

Kharkiv Karazin National University
Faculty of Medicine
Department of Internal Medicine

BRUGADA SYNDROM E: A CASE REPORT

Reporter: 5th course student - Alagbo Habib O.
Supervisors: MD Shmidt E.Y.



In this Case, we describe a male patient who presented with status epilepticus and typical pattern on 12-lead electrocardiography (ECG) after resuscitation, demonstrating generalized convulsive status epilepticus as the initial manifestation of Brugada syndrome.



Clinical Case

Patient Identifying data

- Age:41 years old
- Sex: male

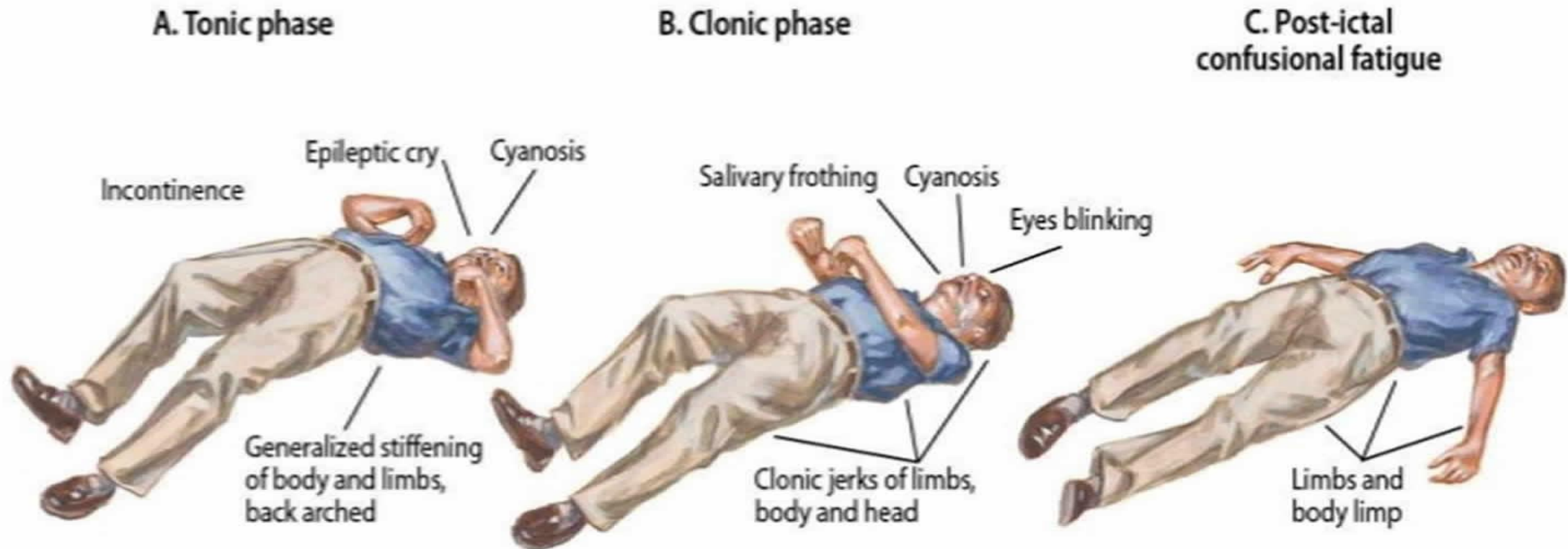


Complaints

Main complaints:

- The patient was brought to the local emergency department (ED) for generalized tonic-clonic movements.

GENERALIZED TONIC-CLONIC SEIZURE



History of the present disease

According to his co-workers, the patient was talking to them without any apparent discomfort, before starting to exhibit tonic-clonic movements that lasted for more than half an hour before arrival to the ED. During resuscitation, VF in ECG records (Figure 1) with seizure activities persisted until direct current (DC) shock was applied and sinus rhythm was restored. The patient was then transferred to the intensive care unit at another hospital as soon as vital signs were stabilized and maintain with medication.





a: ECG recording during the status epilepticus showing ventricular fibrillation.

b: Period of direct current shock.

c: Sinus rhythm was restored after direct current shock was applied.



