QUEENSLAND COURTS

CORONERS COURT OF QUEENSLAND

FINDINGS OF INVESTIGATION

CITATION:	**Non-inquest findings into the death of AD**
TITLE OF COURT:	Coroners Court
JURISDICTION:	BRISBANE
DATE:	09/07/2018
FILE NO(s):	2017/3861
FINDINGS OF:	Ainslie Kirkegaard, Coronial Registrar
CATCHWORDS:	CORONERS: chest pain presentation to emergency department; delay in diagnosis of STE elevation myocardial infarction (STEMI); delayed referral for emergency interventional cardiology; importance of timely review of all available pre-hospital ECG reports

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AD was a 75 year old man who died in intensive care on 6 May 2017 following complications from an anterior myocardial infarction.

AD's death was initially reported to the afterhours coroner who considered the death was not reportable. Following internal clinical review of the management of AD's initial presentation to a tertiary hospital emergency department, the hospital appropriately re-reported the death on 30 August 2017 due to concerns about delay in diagnosis and delay to treatment contributing significantly to the outcome for AD.

AD's medical history

Review of AD's medical records shows he was generally fit and healthy, did not smoke or drink alcohol and exercised regularly. He had been experiencing dizzy episodes during exercise over the preceding six or so months, initially thought to be vestibular vertigo. He was seen at regional hospital emergency department in November 2016 and diagnosed with 1st degree heart block but had not required any further specialist follow up since then. He was not on any regular prescription medication.

AD presented to a tertiary hospital emergency department by ambulance shortly before 6:30am on 3 May 2017 after waking up with a leg cramp and then becoming generally unwell with central chest/epigastric pain about an hour earlier. He had played three hours of badminton the previous evening. AD described the pain as sudden onset sharp retro-sternal chest pain radiating down both arms. He was pale, sweaty, lightheaded and nauseous. After arriving at hospital, AD developed central back pain.

Three ECGs were performed by paramedics between 5:55am and at 6:20am en route to hospital. The summary QAS ECG printed at 6:21am states 'probable acute anterior myocardial infarction' and the paramedic report states 'acute coronary syndrome NSTEMI'.

AD was triaged Category 2 on arrival in the emergency department at 6:23am.

The first hospital ECG was performed at 6:47am. It is documented as "*sinus rhythm, premature supraventricular complexes, AV junctional escapes, causes? e.g. SA block or AV block, intra-atrial conduction delay, anteroseptal infarct, slight intraventricular conduction delay, right precordial ST elevation, consider infarct of acute occurrence inferior ST depression, probably reciprocal, abnormal ECG*." This ECG is countersigned at 7:45am.

A resident medical officer note made at 8:15am documents "*looks uncomfortable, BP 100*" and mentions "*ST elevation/high take off in anterolateral leads*". After Registrar review, the differential diagnoses under consideration were acute coronary syndrome versus dissecting thoracic aortic aneurysm.

AD was given 5mg IV morphine at 7:30am. A second ECG and repeat troponin were requested to be performed at 8:50am. However, it appears the second ECG was not performed until 10:52am and repeat troponin not until 12:50pm.

A CT angiogram was ordered and previous ECGs were obtained from the regional hospital. The CT angiogram was performed at 8:30am and reported at 10:12am as "*no evidence of aortic dissection. Stenosis of the origin of the coeliac trunk under the arcuate ligament and a small fusiform dilatation of the intrarenal abdominal aorta. Right hilar and mediastinal lymphadenopathy without an obvious cause demonstrated*." These findings were immediately discussed with the cardiology registrar.

AD's chest pain settled with morphine and he was noted to be stable.

An entry made at 12:38am notes AD was transferred from the "hot" to the "cold" section of the emergency department and that he still had "*6/10 back upper back pain feeling dizzy*".

The results of the repeat troponin and ECG performed at 12:50pm were reported to the cardiology registrar immediately. The cardiology registrar reviewed AD at around this time. The cardiology registrar's notes describe him as having presented with atypical chest pain, long standing dizziness, concerning ECG changes and bradyarrhythmia. The differential diagnoses under consideration at that time were underlying conduction disease and acute coronary syndrome. The plan was for AD to be admitted for telemetry and for discussion with the cardiology consultant about the need for further investigations.

