity cardiac troponin I (hsTnI); CK, creatine kinase; CRP, C-reactive protein; LDH, lactate dehydrogenase; AST, aspartate transaminase; ALT, alanine transaminase; ND, not determined.

morbidity with sudden cardiac arrest in predisposed individuals.

Considering our patient, he presented with collapse, defined as the inability to walk unassisted, and was extremely confused with an altered mental status and a high body temperature, as occurs in EHS.

EHS is a potentially lethal condition defined by a rectal temperature > 40.5 °C with symptoms or signs of neurological dysfunction and progressive multiple end-organ damage (3, 4).

The precise incidence of EHS is underestimated. Long-distance road races reported an EHS incidence ranging between 1.6 and 2.13 per 1000 finishers without mortality (5, 6).

The first symptoms are usually confusion, drowsiness, or seizures, which can rapidly worsen with delirium and coma, resulting in cardiac arrest (7, 8). Hypotension, tachypnea, vomiting, diarrhea, and biochemical biomarkers indicating mild-to-moderate rhabdomyolysis, acute kidney, liver, heart injury, and coagulopathy are frequent at the onset (8). Hyperthermia with a core temperature >40°C, with neurocognitive dysfunction, is diagnostic — however, cutaneous vasoconstriction may mean that a peripheral temperature measurement is inaccurately low. A core temperature measurement (using a rectal thermometer) is required on any collapsed athlete (9).

The pathogenesis is not entirely clear. Recent reviews have proposed a three-phase mechanism, comprising an acute phase of hyperthermia and neurological disturbances, followed 24–48 hours later by a hematologic–enzymatic phase of inflammation and coagulopathy. The last phase occurs 96 hours after onset and consists of liver and kidney injury (8).

EHS is fatal if it is not recognized and treated promptly within an hour of presentation (the "golden hour"). The treatment is aggressive cooling with immersion in iced or cold water in portable water impermeable bags (9, 10). If not possible, cold towels immersed in ice water must be placed in the groin, neck, and axillae until the rectal temperature, measured every 15 minutes, is less than 38 °C. Effective drugs for EHS do not exist, and dantrolene sodium is ineffective (11, 12). The patient must be brought to the hospital, keeping him cool while in the ambulance, and once in the ED, he must be reassessed every 15-20 minutes if therapy does not result in a rapid drop in temperature or an improvement in the patient's mental condition. Since heat injury inhibits gluconeogenesis, blood sugar levels should always be checked. If the patient cannot drink or has continuing fluid loss from vomiting and/or diarrhea, or has significant hyperthermia, intravenous fluids must be administered if hyponatremia has been excluded ("water intoxication") (13). Up to 1 Liter

i.v. saline solution can be given over 20–30 minutes and may be resumed if clinical signs of hypovolemia persist. The negative prognosis of EHS is strictly related to coma or aggressiveness and disorientation, severe hypotension and tachycardia, rectal temperature > 40 °C and not responding to treatment, chest pain or rapid irregular heart rate, signs of stroke, worsening cramps, rigors, headache, malaise, and a deteriorating level of consciousness.

According to the current literature, the electrocardiographic changes and elevation of cardiac markers observed in patients with EHS do not reflect cardiac ischemia but rather a pathophysiological response (14). The most common changes in the ECG are sinus tachycardia, conduction defect, ischemic ST-T changes, and non-specific changes. Hemodynamic studies showed a high cardiac index and low systemic vascular resistance in EHS patients, consistent with hyperdynamic circulation (8). As reported by Watanabe *et al.*, the combination of dehydration and hyperthermia markedly impaired stroke volume and cardiac output due to reduced left ventricular filling. The decreased preload of the heart is in turn associated with concomitant hypovolemia and reduced venous return, possibly due to blunted blood perfusion induced by enhanced peripheral vasoconstriction and diminished cardiac filling time accompanying tachycardia. In contrast, impaired intrinsic myocardial contractility and relaxation do not appear to be the factors responsible for the cardiovascular strain accompanying progressive dehydration and hyperthermia during prolonged exercise in the heat. These findings highlight the importance of peripheral mechanisms in cardiac performance during intense exercise (15), as previously suggested by González-Alonso *et al.* (16).

Conclusions

EHS is a life-threatening medical condition with a complex pathophysiology involving thermoregulatory failure with a progressive heat-related organ injury that can have fatal consequences. The diagnosis is not always straightforward, and further studies are needed to clearly understand the pathogenesis. With this case report, we want to highlight the importance of early recognition of EHS, even in regions with a Mediterranean climate, and of timely targeted management to avoid irreversible organ damage. "The golden hour" treatment with rapid cooling and fluid administration dramatically changed our patient's prognosis, allowing full recovery.