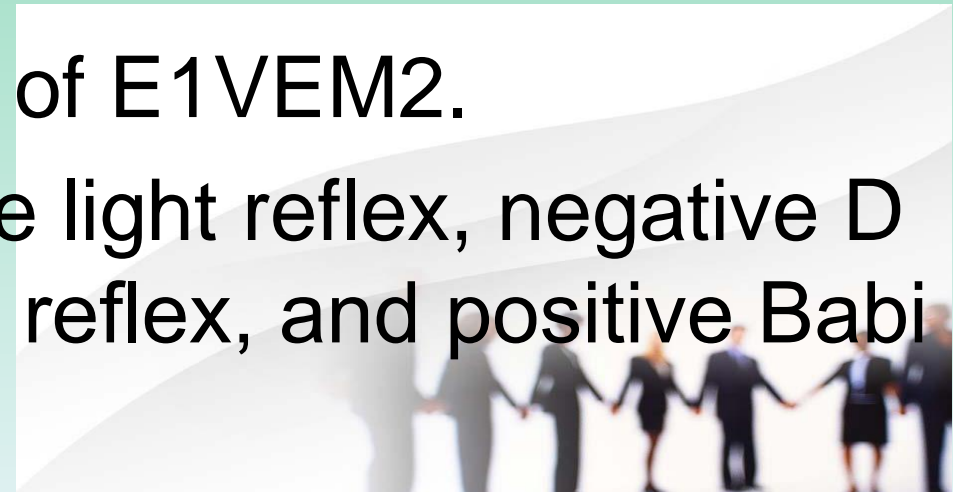
Life history

Patient history revealed no systemic disease or complaints of syncope, convulsion, or chest pain. No drug history or allergic reaction and no surgery or admission records were found. The patient never smoked nor consumed alcohol. There had been no family history of syncope, convulsion, heart disease or sudden death



Physical examination

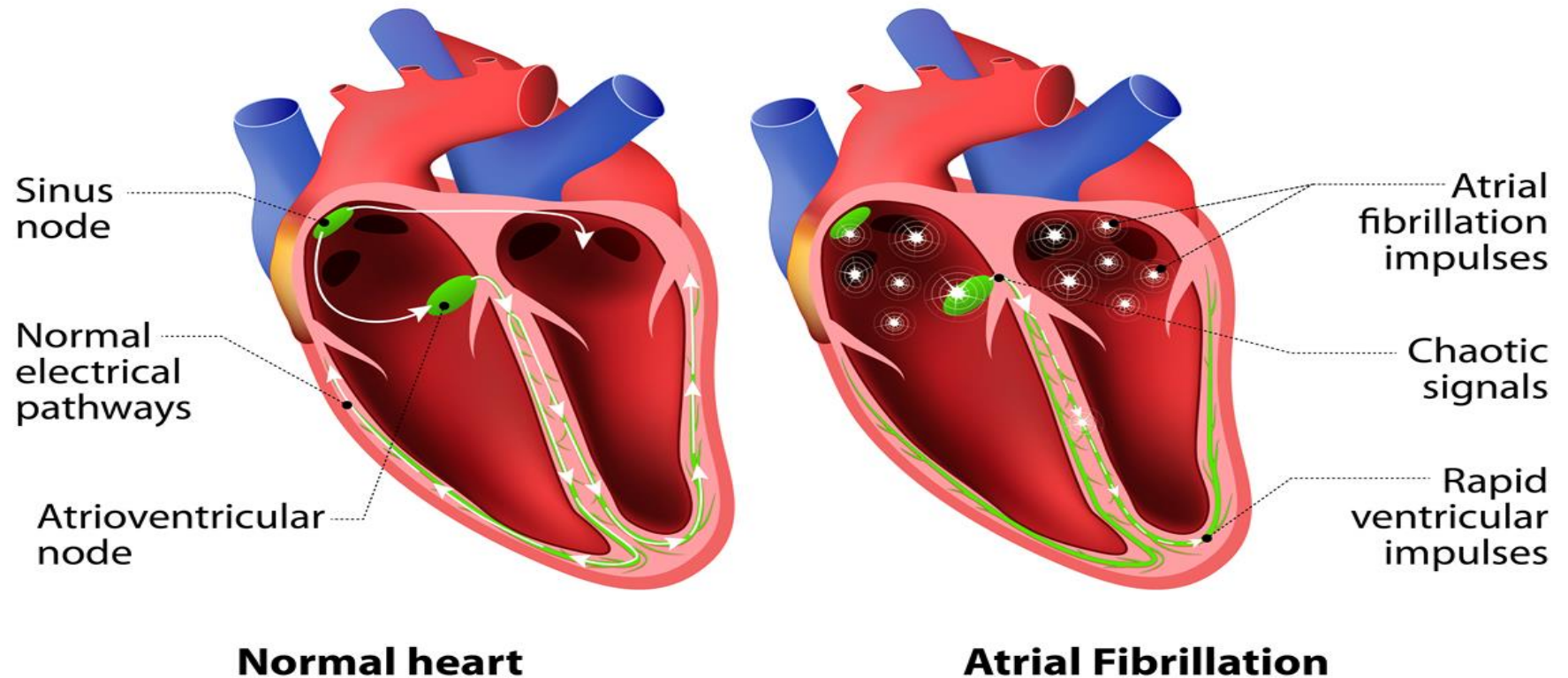
- No physical abnormalities were detected by clinical examination and blood pressure was 130/80 mmHg (on the background of antihypertensive medication) , HR 140 bpm
- Respiratory rate: no significant changes
- Auscultation: clear vesicular sound Over the lungs and present S1 and S2 sounds.
- Abdomen without any changes
- The Glasgow Coma Scale (GCS) status of E1VEM2.
- Neurologic examination revealed positive light reflex, negative Doll's eye sign, hyperreflexic deep tendon reflex, and positive Babinski sign.



