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Early Recognition, Timely Intervention and Immediate CPR and its Outcome in a CKD Patient with Cardiac Arrest

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Abstract

Cardio-respiratory arrest is a real medical emergency. It can present as Pulseless VT (ventricular tachycardia), VF (ventricular fibrillation), Asystole, and PEA (Pulseless Electrical Activity). PEA is defined as any organized rhythm without a detectable pulse.

As per ACLS protocol 2010 guidelines, PEA should be treated with CPR and Epinephrine and / or Vasopressin as charted below and the most important step is to identify any reversible cause and to correct it.

Here in our present case, the 61 yrs old female patient presented as Cardio-respiratory arrest with PEA with severe metabolic acidosis and hyperkalemia.

She was treated as per ACLS guidelines and was revived successfully and she was discharged in a stable condition after 48 hrs.

Keywords : Pulseless Electrical Activity (PEA); Chronic Kidney Disease (CKD); End Stage Renal Disease; Cardiac Arrest; Compressions; Hyperkalemia; Hypercarbia; Metabolic Acidosis; Hemodialysis; Sudden Cardiac Death; Agonal Gasp.

Introduction

Cardio-respiratory arrest in patients of ESRD / CKD is not uncommon and can be due to various reasons like metabolic acidosis, hyperkalemia, hypoxia, coronary thrombosis, hypercarbia. PEA presents as cardio-respiratory arrest which is a real medical emergency. It includes rhythms like Sinus rhythm, Idioventricular rhythms, Post-defibrillation idioventricular rhythms, Ventricular escape rhythms.

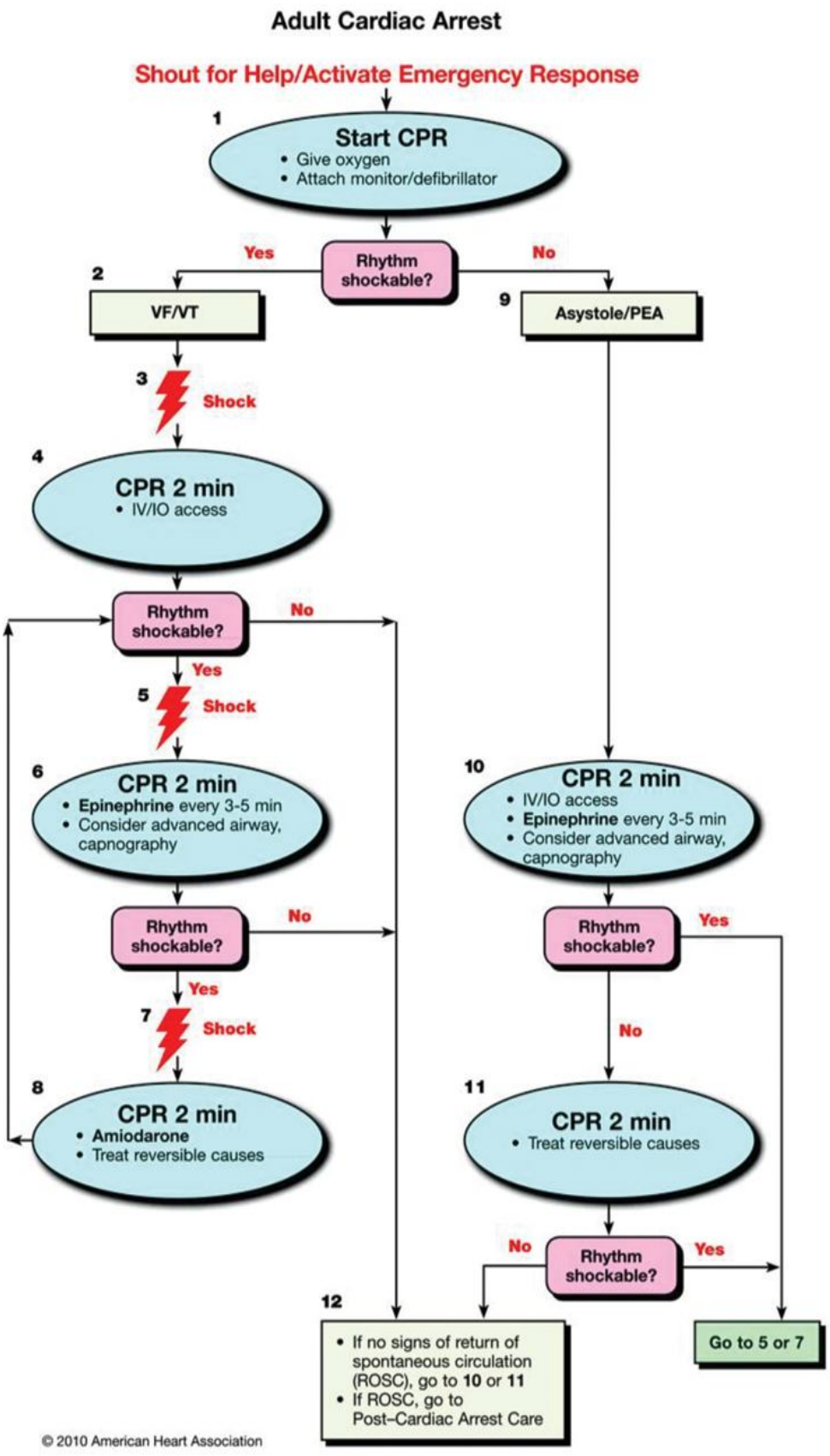
Previously PEA was termed as Electromechanical Dissociation (EMD) to describe patients who displayed electrical activity on cardiac monitor but lacked apparent contractile function because of an undetectable pulse. This means a weak contractile function is present – detectable only by invasive monitoring or echocardiography – but the cardiac function is too weak to produce a pulse or effective CO. This is also the most common initial condition present following successful defibrillation.

