

An Unusual Case Report

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Citation

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Abstract

A 28yr. old software professional was admitted to hospital for elective laparoscopic appendisectomy.

He was ASA – 1 with no drug allergies, good exercise tolerance, not addicted to anything, not having any anaesthesia exposure in the past.

As per ASA guidelines for young healthy male patient without any co-morbid conditions only CBC, viral screening was advised during preanaesthetic evaluation.

Patient was taken to the operation room. General anaesthesia with endotracheal intubation was given.

Anaesthetic plan was like:

Securing 20G IV cannula on lt.forearm, connecting routine monitors such as two lead

ECG, pulseoximeter, capnograph, non-invasive BP monitor etc.

Pre-induction vitals were within normal limits

Inj fentanyl 100µg was given slow IV. Followed by Inj.midazolam 2mg.

100mg of Inj.propofol was used as an induction agent and 50mg of Inj. Atracurium was used as long acting muscle relaxant.

Post induction vitals were also within normal limits except some minor pressor response.

Anaesthesia was maintained with sevoflurane and N₂O: O₂ = 60:40.

Immediately after creating pneumoperitoneum with CO₂. The patient had short run of VPC, s which got converted to VF within seconds..

Quickly pneumoperitoneum was deflated, defibrillator was

fetched and patient was shocked with 360 joules biphasic current. Normal sinus rhythm achieved.

Considering it to be an abnormal response to pneumoperitoneum, surgery was postponed { elective}.

Patient reversed and extubated after close monitoring.

After 2hrs. Of extubation in PACU patient had same episode of VF ,required to be shocked again..

Patient was investigated thoroughly, the baseline ECG was showing RBBB, with minor ST-T changes. 2D echo was having RA dilatation, good LV function, no RWMA.

The baseline ECG of the patient was traced; two yr. back at the time of his employment, had same RBBB pattern, which was neglected in view of no symptoms considering it to be benign.

The specialist consultation was taken, cardiologist labelled it as straightforward case of BRUGADA SYNDROME.

Surgery was postponed and relatives were counseled in detail. But it was a narrow escape for everyone.

DISCUSSION

Something about BRUGADA SYNDROME –

The Brugada syndrome is a genetic disease that is characterised by abnormal electrocardiogram (ECG) findings and an increased risk of sudden cardiac death. It is also known as Sudden Unexpected Death Syndrome (SUDS), and is the most common cause of sudden death in young men without known underlying cardiac disease. This condition is inherited in an autosomal dominant pattern and is more common in males.

The typical patient with Brugada syndrome is young, male, and otherwise healthy, with normal general medical and cardiovascular physical examinations.

Brugada syndrome is an example of a channelopathy; a disease caused by an alteration in the transmembrane ion currents that together constitute the cardiac action potential. Specifically, in 10-30% of cases, mutations in the SCN5A gene, which encodes the cardiac voltage-gated sodium channel Nav 1.5, have been found. These loss-of-function mutations reduce the sodium current (INa) available during the phases 0 (upstroke) and 1 (early repolarization) of the cardiac action potential. This decrease in INa is thought to affect the right ventricular endocardium differently from the epicardium, thus underlying both the Brugada ECG pattern and the clinical manifestations of the Brugada syndrome.

Brugada syndrome has 3 different ECG patterns. Type 1 has a coved type ST elevation with at least 2 mm J-point elevation a gradually descending ST segment and a negative T-wave. Type 2 has a saddle back pattern with a least 2 mm J-point elevation and at least 1 mm ST elevation with a

positive or biphasic T-wave. Type 2 pattern can occasionally be seen in healthy subjects. Type 3 has a saddle back pattern with less than 2 mm J-point elevation and less than 1 mm ST elevation with a positive T-wave. Type 3 pattern is not uncommon in healthy subjects. The pattern seen on the ECG is persistent ST elevations in the electrocardiographic leads V1-V3 with a right bundle branch block (RBBB) appearance with or without the terminal S waves in the lateral leads that are associated with a typical RBBB.

The major manifestation of Brugada syndrome is polymorphic ventricular tachycardia that can degenerate into ventricular fibrillation and cause sudden cardiac death.

AICD implantation is the treatment of choice.

References

r-0. Internet data on Brugada syndrome.

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