Revista Colombiana de Anestesiología
Colombian Journal of Anesthesiology
www.revcolanest.com.co

Case report

Successful extended cerebrocardiopulmonary resuscitation of a sudden death patient: A case report☆

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ARTICLE INFO

Article history:
Received 8 December 2013
Accepted 23 March 2014
Available online 17 May 2014

Keywords:
Sudden death
Resuscitation
Cardiac arrest
Pacemaker
Coronary disease
Valve disease
Hypoxic ischemic encephalopathy

ABSTRACT

This is the case of a 63-year-old patient, who is a plastic surgeon and has a history of aortic valve replacement, hypertension, pacemaker and anticoagulation, who experienced cardiac sudden death in the OR. Basic and advanced life support maneuvers were initiated; there was evidence of ventricular fibrillation and the patient was defibrillated 4 times unsuccessfully. Epinephrine, bicarbonate, amiodarone and lidocaine were administered. The patient alternated between ventricular fibrillation, pulseless electrical activity and asystole. Resuscitation was maintained throughout the process which lasted one hour and 45 min, including transfer to a third level clinic where the patient was considered to be asystolic. Following an additional discharge and amiodarone, the patient recovered spontaneous circulation; the vital signs were normalized and the patient remained in the ICU under hemodynamically stable conditions. After 18 h, the patient woke up with no evident neurological damage and remained in the ICU for one month for treatment of the ischemic-reperfusion syndrome. After 20 more days of physical therapy in his hospital room, the patient was discharged with no neurological deficit and a recommendation for home-based rehabilitation. Three months later, the patient is doing perfectly well and leading an active family, social and labor life.

This narrative discussion considers some interesting aspects reported by other authors on the topic, based on a bibliography search in Medline, Lilacs, Scielo, and Ovid.

☆ Please cite this article as: Reyes JG. Reanimación cerebrocardiopulmonar prolongada exitosa en un paciente con muerte súbita: un reporte de caso. Rev Colomb Anestesiol. 2014;42:229–233.

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Reanimación cerebrocardiopulmonar prolongada exitosa en un paciente con muerte súbita: un reporte de caso

Palabras clave:
Muerte súbita
Reanimación
Paro cardiaco
Marcapasos
Enfermedad coronaria
Valvulopatías
Encefalopatía
isquémica-hipòxica

RESUMEN

Se reporta el caso de un paciente de 63 años de edad, cirujano plástico facial de profesión, con antecedentes cardiovasculares de reemplazo valvular aórtico, hipertensión arterial, marcapasos y anticoagulación, quien presenta muerte súbita de origen cardíaco en salas de cirugía. Se inician maniobras de reanimación básica y avanzada, se evidencia fibrilación ventricular y se desfibrila en 4 oportunidades sin éxito, se aplican epinefrina, bicarbonato, amiodarona y lidocaína. El paciente alterna entre fibrilación ventricular, actividad eléctrica sin pulso y asistolia. La reanimación se mantiene constante durante todo el proceso, que dura 1 h y 45 min, incluyendo el traslado a una clínica de tercer nivel, en donde consideran que el paciente está en asistolia. Tras otra descarga y más amiodarona, el paciente recupera la circulación espontánea, se normalizan los signos vitales y se deja en la UCI en condiciones hemodinámicamente estables. A las 18 h el paciente despierta sin daño neurológico evidente, permanece en la UCI por un mes, resolviendo los problemas relacionados con el síndrome isquemia-reperfusión, y luego de 20 días más con fisioterapia en la habitación, el paciente es dado de alta con recomendaciones de rehabilitación en el hogar sin ningún déficit neurológico. A los 3 meses del evento el paciente se encuentra reintegrado a la vida familiar, social y laboral en perfectas condiciones. Luego de la búsqueda bibliográfica en las bases de datos médicas electrónicas de: Medline, Lilacs, Scielo y Ovid, se discuten en esta revisión narrativa algunos aspectos interesantes reportados por otros autores en relación con este tema.