There are several reversible causes of PEA, popularly called 5 H's and 5 T's, including Hypovolemia, Hypoxia, Hydrogenion (acidosis), Hypo-/Hyperkalemia, Hypothermia and Tension pneumothorax, Cardiac Tamponade, Toxins, Coronary Thrombosis, Pulmonary Thrombosis.

In our case, cardiac arrest could have been due to hyperkalemia, and / or acidosis. The treatment of PEA is primarily directed towards treating the underlying cause as per the ACLS protocol.

On initial examination, if the patient is in cardiac arrest (unresponsiveness, no pulse / BP, no spontaneous respiration or only gasping), immediate chest compressions needs to be started within 10 seconds of identifying the diseased condition, i.e., PEA here. Drugs like Epinephrine 1mg every 3-5minutes or Vasopressin 40 U to replace the 1st or 2nd dose epinephrine can be given, along with other drugs as per the underlying cause of PEA.

The ACLS protocol for the treatment of PEA is charted as below.



CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy
- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy
- **Epinephrine IV/IO Dose:** 1 mg every 3-5 minutes
- **Vasopressin IV/IO Dose:** 40 units can replace first or second dose of epinephrine
- **Amiodarone IV/IO Dose:** First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes
- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Extract from ACLS (AHA) 2010 guidelines

Case History

A 61 years old female patient was brought by her attendants in a wheelchair in an unresponsive, unconscious and gasping state on 8th July 2015 at around 08:30 AM.

She was said to be in this state for about 20 minutes before her presentation in the ED. She was a known case of Diabetes mellitus, Hypertension, CAD Post-PTCA+Stenting and ESRD on maintenance hemodialysis (twice/week; last HD was 4 days back).

On examinations, patient was unconscious, unresponsive, and was gasping. Pulse was not palpable, BP was not recordable, SPO2 was 60% at room air. Cardiac monitor showed PEA.

Patient was immediately put on Bag-Mask ventilation and effective CPR was started as per ACLS protocol.

