Case Report / Olgu Sunumu

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Can Bullying Stop a Child's Heart? A Rare Case of Commotio Cordis and Hypoxic-ischemic Encephalopathy

Çocukta Zorbalık Kalbi Durdurabilir mi? Komotio Kordis ve Hipoksik-iskemik Ensefalopatiye Nadir Bir Olgu

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Abstract

Commotio cordis is a rare but potentially fatal condition characterized by sudden cardiac arrest following blunt, non-penetrating chest trauma, most commonly in adolescent males. Mortality is particularly high when the event occurs outside organized sports settings, where delayed cardiopulmonary resuscitation (CPR) and lack of immediate defibrillation are more common. A previously healthy 14-year-old male collapsed immediately after receiving a blunt chest impact caused by peer aggression at school. Bystander CPR was initiated, and return of spontaneous circulation was achieved after 25 minutes, following multiple defibrillation attempts. Despite stabilization of cardiac rhythm in the pediatric intensive care unit, the patient developed hypoxic-ischemic encephalopathy secondary to ventricular fibrillation-induced arrest, resulting in severe, persistent neurological sequelae, including spasticity and severe motor impairment. The Glasgow Coma scale score was 5, and the modified Rankin Scale score remained 5 at follow-up. Mortality and morbidity rates are higher in non-sport-related commotio cordis cases due to delayed recognition, limited access to automated external defibrillators (AEDs), and low public awareness. Early intervention significantly impacts both survival and neurological outcomes. This case highlights the potential severity of commotio cordis triggered by bullying and emphasizes the importance of rapid resuscitative efforts. Early diagnosis, prompt CPR, and timely defibrillation reduce mortality from commotio cordis. Post-arrest neurological monitoring is crucial for managing hypoxic complications. Implementing CPR training and ensuring AED availability in schools may reduce morbidity and improve survival in non-sport-related cases.

Keywords: Commotio cordis, bullying, hypoxic-ischemic encephalopathy, cardiopulmonary resuscitation, pediatric intensive care

Öz

Komotio kordis, nadir ancak potansiyel olarak ölümcül bir durum olup genellikle ergen erkeklerde görülen, künt ve penetrasyon yapmayan göğüs travmasına bağlı ani kardiyak arrest ile karakterizedir. Olayın spor dışı ortamlarda meydana gelmesi, kardiyopulmoner resüsitasyonun (CPR) gecikmesi ve anında defibrilasyonun olmaması nedeniyle mortalitenin özellikle yüksek olduğu durumları oluşturur. Daha önce sağlıklı olan 14 yaşında bir erkek çocuk, okulda akran saldırısına bağlı göğsüne aldığı künt darbe sonrasında ani olarak yere yığıldı. Çevrede bulunan kişiler tarafından CPR başlatıldı ve birden fazla defibrilasyon girişimi sonrasında 25 dakika içinde spontan dolaşım geri döndü. Çocuk yoğun bakım ünitesinde kardiyak ritim stabilizasyonu sağlanmasına rağmen hastada ventriküler fibrilasyona bağlı arrest sonucunda hipoksik-iskemik ensefalopati gelişti; bunun sonucu olarak spastisite ve ciddi motor bozukluk gibi kalıcı nörolojik sekeller ortaya çıktı. Takipte Glasgow Koma skalası 5, modifiye Rankin skalası skoru ise 5 olarak kaldı. Spor dışı komotio kordis olgularında mortalite ve morbidite, tanının gecikmesi, otomatik eksternal defibrilatöre (AED) sınırlı erişim ve düşük kamu farkındalığı nedeniyle daha yüksektir. Erken müdahale hem hayatta kalma oranını hem de nörolojik sonucu önemli ölçüde etkiler. Bu olgu, zorbalık kaynaklı komotio kordisin potansiyel ciddiyetini göstermekte ve hızlı resüsitatif girişimlerin önemini vurgulamaktadır. Komotio kordiste erken tanı, hızlı CPR ve zamanında defibrilasyon mortaliteyi azaltır. Arrest sonrası nörolojik takip, hipoksik komplikasyonların yönetimi açısından kritik öneme sahiptir. Okullarda CPR eğitiminin uygulanması ve AED erişiminin sağlanması, spor dışı olgularda morbiditeyi azaltabilir ve hayatta kalmayı artırabilir.