Clinical case

The case is of a 63-year-old patient, facial plastic surgeon, with a relevant cardiovascular history: 1 – Aortic valve replacement with mechanical prosthesis performed 10 years ago because of aortic insufficiency; 2 – Anticoagulation with warfarin; 3 – Pacemaker because of a third degree AV block as a result of valve surgery; 4 – HBP treated with metoprolol 25 mg-day; 5 – Dislipidemia and undergoing statin treatment. The patient experienced a cardiac arrest just when he was about to start a surgical procedure in the OR. The patient was immediately placed on a stretcher in the OR. The clinical examination revealed an unconscious, cyanotic patient, pulseless and with dilated and fixed pupils. The Code Blue/5 link was immediately activated and basic and advanced life support was initiated. An orotracheal tube was placed, the anesthesia machine was connected for manual and mechanical ventilation and a cardiac massage was started alternating 30 compressions and 2 ventilations. The ulnar vein was catheterized and the patient was under monitoring which showed ventricular fibrillation that was unsuccessfully treated with a monophasic discharge of 360 Jules; three additional 360 Jules defibrillations were attempted unsuccessfully, with asystole tracing. The physician ordered the administration of epinephrine 1 mg every 3–5 min, lidocaine 1 mg/kg, bicarbonate 1 vial and amiodarone 150 and 300 mg. Under this persistent and uninterrupted resuscitation regime, 60 min elapsed with on-and-off ventricular fibrillation, pulseless electrical activity and asystole. Clinically the patient remained cyanotic, pulseless and with no signs of spontaneous circulation recovery. Past cardiovascular events will explain the difficulty in rapidly obtaining sinus rhythm and effective spontaneous circulation. The clinic cardiologist found no pacemaker activity and agreed with the anesthesiologist that the patient was asystolic. The decision was made to transfer the patient to the closest third level clinic and during transport ventricular fibrillation (VF) was interpreted and a new 200 Jules discharge was administered with the bifasic Automatic External Defibrillator with no favorable response.

The third level emergency colleagues felt that the arrest was irreversible, the electrical activity was absent and resuscitation was already too extended (105 min); however, a few minutes later the patient recovered his femoral pulse and cyanosis started to disappear. An echocardiogram showed a functioning heart with an ejection fraction (EF) of 35%. The massage was stopped and the norepinephrine/amiodarone drip was increased, rapidly accomplishing the sinus rhythm, BP 95/45, $\%O_2$ Sat 95% and capnography 50 mmHg. The patient was transferred hemodynamically stable to the Intensive Care Unit (ICU) in order to continue with the fifth link (post-heart arrest integrated care).

Arterial gasses at admission to the ICU showed a pH of 6.95, HCO_3 15.4, $PaCO_2$ 70, BE -16.7, $\%O_2$ SAT 59%, and lactate 5.5. In the immediate phase following the post-arrest syndrome in the ICU, coronary angiography, brain-CT and continuous EEG for 6 h were performed, all with normal results, ruling out any coronary disease or major cerebrovascular events. Sedation was lowered at 18 h and a first evaluation was done for a neurological prognosis, finding normal bilateral pupillary

and corneal reflexes. Although the patient was somnolent, he responded to verbal and pain stimuli. The patient had a Glasgow score of 15/15 at three days, and ischemic hypoxic encephalitis was ruled out.

The patient stayed in the ICU for one month until complete recovery of the effects from total body ischemia and the reperfusion phenomenon leading to ischemic liver failure (creatinine 6.0) and ischemic liver failure that required hemodynamic support with norepinephrine for 4 days, mechanical ventilation with orotracheal intubation/tracheostomy for 15 days and renal replacement therapy with hemodialysis for one month, until normal renal filtration was achieved. Considering the high risk of recurrence of a sudden death event due to ventricular tachycardia or fibrillation (VT/VF), the pacemaker was replaced with an implantable cardioverter defibrillator (ICD) free of complications.

At 40 days of hospitalization the patient was transferred to his room to continue with the neuromuscular rehabilitation phase secondary to the myopathy – neuropathy syndrome of the critical patient with physical impairment secondary to prolonged immobilization. At 2 months, the only neurological deficit the patient presented was mild to moderate muscle strength loss and was discharged from the clinic with a recommendation for home physical therapy.