Two large bore IV cannulas were inserted and ABG was sent to the lab. During CPR inj epinephrine 2mg (1+1) was given. After 5 cycles (2 minutes) of CPR, ROSC was achieved in the ED.

Patient was intubated with ETT size 7.5 after giving Inj Etomidate 18mg and Inj Rocuronium 75mg and she was put on the ventilator with the following ventilator settings: ACV mode, FiO2 1.0, f 15, PEEP 5, TV 500.

Her ABG analysis showed: pH 6.88/ PO2 72mmHg / PCO2 84mmHg/ HCO3 15mmol/L/ Na+ 138mmol/L/ K+ 6.101mmol/L / Lactate 3.9

Inj Calcium Gluconate 10% 10ml was given over 10 minutes. Inj Fentanyl infusion was started @50mcg/hr. Inj Sodium bicarbonate 200ml was given stat.

Post-ROSC and Intubation, Pulse was 98/min regular, BP 170/110 mmHg, RR 18/min regular, SPO2 100% with FiO2 1.0 on ventilator. Patient was afebrile and RBS was 260 mg%

On Systemic Examination

RS: AE B/L equal but B/L basal crepts present. CVS: S1 S2 Normal, No murmur/bruit

PA: Soft, non-distended, BS+; No organomegaly

Neuro: Sedated and Paralysed; Pupils B/L 2mm and sluggishly reacting to light. Extremities: Warm, B/L Pedal edema++

AMPLE History

A - No known allergies

M - Regular Medication details not available

P - Known DM/HTN/CAD Post-PTCA+S/ ESRD on maintenance hemodialysis (2/week)

L - She had light breakfast that morning.

E - Events as per given history above.

She was provisionally diagnosed as PEA (Pulseless Electrical Activity) with Severe Metabolic Acidosis.

More investigations were sent as follows: CBC, LFT, KFT, ECG, CXR PA.

Foley's catheter no 14 was inserted and urine flow observed. Ryle's tube no 16 was inserted and its position was confirmed.

The following medications were given in the ED:

- Inj Emeset 4mg IV stat.
- Inj Pantoprazole 40mg IV stat.
- Inj Calcium Gluconate 10% 10ml was given over 10 minutes.
- Inj Dextrose 25% + Insulin 10 units IV stat.
- Inj Noradrenaline 5mcg/hr infusion started.
- Inj Fentanyl 50mcg IV stat and @ 50mcg/hr infusion started.

The case was discussed with the Nephrologist and the patient was admitted in ICU.

Cardiolology reference was given.

Her Vitals after 45 minutes of presentation

P 95/min regular BP 120/80 mmHg (on Noradrenaline @ 2.5mcg/hr) SPO2 100% on ventillator Cardiologist saw the patient at 09:25 AM and advised Troponin I. Guarded prognosis was explained to the attendants.

Patient was shifted to ICU AT 09:45 AM.

Patient was seen by Nephrologist and was started on SLED around 3 PM. Patient started gaining her consciousness by afternoon and was extubated in the evening.

Noradrenaline was tapered off and was stopped. Her vital stats were maintained throughout the day.

Reports of initial blood sampling

Urea 143mg/dl, Creatinine 9.83mg/dl, Na 135mmol/l, K 6 mmol/l, Calcium 8.2 mg/dl, Magnessium 2.9mg/l, Phosphorus 4mg/l, Hb 9.5gm/

dl, TLC 19900,Troponin-I Negative, Liver function test WNL. CXR showed B/L Infiltrates

Treatment given during the hospital stay

Tab. Azithromycin , Cap. Ecosprin AV, Inj. Elores (Ceftriaxone+Sulbactam), Inj. Novorapid , Inj. Ranitidine, Tab. Shelcal, Sub-Whey Protein Powder and other supportive medications. She remained stable in the ward.

Foley's catheter was removed the following morning (09/07/2015)

She underwent Hemodialysis on 9th July with ultrafiltrate of 2 L and was shifted to ward.