Preliminary diagnosis

- Arrhythmia
- Hypoxic encephalopathy

Cardiac arrhythmia



Plan of investigation

- Minimum investigation:
 - Complete blood count
 - Biochemical Blood analysis (Troponin I, CK-MB)
 - ECG
 - Xray
 - CT
 - EchoCG
 - Electroencephalography



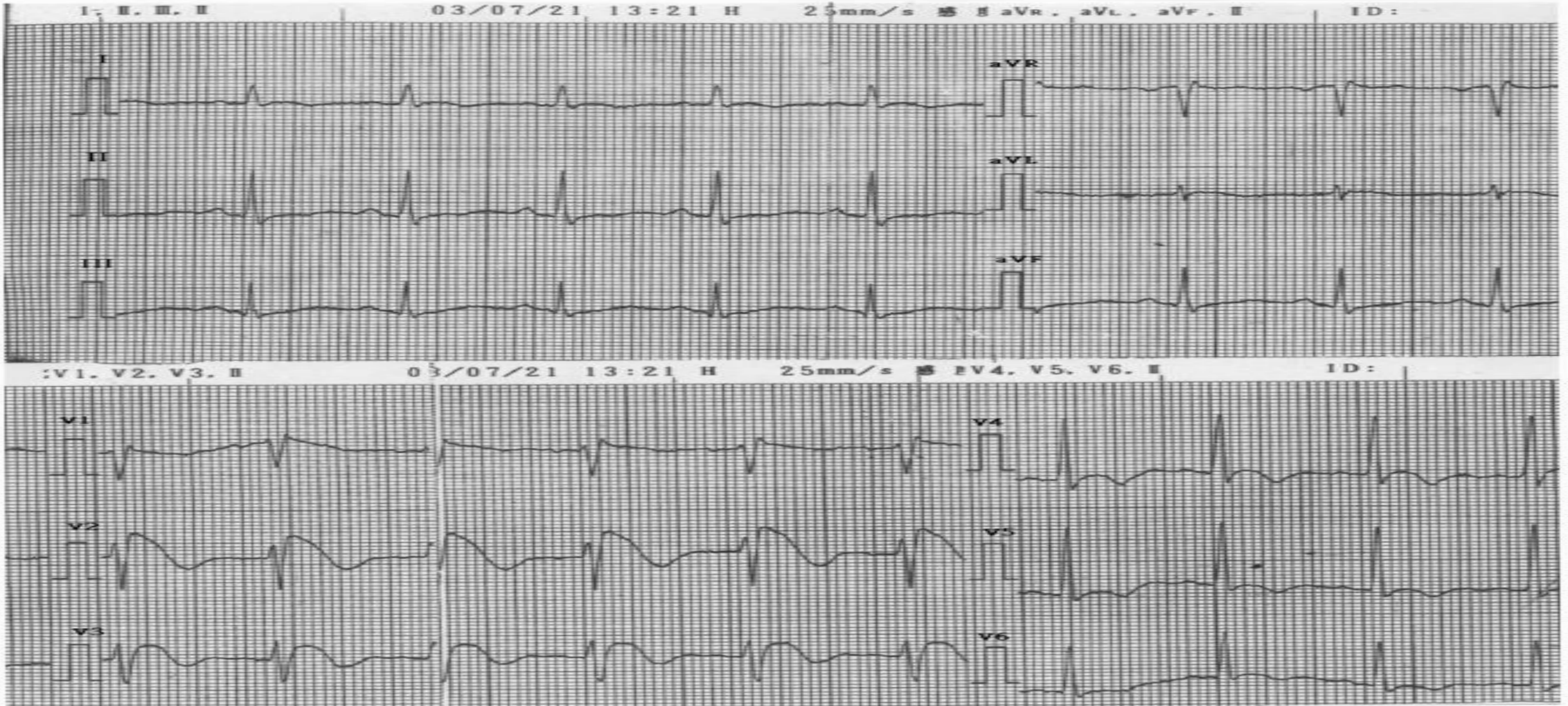
Blood & Biochemical test

The complete blood count and chemistry panel were within reference ranges, except for glutamate-oxalacetate transaminase and glutamate-pyruvate transaminase, which were 94 IU/L and 136 IU/L, respectively. Creatinine phosphokinase and creatinine phosphokinase isoenzyme MB were 378 IU/L and 65 IU/L, respectively. Troponin I was initially 0.123 ng/mL and subsequently peaked at 0.995 ng/mL.