Anahtar Kelimeler: Komotio kordis, zorbalık, hipoksik-iskemik ensefalopati, kardiyopulmoner resüsitasyon, çocuk yoğun bakım

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Introduction

Commotio cordis is a rare yet catastrophic clinical entity defined by sudden cardiac arrest following blunt, non-penetrating trauma to the precordial region. It is most frequently in adolescent males aged 10-18 years.[1] Affected individuals typically have structurally normal hearts, with the event precipitated by the abrupt onset of ventricular fibrillation. The pathophysiological mechanism is attributed to mechanical activation of mechano-sensitive ion channels occurring during a narrow vulnerable window of ventricular repolarization, particularly the ascending phase of the T wave, which precipitates malignant ventricular arrhythmias.[2,3]

The diagnosis is primarily based on a clear temporal association between chest trauma and cardiac arrest, supported by electrocardiographic findings and the exclusion of alternative causes of sudden cardiac arrest. Notably, non-sport-related cases—including those arising from daily activities, interpersonal violence, or bullying—have been associated with significantly higher mortality rates.[4] Early initiation of cardiopulmonary resuscitation (CPR) and prompt defibrillation remain the most critical determinants of survival, as mortality may reach 80-90% in the absence of timely intervention.[5]

We report a rare case of commotio cordis precipitated by school bullying and complicated by hypoxic-ischemic encephalopathy, highlighting the potentially devastating cardiac and neurological consequences of interpersonal physical aggression in pediatric populations.

Case Report

A previously healthy 14-year-old male collapsed immediately after sustaining a blunt chest impact from a peer at school. There was no personal or family history of syncope, sudden cardiac death, inherited arrhythmia syndromes, cardiovascular disease, or substance use. School staff immediately activated emergency medical services, and the sequence of events is summarized in Table 1.

On initial assessment, the patient was pulseless. Basic life support was initiated by trained school personnel, followed by advanced life support upon the arrival of emergency medical services. Endotracheal intubation was performed, and the patient was transferred under continuous cardiac monitoring.

During transport, approximately 15 minutes after the event, ventricular fibrillation was documented. Following sequential defibrillation attempts and intravenous administration of amiodarone, return of spontaneous circulation (ROSC) was achieved after the fourth shock at minute 25 (Table 1, Figure 1).

Upon admission to the pediatric intensive care unit (PICU), the Glasgow Coma scale (GCS) score was 4 while the patient was sedated. Pupillary light reflexes were bilaterally diminished, with absent motor responses and marked spasticity, corresponding to a modified Rankin scale score of 5. Vital signs on admission were as follows: blood pressure 112/72 mmHg, heart rate 136 beats/min, respiratory rate 23 breaths/min, and oxygen saturation 97%. No external signs of thoracic trauma were observed.

Electrocardiography revealed sinus rhythm with a corrected QT interval of 0.39 seconds and no ischemic changes. Transthoracic echocardiography demonstrated a structurally normal heart with a left ventricular ejection fraction of 55%. Computed tomography of the chest, abdomen, brain, and cervical spine showed no acute pathological findings.

Laboratory evaluation revealed markedly elevated troponin T levels (651 ng/L; reference range 0-14 ng/L) and leukocytosis (20.76×10^3/μL). An arterial blood gas analysis indicated metabolic acidosis (pH 7.30; HCO_3^- 17.4 mmol/L; base excess -9.1). Other hematological and biochemical parameters were within normal limits (Table 2). The elevated troponin level was considered secondary to global myocardial ischemia related to cardiac arrest and repeated defibrillation rather than structural myocardial injury, given the absence of ischemic electrocardiographic changes and normal echocardiographic findings.

Therapeutic management included continuous lidocaine infusion (20 μg/kg/min), hyperosmolar therapy with 3% sodium chloride and mannitol, and antiepileptic treatment. Strict normothermia was maintained as part of post-cardiac arrest neuroprotective care. Serial electrocardiograms remained unremarkable, and 24-hour Holter monitoring revealed no arrhythmias.