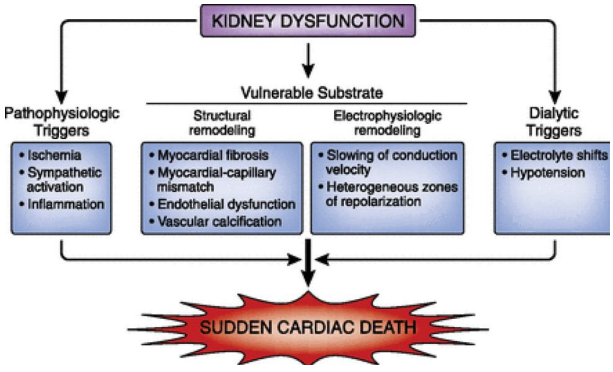
Reports

Na 130mmol/l, K 5.9mmol/l

She was discharged after 48 hrs in stable condition on 10/07/2015.

Discharge Medications

- Tab. Azithromycin 500mg once daily.
- Cap. Ecosprin AV 1 Capsule at bedtime.
- Inj. Elores 1.5gm IV twice daily.
- Inj. Novorapid SQ thrice daily started with low dose sliding scale.
- Tab. Ranitidine 150mg thrice daily.
- Tab. Shelcal 500mg thrice daily.
- Sub Whey Protein powder 2tsf twice daily.



After Discharge

She was followed up and hemodialysed on OPD basis thrice/week, last being on 25/07/2015, without any complications (followed up until 25/07/15).

Discussion

Chronic Kidney Disease (CKD) affects 13% of adults in the USA.

The majority of cardiovascular-related deaths in ESRD are attributable to SCD (Sudden Cardiac Death) events.

The incidence of SCD in the USA ranges approximately between 180,000 and 450,000 cases annually. Despite major advances in CPR and post-ROSC care, survival to hospital discharge after cardiac arrest remains very poor, estimated to be only 7.9% among out-of-hospital cardiac arrests that were eventually treated by emergency medical personnel.

The prognosis from cardiac arrests is even worse in patients with kidney dysfunction in which survival chances decreases with a declining GFR. The likelihood of survival following cardiac arrest is further low in dialysis patients.

Structural and electro-physiologic remodeling of the heart, vascular calcification and fibrosis, autonomic dysregulation, and volume and electrolyte shifts are some of the underlying processes thought to explain the increased predisposition for SCD in people with CKD.

This patho-physiology is depicted as in the flow chart below:

In patients with CKD, cardiomyopathy commonly occurs because of LV pressure and volume overload. Both atherosclerotic and arteriosclerotic vascular diseases also occur frequently.

This adverse cardiomyopathic and vasculopathic milieu predisposes individuals with CKD to arrhythmias, conduction abnormalities, and sudden cardiac death, which is likely to be exacerbated by electrolyte shifts, divalent ion abnormalities, diabetes, and sympathetic over-activity, in addition to inflammation and possibly iron deposition.

Impaired baroreflex effectiveness and sensitivity, as well as obstructive sleep apnea might also contribute to the risk of sudden death.

Cardiac arrest, due to whatsoever reason, does not give much time for interventions.

Therefore early recognition of cardiac arrest, immediate interventions like CPR and ACLS drugs administration and treating the underlying cause, are most important in not only reviving the patient but also to reduce mortality after revival. Agonal gasps can mislead the medical staff in detecting cardiac arrest. Agonal gasps are not adequate breathing; it

looks like the patient is drawing air in very quickly but if the patient is not responding to commands, it is actually a sign of cardiac arrest and must be intervened quickly, as done in our case.

The more the delay in interventions, the less the chances of survival and if revived, more the chances of end-organ damage like brain and kidneys.

This case report shows that early recognition of cardiac arrest and timely interventions and correcting the possible underlying cause of PEA, lead to revival of the patient without any end-organdamage.

Conclusion

Any cardiac arrest patient should be intervened early.

As Emergency physicians any such presentation should be considered with evidence based approach (as the ACLS 2010 guidelines in our case).

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