ECG

- Conclusion : Sinus Trachycardia. V1-V3 RSR' pattern and S T Segment elevation



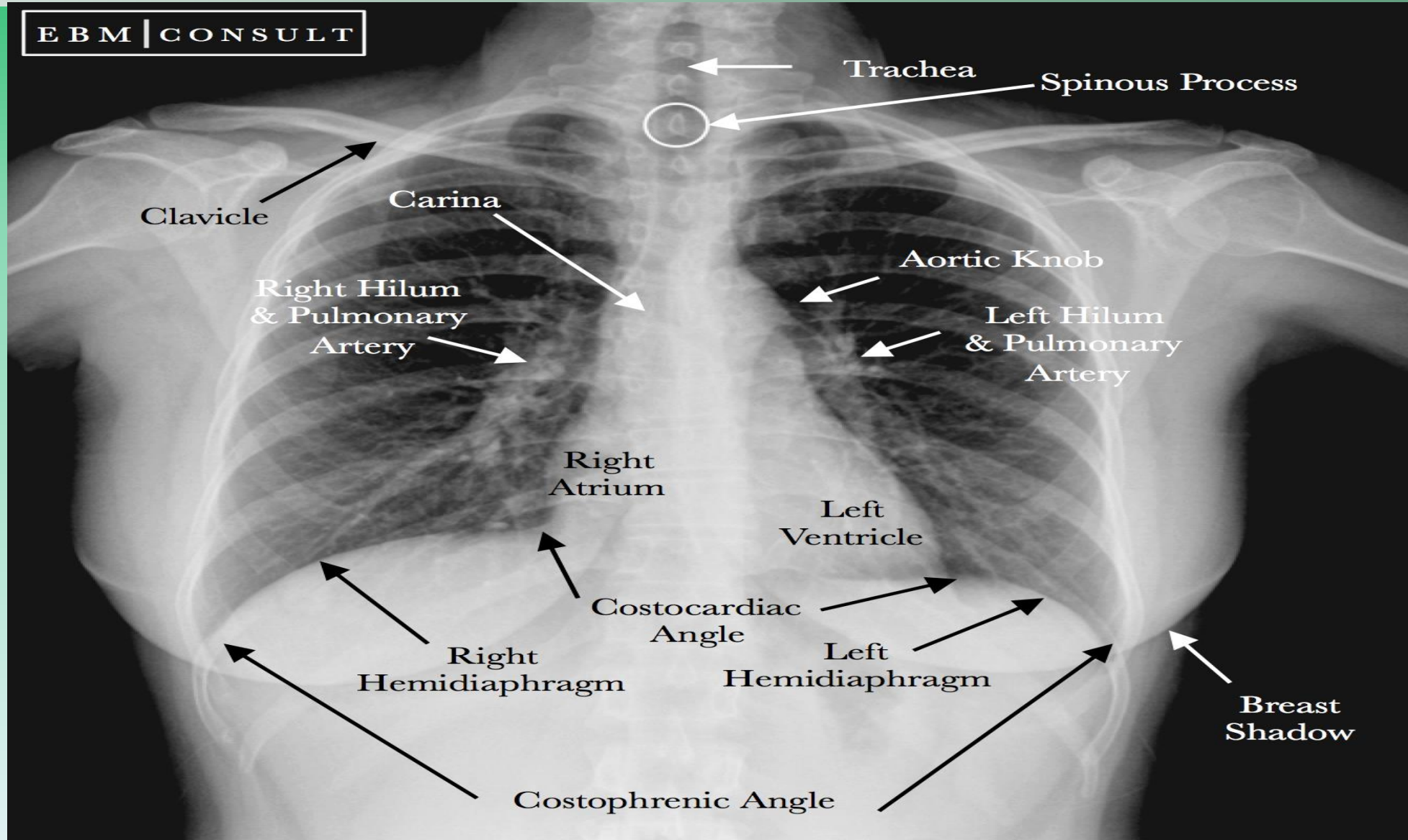
EchoCG

Normal



Chest Xray

- No cardiopulmonary abnormality



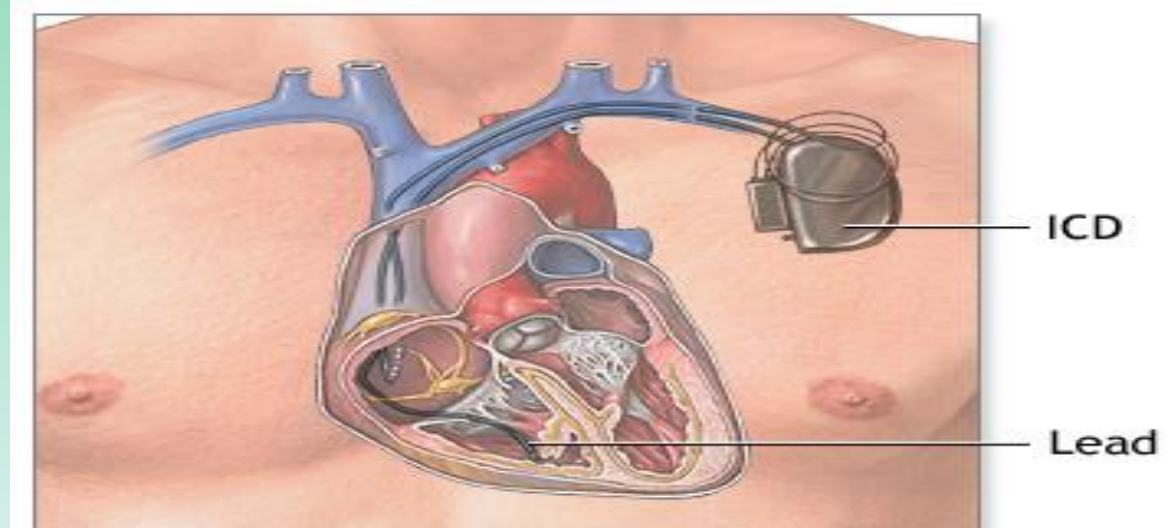
Clinical Diagnosis

- Main disease: Brugada Syndrome
- Concomitant diseases: Hypoxic encephalopathy



Treatment

- Supportive treatment was arranged for the patient, including tracheostomy, stress ulceration prevention and rehabilitation.
- Follow up plan: Holter monitoring, repeat Xray and labs, and serial ECGs
- Surgical: implantable cardioverter-defibrillator (ICD) implantation



An implantable cardioverter-defibrillator (ICD) detects a rapid heartbeat coming from the bottom of the heart