Informed Consent: Written informed consent was obtained from the patient concerned.

Ethic Committee: As this was a descriptive case report and data was collected without patient identifiers, ethics approval was not required under our hospital's Institutional Review Board guidelines.

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g., consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

Authors Contribution: EP collected details of the case. EP and CG drafted the manuscript. EP, GV, LR, AB and AC cared for the patient. GC, BB, NS, LR and AV critically revised the manuscript. All authors approved the final version and stated the integrity of the whole work.

References

1. Dunn J, Grider MH. Physiology, Adenosine Triphosphate. (Updated 2023 Feb 13). In: StatPearls (Internet). Treasure Island (FL): StatPearls Publishing; 2023.
2. Trangmar SJ, González-Alonso J. Heat, Hydration and the Human Brain, Heart and Skeletal Muscles. Sports Med. 2019;49(Suppl 1):69-85. doi:10.1007/s40279-018-1033-y.
3. Lawrence E. Armstrong. In: Heat Exhaustion. Exertional Heat Illness; 2020. pp. 81-115.
4. Garcia CK, Renteria LI, Leite-Santos G, Leon LR, Laitano O. Exertional heat stroke: pathophysiology and risk factors. BMJ Med. 2022;1(1):e000239. doi:10.1136 /bmjmed-2022-000239.
5. Breslow RG, Shrestha S, Feroe AG, et al. Medical tent utilization at 10-km road races: injury, illness, and influencing factors. Med Sci Sports Exerc. 2019;51:2451–7. doi:10.1249/MSS.0000000000002068.
6. Demartini JK, Casa DJ, Stearns R, et al. Effectiveness of cold water immersion in the treatment of exertional heat stroke at the Falmouth road race. Med Sci Sports Exerc. 2015;47:240–5. doi:10.1249/ MSS.0000000000000409.

7. Currie A, McDuff D, Johnston A, et al. Management of mental health emergencies in elite athletes: a narrative review. British Journal of Sports Medicine. 2019;53:772-778.
8. Yezli, S, Yassin Y, Ghallab,S, et al. Classic heat stroke in a desert climate: A systematic review of 2632 cases. Journal of internal medicine. 2023; 294(1):7-20. doi.org/10.1111/joim.13633.
9. Walter E, Steel K. Management of exertional heat stroke: a practical update for primary care physicians. Br J Gen Pract. 2018;68(668):153-154. doi: 10.3399/bjgp18X695273.
10. DeGroot DW, O'Connor FG, Roberts WO. Exertional heat stroke: an evidence-based approach to clinical assessment and management. Exp Physiol. 2022;107(10):1172-1183. doi:10.1113/EP090488.
11. Bouchama A, Cafege A, Devol EB, Labdi O, el-Assil K, Seraj M. Ineffectiveness of dantrolene sodium in the treatment of heatstroke. Crit Care Med. 1991;19(2):176-180. doi:10.1097/00003246-199102000-00011.
12. Channa AB, Seraj MA, Saddique AA, Kadiwal GH, Shaikh MH, Samarkandi AH. Is dantrolene effective in heat stroke patients? Crit Care Med. 1990;18(3):290-292. doi:10.1097/00003246-199003000-00009.
13. Murray R. Rehydration strategies--balancing substrate, fluid, and electrolyte provision. Int J Sports Med. 1998;19 Suppl 2:S133-S135. doi:10.1055/s-2007-971978.
14. Egaimi M, Seo H, Bashir S. A Case of Heat Exhaustion Masquerading as ST-Elevation Myocardial Infarction. Cureus. 2022;14(10):e30495. doi:10.7759/cureus.30495.
15. Watanabe K, Stöhr EJ, Akiyama K, Watanabe S, González-Alonso J. Dehydration reduces stroke volume and cardiac output during exercise because of impaired cardiac filling and venous return, not left ventricular function. Physiol Rep. 2020;8(11):e14433. doi:10.14814/phy2.14433.
16. González-Alonso J, Mora-Rodríguez R, Below PR, Coyle EF. Dehydration markedly impairs cardiovascular function in hyperthermic endurance athletes during exercise. J Appl Physiol. 1997;82(4):1229-1236. doi:10.1152/jappl.1997.82.4.1229.

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Received: 15 June 2023
Accepted: 15 July 2023
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