AD was reviewed again at around 2:40pm at which time he was diagnosed with anterior ST elevation. His troponin level had risen to 10. He was reporting only mild chest pain. The ST elevation was noted to be coming down and he was haemodynamically stable. He was consented for emergency percutaneous catheter intervention and transferred to the coronary cath lab. He underwent coronary angiography, left heart catheterisation and insertion of four stents into a single coronary artery. The coronary angiogram findings were thought to be consistent with sudden coronary artery dissection rather typical atherosclerotic based disease.

A very detailed entry made by the admitting consultant, Dr PS, that afternoon notes that the first ECG performed in the emergency department showed anterior ST elevation but the cardiology team was not contacted until around 12:30pm. AD underwent CT angiogram to exclude aortic dissection and was then transferred to "Cold" within the emergency department despite an evolving ST elevation and ongoing chest pain. Dr PS's entry notes that AD was referred to the cardiology registrar as "conduction disturbance" given the emergency team's concern about recent episodes of presyncope and ?LAFB on ECG. He was not triaged as requiring urgent cardiology review. Dr PS's entry indicates that he was not informed of AD's ECG results until around 2:35pm at which time arrangements were made to transfer AD straight to the cath lab.

AD was admitted to the Coronary Care Unit at around 8:00pm for post-procedural monitoring. He was commenced on dual antiplatelet therapy and for a transthoracic echocardiogram the following day. Dr PS noted AD was at high risk of antero-apical dysfunction and possibly at risk of acute pulmonary oedema overnight; he was for urgent review and diuresis in the event he became acutely breathless.

He was reviewed by medical officers three times overnight for chest pain, ST elevation, elevated troponin level, neck pain, shortness of breath and atrial fibrillation. He was managed with glyceryl trinitrate (GTN), digoxin, heparin infusion and intravenous frusemide for pulmonary oedema. He was identified as being as high risk for cardiogenic shock.

AD's condition deteriorated throughout the next day, 4 May, with hypotension, oliguria (despite diuresis), poor arterial blood gases and ongoing atrial fibrillation. An urgent bedside transthoracic echocardiogram (TTE) performed that morning showed significant effusion over the right ventricle and severe left ventricular dysfunction. Dr PS's differential diagnoses were *"?LV rupture (semi-contained) -> pericardial collection."*

AD was urgently returned to the cath lab at 1:32pm in an "*extremely compromised state*". Dr PS initially attempted pericardiocentesis but this was complicated by difficulties advancing the wire into the pericardial space. AD became very hypotensive during the procedure despite inotropic support. Coronary angiogram demonstrated prominent coronary vasospasm but no occlusive lesions and the stents were patent. There was poor flow in the left anterior descending artery but there was no suggestion of coronary artery perforation. An intra-arterial

balloon pump was then inserted. AD was intubated and cardioverted with immediate haemodynamic benefit. Repeat pericardiocentesis with TOE guidance was successful, draining 250ml of heavily blood stained, non-clotting fluid with immediate effect.

Dr PS documented several possible causes for the pericardial collection including microperforation of the left ventricle, haemorrhagic effusion related to myocardial infarction or reperfusion injury and coronary microperforation.

Given significant concern about the possibility of microperforation, AD was then transferred to a cardiothoracic hospital intensive care unit for further investigation and management. Cardiothoracic review determined there was no indication for surgical intervention. AD continued to deteriorate with multi organ failure. After discussion with his family, he was transitioned to comfort cares and died on 6 May 2017.

The treating team discussed AD's death with the on-call coroner. That discussion is documented in the medical record as "*explained full story including delayed diagnosis. Coroner would consider this death by natural causes and for cause of death as anterior myocardial infarction.*"

Autopsy findings

A partial non-coronial consented hospital autopsy (chest and abdomen only) was performed by an anatomical pathologist at the cardiothoracic hospital on 9 May 2017. The autopsy revealed extensive acute myocardial infarction with marked reperfusion injury and associated pericarditis. There was no evidence of perforation and no significant re-accumulation of pericardial effusion. There was coronary artery atherosclerosis but no dissection of the stented left anterior descending coronary artery. There was also diffuse hepatic necrosis and likely acute kidney injury which the pathologist considered together resulted in multiorgan failure causing the death.