Follow up

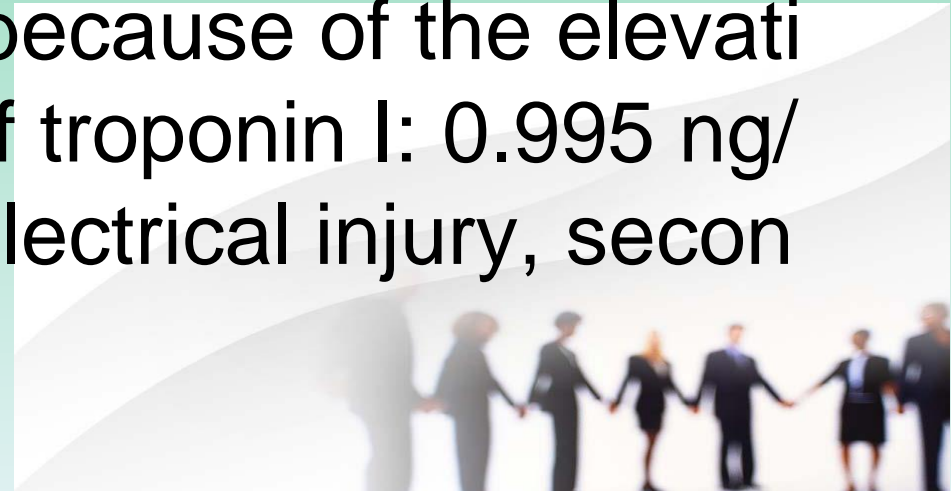
Three weeks later, Physical examination was entirely normal. Laboratory evaluations remained within reference ranges. Repeat chest plain film revealed no active cardiopulmonary lesions. Serial ECGs continued to show RSR' pattern with ST-segment elevation in V1 through V3. There were no other abnormalities on Holter monitoring

The patient continued in rehabilitation programs, which included passive range of motion, pressure sore and thrombosis prevention, oral hygiene education and aspiration pneumonia prevention, and contracture prevention by orthoses. The patient received implantable cardioverter-defibrillator (ICD) implantation subsequently, and is currently doing well.



