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Editorial

Immediate coronary angiography after cardiac arrest—Friend or foe?

Increasing number of out-of-hospital cardiac arrest (OHCA) patients are being admitted to hospitals due to improvement in pre-hospital "chain of survival" [1]. The majority of patients remain comatose after return of spontaneous circulation and the aetiology of cardiac arrest and patient's long term prognosis cannot be easily determined in the acute setting although finding and treating the cause of the arrest can improve morbidity and mortality as well reducing the arrest recurrence. Both pathological and angiographic studies established that coronary heart disease is the most common cause underlying OHCA, accounting for 50–70% of aetiologies in adults [2]. As a consequence, coronary angiography (CAG) is routinely performed as a part of diagnostic algorithm. However, there are other possible causes of OHCA, as illustrated by the high rate of either stable unobstructive or even normal coronary angiograms. In these patients, a neurological or a pulmonary cause should be suspected, and appropriate morphologic investigations (ultrasound, computed tomography (CT) scan) should be performed to investigate such causes.

Several studies have suggested that immediate CAG (iCAG) with percutaneous coronary intervention (PCI) may improve hospital survival in patients with cardiac arrest that have a concomitant acute coronary lesion [3–5]. Identifying these patients has proven challenging. Pre-arrest symptoms reporting in this setting is not uniform and often depends on the presence of bystanders witnessing the event. The decision to perform iCAG is often made with incomplete information and is usually based on post-arrest 12-lead ECG. In patients with STEMI in post-resuscitation ECG a culprit coronary lesion is found in more than 90% of patients [6] and these patients are routinely taken directly to catheterization laboratory via STEMI "fast track". Data from non-randomized trials also show that almost one third of post-arrest patients without ST elevation on ECG will be found to have an acute culprit lesion that could potentially benefit from emergent PCI. Guidelines therefore suggest that it is reasonable to perform CAG also in these patients if non-coronary cause of arrest is not found [7]. But do we do a harm in a small percentage of patients with non-cardiac cause of arrest with delaying other diagnostic and therapeutic procedures?

In the present issue of Resuscitation Stephane Legriel and co-workers from Paris Sudden Death Expertise Centre published data from their registry of OHCA patients [8]. Their primary focus was on 247 (7%) patients with neurological cause of OHCA (OHCA-NC) and their aim was to identify pitfalls in diagnostic algorithm that could potentially lead to delayed and inappropriate patient management. A retrospective etiological diagnosis of neurovascular causes for cardiac arrest was given in 47% patients, alcohol/drug poisoning in 22%, traumatic brain/medullar injury in 14%, seizure/status epilepticus 13% and other neurological causes in 4%. Overall in-hospital and 1-year mortality of selected cohort was 87% and 90%, respectively. All OHCA-NC patients with CAG as their first diagnostic procedure died during their ICU stay in contrast to 8.7% survival with favourable neurological outcome in the other group. At a first glance we could speculate that the survival difference could be attributable to diagnostic delay and therefore treatment delay in iCAG patients but the two groups were not comparable in all risk factors for bad outcome. Statistically important difference was in median time from collapse to CPR (no flow time) with much longer delay in iCAG group [7 min [4–10] in patients with iCAG versus 4 min (0–10) in patients without iCAG; $p = 0.005$]. Another difference that could cause increased mortality in iCAG patients is antiplatelet and/or anticoagulant therapy which was given in 8 (14%) and 1 (0.5%) patients with and without iCAG ($p < 0.0001$), respectively. The authors also did not explain why in their opinion were 18% of patients with traumatic brain injury and 10% with drug poisoning taken to cath lab before obtaining brain CT or laboratory results. Importantly, the study showed that two thirds of OHCA-NC patients had neurological signs/symptoms before arrest and that shockable rhythm was found in only 6.7% of OHCA-NC patients.

What can we learn from the study results? In our opinion the diagnostic strategy in OHCA patients should be guided by the patient's previous history, by prodromes observed before collapse, some suggestive circumstantial context, the presence of several physical examination signs at scene, first presenting rhythm and post-resuscitation ECG. In simple cases with no neurological prodromes or trauma, VF/VT as primary documented rhythm and ST segment elevation in ECG there is no dilemma to proceed directly to cath lab. In other case patients deserve a short "emergency department stop" to exclude non-cardiac causes of OHCA and to access their prognosis better. Head CT scan is definitely one of such important procedures that can demonstrate acute abnormalities which can result in significant changes in patient management. This way we can avoid unnecessary and potentially harmful cath lab admissions. Another important safety issue is not to apply antiplatelet/anticoagulation drugs before definite diagnosis as they can cause harm in OHCA-NC patients and have no proven benefit if applied very early in the course of treatment even if coronary pathology is responsible for the arrest [7]. Primary PCI should similarly be reserved for clear culprit lesions only. A subset of OHCA patients without ST elevation frequently have critical stenosis of coronary arteries. However, it is difficult to attribute the cause of the arrest to the diseased coronary artery especially in the absence of clearly distributed electrocardiographic findings. PCI and its associated antiplatelet therapy before excluding neurovascular causes of arrest could lead to critical complications.

Lastly, even an extensive imaging procedure involving both CAG and CT-scan will leave about 40% of resuscitated patients without clear etiological diagnosis [9]. Therefore, further investigations are mandatory to identify the underlying cause as studies show that aggressive diagnostic and therapeutic approach improves OHCA patients' outcome [10].

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Conflict of interest

None.

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