At 3 months and after numerous neurological, psychiatric and psychological controls and using neurological evaluation scales such as Glasgow, Folstein Mini-Mental, Rankin modified, NIH CVA, Barthel index (measures dependency) and special sensitive and motor explorations, the patient was declared recovered and free of neurological/cognitive deficit. At present the patient is fully functional socially and family wise, and is working as a surgeon.

Discussion

Sudden death is defined as an unexpected and abrupt death in the absence of trauma that usually does not present any preliminary symptoms or, if these occur, they manifest themselves a few minutes before death occurs.[1–3] Between 75 and 85% of adult sudden deaths are cardiac in origin. The rest of the non-cardiac causes do not frequently result in immediate cardiac arrest and the discussion is then whether the event was or not witnessed.[4,5]

In terms of sudden cardiac death (SD), it has been found that 75–80% of the events are coronary in origin, 15–20% are related to structural acquired heart disease – i.e. hypertrophic cardiomyopathy, arrhythmogenic dysplasia of the right ventricle, dilated cardiomyopathy, and acquired heart disease of valvular and hypertensive origin, among others. A remaining 5% of the events have a primary electrical origin and a positive family history such as long QT, Brugada, and Wolff-Parkinson-White with aberrant conduction syndromes, among others.[6–8]

The incidence of SD in the United States ranges from 850 to 950 cases/million/year,[8] in Spain the incidence is of 192 cases/million/year[9] and in Argentina 732 cases/million/year.[10] There are no statistics on SD in Colombia. Despite the massive implementation of basic and advanced resuscitation courses for physicians and the general public, in addition to the provision of Automated External Defibrillators in public areas and its corresponding training, mortality in the US continues to be very high, with a survival of only 7.9%,[8] a figure not very different from the Argentinian at 5%.[10] Studies show that this is due to the fact that most events are not witnessed and occur at home, in the office or shopping malls where response is usually late and inadequate. In the intrahospital environment, successful resuscitation is accomplished in 20–30% of the cases, frequently with severe neurological sequelae and a low percentage of patients discharged free from neurological deficits. One explanation may be that in a hospital, patients usually exhibit more severe or terminal illnesses that are difficult to resuscitate or have a do-not-resuscitate code and not enough effort to resuscitate is made.[11,12]

This case illustrates very clearly the pathophysiology of the event.[1,9,13] A patient with aortic insufficiency secondary to a congenital bicuspid valve for more than 30 years develops a dilated and hypertrophic ventricle and, despite the mechanical valve replacement at 51 years of age, the ventricular damage is not completely reversed; additionally, there is an intraoperative injury of the bundle of His that led to a third grade AV block that required a pacemaker, in addition to chronic high blood pressure treated with metoprolol. All of these result in a heart vulnerable to transient ischemic events that disrupt the oxygen supply/demand ratio in a heart with no coronary disease.[14–17] Consequently a ventricle under these conditions subject to intensive sympathetic stimulation undergoes ischemia that triggers malignant arrhythmia – VT/VF – and hence SD. If the response time exceeds 3–5 min, the neurological injury will be irreversible and if more than 10 min, the probability of a successful resuscitation is almost nil.[18,19] Resuscitation procedures extended beyond 20 min result in high neurological morbidity and beyond 10 min in asystole are useless.[20,21] The International Committees recommend termination of the maneuvers after 20 min, except for patients achieving VT/VF rhythms when the decision of when to terminate resuscitation becomes more difficult because the rhythm diagnosis is confusing and the patients alternate from one rhythm to another.[22,23] Rating scales have been designed for this purpose, in order to define do-not-resuscitate codes and others to determine the neurological prognosis of inpatients who develop cardiac arrest.[24] A very different situation is that of an outpatient from which little is known about his/her history or functional class at the time of the event.