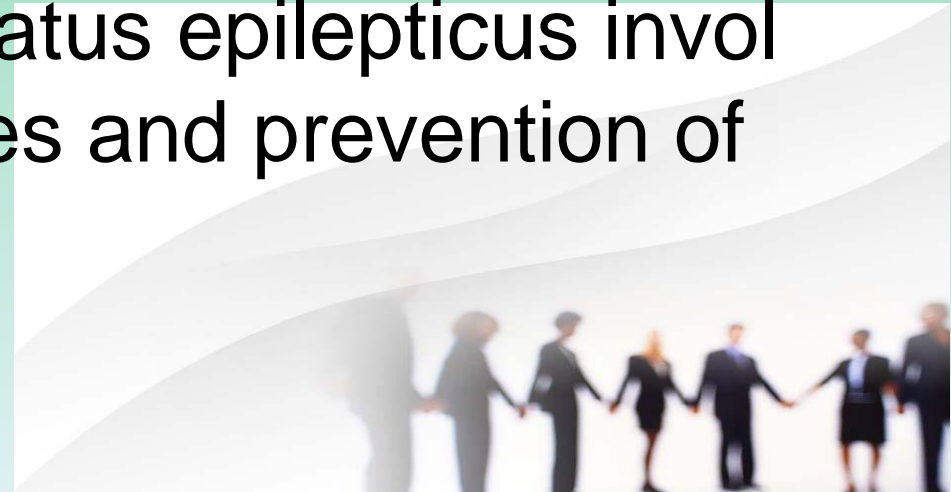
Conclusion

In the present case, the patient had been in perfect health when he suddenly presented in status epilepticus. ECG on arrival revealed ventricular fibrillation, and sequential series revealed Brugada-type patterns. These signs, combined with normal echocardiogram, were supportive evidence for the diagnosis of Brugada syndrome. Although we cannot exclude the possibility of myocardial infarction because of the elevation in cardiac enzyme (the peak level of troponin I: 0.995 ng/mL), this was most likely the result of electrical injury, secondary to DC Shock.



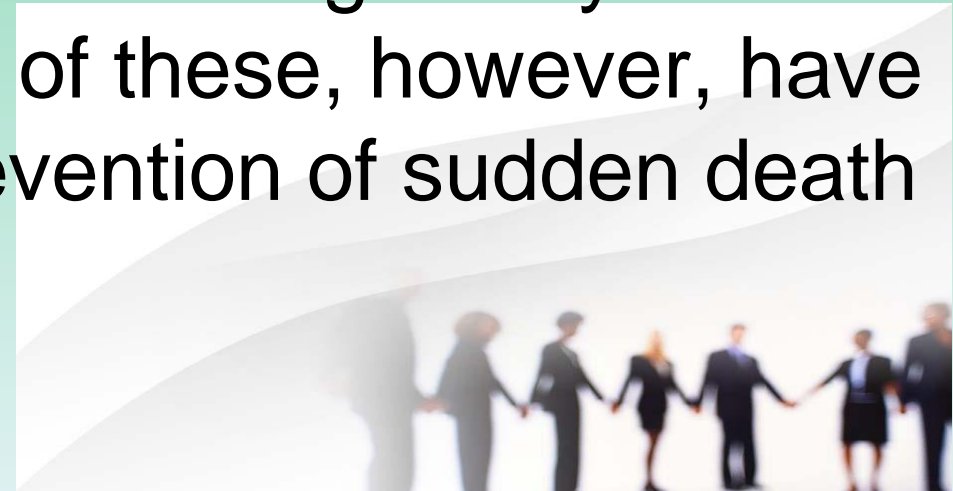
Conclusion

Classically, patients with Brugada syndrome generally have no evident systemic diseases in their past history. All too often, syncope or sudden cardiac death is the only symptom in these patients. In some cases, sudden death is the first symptom of the disease [7]. To our knowledge, this case is the first that demonstrates status epilepticus as the initial symptom of Brugada syndrome. Therapy of status epilepticus involves management of precipitating causes and prevention of seizure recurrence.