Table 1. Timeline of clinical events and resuscitation following blunt chest trauma

Time (min)	Intervention	Details
0	Chest impact	Blunt precordial trauma due to peer bullying; immediate loss of consciousness
0-10	CPR initiated	Basic life support provided by teachers, followed by advanced life support by EMS
10	Hospital transfer	Transported to district state hospital under continuous monitoring
10-25	ACLS	Endotracheal intubation, four defibrillation attempts, intravenous amiodarone administered
25	ROSC	ROSC achieved; sinus rhythm restored

CPR: Cardiopulmonary resuscitation, ACLS: Advanced cardiac life support, ROSC: Return of spontaneous circulation, EMS: Emergency medical services

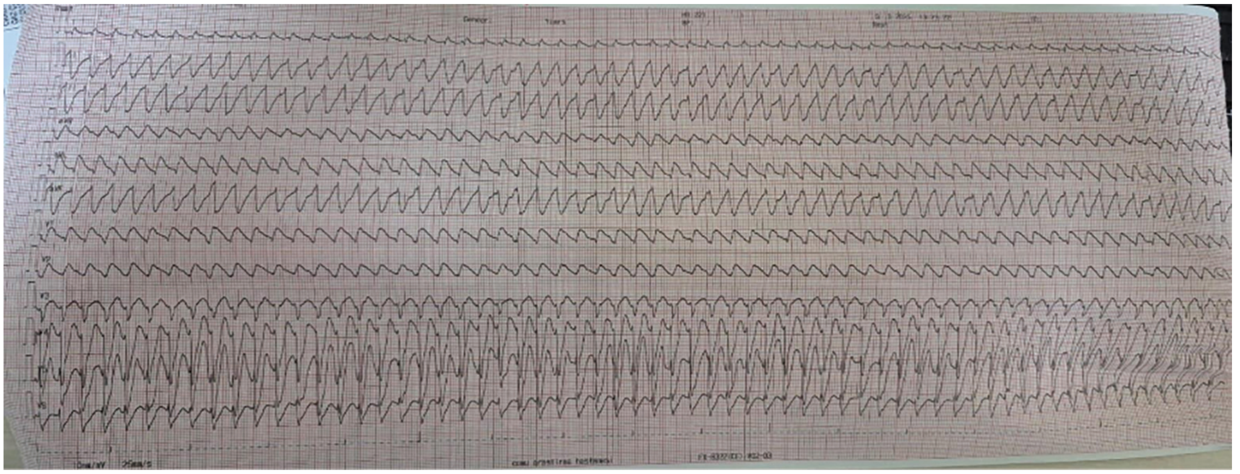

Figure 1. Electrocardiographic recording demonstrating ventricular fibrillation obtained during hospital evaluation
Continuous cardiac monitoring demonstrated ventricular fibrillation characterized by wide-complex, irregular, high-frequency electrical activity. Following sequential defibrillation, sinus rhythm was successfully restored

Table 2. Laboratory findings at PICU admission			
Parameter	**Result**	**Normal range**	**Comment**
Hemoglobin (g/dL)	15.6	12-16	Normal
Leukocytes (×10^3/µL)	20.76	4-11	Stress-related leukocytosis
Platelets (×10^3/µL)	416	150-450	Normal
Troponin T (ng/L)	651	0-14	Elevated, likely secondary to cardiac arrest
PT (INR)	1.24	0.8-1.2	Mild prolongation
pH	7.30	7.35-7.45	Acidemia
HCO_3^- (mmol/L)	17.4	22-26	Metabolic acidosis
Base excess (mmol/L)	-9.1	-2 to +2	Base deficit

PICU: Pediatric intensive care unit, PT: Prothrombin time, INR: International normalized ratio, HCO_3^-: Serum bicarbonate

On the fifth hospital day, brain magnetic resonance imaging demonstrated diffuse T2-weighted hyperintense signal abnormalities involving the bilateral thalami, basal ganglia, and cortical gray matter, consistent with hypoxic-ischemic encephalopathy (Figure 2). Neurological improvement was limited: the GCS score increased to 5, but severe spasticity and motor deficits persisted. Despite stable cardiac function, profound neurological sequelae remained evident. A summary of the cardiac and neurological evaluations is provided in Supplementary Table 1. Written informed consent for publication of this case report and the accompanying clinical data/images was obtained from the patient's parents.