Tertiary hospital clinical review outcomes

Due to the treating team's concerns about potential delays in identifying and managing AD's STEMI, a SAC 1 incident was reported leading to a formal clinical incident analysis of the care he received.

The clinical review team noted:

- the emergency department was particularly busy that day with several very ill patients requiring immediate intervention;
- emergency department staff reported that AD appeared stable and very settled;
- AD reported features suggestive of a dissecting aortic aneurysm or conduction disease but on review was comfortable, texting and dozing at times;
- the treating team focussed on investigation of a dissecting aortic aneurysm or conduction disease and missed the anterior myocardial infarct;
- AD was transferred to the cath lab approximately nine hours post-admission and 10 hours to reperfusion; 11 hours from self-reported onset of symptoms – the review team discussed the reduction in benefit for revascularisation at this late stage and possible adverse effects of late reperfusion but it was agreed that PCI was the usual course of action at this point in time.

The review team concluded there was a delay in identifying the STEMI by both emergency department and cardiology staff. They identified two points where escalation may have led to

a different outcome for AD, firstly on admission, the initial ECG at 6:47am showed a STEMI and should have led to immediate referral to interventional cardiology and secondly, when the CT angiogram chest was reported and ruled out suspected dissection. The review team considered that had AD been transferred for PCI earlier, the procedure could potentially have been less complicated and the extent of the injury might have been less.

The initial hospital ECG report was noted not to be in the standard format and the STEMI was not identified with capital letters, bolding and an asterisk in the text at the top of the ECG as it would be normally. The review team considered this text box would have highlighted the STEMI and potentially reduced the risk of this information being missed by emergency department and cardiology staff. Further, the 12 lead ECG was not arranged in its usual format. Later ECGs identified the STEMI in the text box. The review team held divergent views about whether this needed to be escalated as a concern, with some members strongly of the opinion that clinicians must always interpret the ECG report and not rely on the text box for guidance. Consequently no recommendation was made around this. However the ECG machine in question was identified and sent for re-calibration.

The review team also noted that not all ECGs were countersigned by the most senior medical officer on the floor as required by emergency department protocol, though it appeared they had been reviewed.

It was considered the emergency department already has an ongoing robust program for interpreting ECGs, so no recommendation was made in relation to this issue.

The learnings arising from this review were shared with emergency department and cardiology staff and AD's case also discussed at both departmental Morbidity & Mortality review meetings.

Independent interventional cardiology review

I arranged for an independent interventional cardiologist, Dr Paul Garrahy, to review AD's clinical management and advise whether the delays identified by the tertiary hospital's clinical incident analysis were significantly outcome changing for him. Dr Garrahy is the Director of Cardiology at the Princess Alexandra Hospital.

Dr Garrahy reviewed the coronary angiogram and angioplasty films from the procedure on 3 July 2017 noting they reveal AD had three vessel coronary artery disease. It was clear to him the angioplasty procedure to the left anterior descending artery was technically difficult and performed with skill and care. He disagreed with the conclusion it was "a success" as there was residential significant narrowing in the proximal left anterior descending artery upstream from the long (small calibre) mid left anterior descending artery stent complex with less than TIMI III flow into the distal and apical left anterior descending artery territory. This result (obtained more than 10 hours from onset of symptoms) in particular the abnormal flow was not unexpected given the late infarct reperfusion. However, he considered the final result was unlikely to have significantly improved AD's chances of surviving this large, completed anterior myocardial infarction.

Dr Garrahy described AD's subsequent clinical progress – severe cardiogenic shock with atrial arrhythmias - as not unexpected. A haemorrhagic pericardial effusion developed which required return to the cath lab that following day for percutaneous drainage. This most likely occurred as a complication of the late reperfusion and was recognised and managed appropriately.

Dr Garrahy identified the following critical delays in AD's management:

1. AD was triaged as Category 2 despite the history of "*5/10 central chest/epigastric pain with diaphoresis*" and some rhythm abnormalities. QAS had recorded pain radiating to the arms and had recorded acute coronary syndrome as their provisional diagnosis. Dr Garrahy suggested this symptom complex might well have triggered Category 1 urgency – Category 2 status meant the first repeat ECG was performed 20 minutes after arrival in the emergency department.