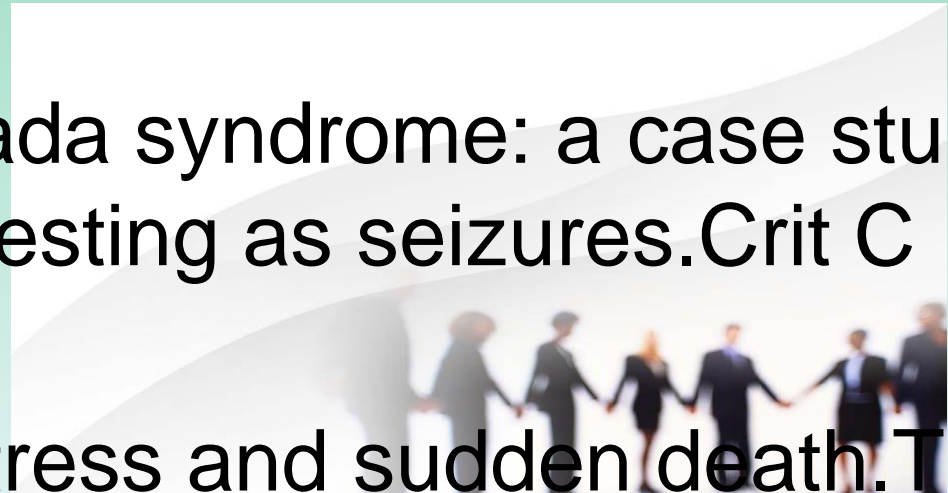
Conclusion

At present, ICD implantation is the most effective method in symptomatic patients with Brugada syndrome [4,9]. However, controversy exists in the treatment of asymptomatic patients. Asymptomatic patients should undergo electrophysiologic study, and if inducible, ICD implantation is recommended [9]. Agents that boost the calcium current, such as isoproterenol, and some class IA agents, such as quinidine and tedisamil, have been shown to be effective in normalizing ST-segment elevation in patients with Brugada syndrome, and in controlling electrical storms. None of these, however, have demonstrated long-term efficacy in the prevention of sudden death.



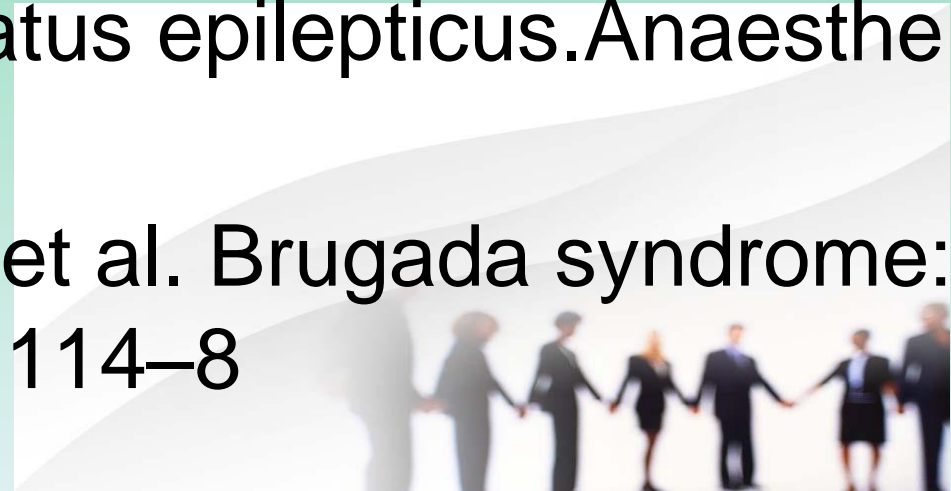
References

1. Huang, C. C., Chen, T. W., Lin, F. C., & Huang, M. H. (2005). Status epilepticus as an initial presentation of Brugada syndrome: a case report. *The Kaohsiung journal of medical sciences*, 21(8), 387-391.
2. Paydak H, Telfer EA, Kehoe RF, et al. Brugada syndrome: an unusual cause of convulsive syncope. *Arch Intern Med* 2002;162:1416–9.
3. Yager M, Benson J, Kamajian M. Brugada syndrome: a case study of aborted sudden cardiac death manifesting as seizures. *Crit Care Nurse* 2001;21(1):38, 40, 42–6.
4. Schwartz PJ, Zaza A, Locati E, et al. Stress and sudden death. *T*



References

5. Grubb BP, Gerard G, Roush K, et al. Differentiation of convulsive syncope and epilepsy with head-up tilt testing. *Ann Intern Med* 1991;115:871–6.
6. Wilde AA, Antzelevitch C, Borggrefe M, et al. Proposed diagnostic criteria for the Brugada syndrome. *Eur Heart J* 2002;23:1648–54.
7. Chapman MG, Smith M, Hirsch NP. Status epilepticus. *Anaesthesia* 2001;56:648–59.
8. Antzelevitch C, Brugada P, Brugada J, et al. Brugada syndrome: a decade of progress. *Circ Res* 2002;91:1114–8



Thank you