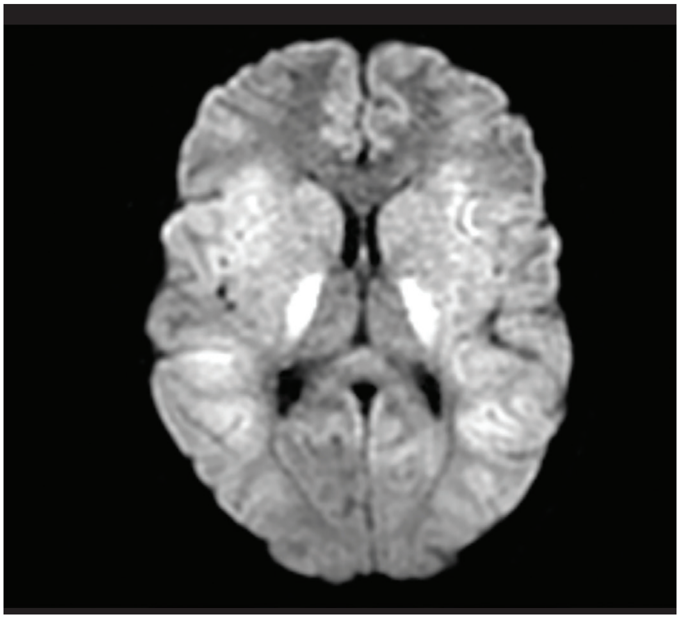

Figure 2. Cranial magnetic resonance imaging (MRI) findings
Brain MRI obtained on hospital day 5 demonstrating diffuse T2-weighted hyperintense signal abnormalities involving the bilateral thalami, basal ganglia, and cerebral cortex, consistent with hypoxic-ischemic encephalopathy

Discussion

Although commotio cordis is most commonly associated with athletic activities, approximately one-quarter of reported cases occur in non-sport settings, including routine activities, accidental trauma, and interpersonal violence.[2] These non-sport-related presentations are associated with higher mortality, largely due to delayed initiation of CPR, limited access to defibrillation, and reduced public awareness.

The proposed pathophysiological mechanism involves a sudden increase in left ventricular intracavitary pressure at the moment of chest impact, triggering ventricular fibrillation via mechano-electric feedback.[6] Experimental models have identified a narrow vulnerability window—approximately 10-20 milliseconds during the ascending phase of the T wave—during which the myocardium is particularly susceptible to lethal arrhythmias.[3,6] Survival is highly time-dependent; CPR initiated within the first three minutes is associated with survival rates of 25-40%, whereas delays beyond three minutes reduce survival to as low as 3-5%.[2,7] In the present case, early bystander CPR and repeated defibrillation resulted in ROSC at minute 25, effectively restoring cardiac stability. However, the prolonged low-flow, hypoxic state likely contributed to severe hypoxic-ischemic brain injury, as reflected by diffuse findings on magnetic resonance imaging and persistent neurological deficits. These findings underscore that, even when cardiac recovery is achieved, commotio cordis may be complicated by devastating neurological outcomes—particularly in non-sport-related incidents where recognition and intervention may be delayed.[1]

This case expands the limited literature on bullying-related commotio cordis and emphasizes that seemingly minor interpersonal violence in school settings can result in catastrophic cardiac and neurological outcomes. Increasing CPR literacy among school personnel, ensuring widespread availability of automated external defibrillators (AEDs), and enhancing awareness of bullying-related physical trauma may be lifesaving and reduce long-term morbidity.

The neurological outcomes described are limited to the observed follow-up period, and long-term prognosis could not be fully evaluated. Advanced diagnostic investigations, including genetic testing and detailed electrophysiological studies, were not performed; therefore, underlying channelopathies cannot be entirely excluded. Nevertheless, the clear temporal relationship between chest impact and cardiac arrest, together with normal cardiac imaging and rhythm evaluation, strongly supports the diagnosis of commotio cordis.

Conclusion

Early recognition of commotio cordis, immediate initiation of CPR, and timely defibrillation are essential for reducing mortality. However, prolonged hypoxic-ischemic injury may result in irreversible neurological sequelae despite successful cardiac resuscitation, underscoring the importance of comprehensive PICU management and neuroprotective strategies. Preventive strategies, including structured CPR education and ensuring AED availability in school settings, are crucial to mitigating the impact of non-sport-related commotio cordis.

Ethics

Informed Consent: Written informed consent for publication of this case report and the accompanying clinical data/images was obtained from the patient's parents.

Footnotes

Authorship Contributions

Surgical and Medical Practices: F.B., T.S.A., D.A., Concept: F.B., T.S.A., D.A., Design: F.B., T.S.A., Data Collection or Processing: F.B., T.S.A., D.A., Analysis or Interpretation: F.B., T.S.A., D.A., Literature Search: F.B., T.S.A., Writing: F.B., T.S.A., D.A.

Conflict of Interest: No conflict of interest was declared by the authors.

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Supplementary Table. https://d2v96fxpocvxx.cloudfront.net/1fe4afca-adad-4137-be7c-36a5e5ab6910/content-images/a5b2e7f6-0ab8-466f-9641-03b35695f996.pdf

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