Epidemiological studies show interesting coincidences with this case; i.e., these events are more frequent in males aged 58 ± 12 years, and present more often on Monday morning between 6 a.m. and noon – which was exactly the case with this particular patient. The event occurred on a Monday at 9:30 a.m., and stressed the importance of the autonomic nervous system as the triggering factor for these events. The physiology evidences a marked increase in the sympathetic activity on Monday morning because of increased emotional and physical stress.[8] There have been more than 20 coronary disease and sudden death-related mutations identified; the lower incidence of these events among the Mediterranean population versus Anglo Saxons is also notable,[25,26] probably

as a result of the Mediterranean diet and life style.[27,28] In this particular patient, a healthy diet and good lifestyle habits during the last 12 years could have protected him from coronary disease. SD occurs more often during the wintertime[8,13] and it will be interesting to know the relationship between climate and altitude in Colombia.

With regard to the use of defibrillators and the comparison between monophasic vs. biphasic, the current recommendation is to use biphasic defibrillators that render better clinical outcomes with lower discharge energy, are safer for both the patient and the resuscitator, are easier to use and are less harmful for circuits and pacemaker materials that may result in electrical failures and EEG diagnostic errors during resuscitation.

Another important aspect in this case has to do with a potential pacemaker failure that caused severe bradyarrhythmia and consequently asystole. The literature alerts about the risks of electromagnetic interferences in pacemakers such as pacemaker circuit failures,[29] including mobile phones, metal detectors, transformers, high voltage lines, nuclear resonance, electro-scalpel, defibrillators, ionizing radiation, etc. Some studies recommend not to store the mobile phone on the same side as the generator and to listen through the opposite ear. When checking the memory of pacemakers from patients who died from SD, the most frequent fatal arrhythmia was ventricular tachycardia.[30] So, along these lines, and keeping in mind that only three leads are used, a very interesting trial showed that during resuscitation of patients with pacemakers, it is very difficult to differentiate an asystole rhythm from fine ventricular fibrillation and in case of doubt, the authors recommend to always defibrillate.[31] Pacemaker technology advances have improved patients' quality of life and life expectancy, providing pacemaker capabilities, resynchronization therapy and cardioversion-defibrillation function in the presence of malignant arrhythmia such as VT/VF, all in one single device.[32–34] One of the indications for this technology is a resuscitated SD patient, such as this particular case in point.[35,36]

Reviewing the risk factors for CV disease we must take into account that according to the World Bank, Colombia is considered a low and medium-income country and is in the third epidemiological transition phase that is characterized by an increasing number of degenerative diseases and human-caused diseases such as cerebrovascular conditions, and specifically cardiovascular pathology with a 35–65% mortality due to risk factors such as a sedentary lifestyle, high blood pressure, diabetes, dyslipidemia, smoking, obesity and a growing elderly population.[37] Based on these facts, the incidence of SD in Colombia is expected to rise and thus the medical schools are required to strengthen their basic and advanced life support programs for both undergraduate and graduate students, as a basic and obligatory skill that physicians should have and share with the rest of the health care staff at their work places. We, the anesthesiologists, play a critical role in ongoing education on these topics and in organizing blue code teams. A Bill was submitted for the approval of the Colombian Congress 2 years ago promoting the obligatory installation of automatic external defibrillators (AED) in public areas and suggests training the general population in basic life support.[19]

Conclusion

This review helped in identifying several factors that could have contributed to a successful resuscitation of this patient: 1 – Witnessed SD event that developed in the OR; 2 – Basic and advanced immediate resuscitation directed by an experienced anesthesiologist with the support of 5 doctors and 4 nurses; 3 – perseverance with resuscitation because of emotional labor and family considerations (the patient was a colleague); 4 – Probably anticoagulation prevented major ischemia of the brain and the heart; 5 – A previously healthy patient, with controlled associated pathologies, professionally active and a healthy lifestyle.

Managing a SD event requires an analysis of the type of patient we are dealing with and doing a rapid prognosis to decide whether it is worthwhile insisting and using every available resource, since those valuable minutes represent the life of a patient, a family, a business and a society as a whole.

The probability to survive a SD event in Colombia is very low and even more so if the patient recovers with no neurological deficit. What happened with this patient is the sum of some favorable and unlikely circumstances that, together with a persevering attitude, resulted in perfect recovery.

Funding

None.

Conflict of interests

The authors have no conflicts of interest to declare.

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