2. The first hospital ECG performed at 6:47am showed 2-3mm of ST elevation V2-V4, 1.5mm elevation V5 along with peaked waves and inferior ST depression. Dr Garrahy explained these changes are incremental to the 2mm ST elevation on the QAS ECG from 6:20am and were diagnostic of anterior ST elevation myocardial infarction (STEMI) and should have prompted an immediate call to interventional cardiology. In isolation, "high take off" might have been considered as noted by the admitting resident medical officer, but in sequence with the pre-hospital ECGs, the changes were diagnostic of STEMI. Dr Garrahy suspected the emergency department staff may not have studied the QAS pre-hospital ECGs to carefully track the obvious progressive changes; he considered that if they had, they would not have questioned "high take-off" as this appearance was not present on the first QAS ECG.

   Dr Garrahy did not accept that the layout-format of the first hospital ECG had anything to do with the failure to recognise the STEMI. Rather, emergency department staff need to study and note all available ECG readings including pre-hospital ECGs.

3. A further critical delay, perhaps the result of staff changeover from night to morning, was the failure to expedite and quickly follow up the requested CT aortogram. Dr Garrahy considered that while it was reasonable (given the developing back pain) to consider aortic dissection, this investigation should have been completed with at least a preliminary report within one hour of patient arrival, with a negative result prompting an immediate return to the patient to reconsider the diagnosis. Dr Garrahy noted the previous regional hospital ECGs, fax time stamped at 9:56am, showed a dramatically different tracing to that on 3 May.

4. The second hospital ECG recorded at 10:52am (two hours later than requested) demonstrates anterior Q waves, ST elevation and the report states anteroseptal infarct. Dr Garrahy suggests that AD's movement from the "hot" to the "cold" area in the emergency department at midday indicated that staff were not aware of the ECG evidence of the urgency of AD's clinical status.

There was a long delay to arrival in the cath lab with the time of needle to skin documented at 5:15pm. By this time, AD's ECG showed a fully evolved large anterior myocardial infarction with Q waves extending from leads I, aVL, V1-V4 with minimal R wave forces remaining in V5 and V6 – this constitutes a very large (late/completed) anterior myocardial infarction.

Dr Garrahy considered the aetiology of the coronary pathology, noting Dr PS had questioned whether given the behaviour of the artery during the procedure (its unusual tortuosity and response to stenting), spontaneous coronary artery dissection might have been the pathological process leading to occlusion. Noting AD did have triple vessel coronary artery disease, Dr Garrahy considered on balance, the arterial pathology was more likely to have been atherosclerotic. However, the final stent result was not fully satisfactory in that the distal flow in the left anterior descending artery was still reduced (TIMI II) and there was diffuse moderate abnormality in the proximal left anterior descending artery upstream from the stent. He noted that while the artery was still patent when the repeat angiogram was performed on

4 May, on those films there is a linear opacity in the proximal left anterior descending artery with patent distal stents, TIMI II flow and severe vasoconstriction, this appearance again raising the possibility that ?retrograde dissection of the proximal left anterior descending artery had occurred. However, regardless of the pathology (atherosclerotic or sudden coronary artery dissection), Dr Garrahy considered that angioplasty and stenting was the appropriate treatment for the presentation on 3 May 2017.

Dr Garrahy explained that timely reperfusion is the primary therapeutic goal for patients with STEMI. Delay to reperfusion increases the risk of death – for patients with acute STEMI subject to late (>6 hours) reperfusion following infarction, the short and medium term mortality rates exceed 10% (up from 3.4% for patients presenting <6 hours) and for patients with cardiogenic shock subsequent to infarction the 30 day mortality rate exceeds 50%.

Dr Garrahy considered that despite there being some "minor clinical distractions" in AD's presentation (dissection, arrhythmia), the failure to recognise the true diagnosis and the delay to treatment were unacceptable errors. While acknowledging it is possible AD may have died even had he received immediate angioplasty on arrival at the tertiary hospital on 3 May 2017, Dr Garrahy considered it was clear the delay to diagnosis and the delay to treatment very substantially increased the risk that this infarction event would prove fatal.

Tertiary hospital's response to Dr Garrahy's opinion

I provided the tertiary hospital with an opportunity to consider and respond to Dr Garrahy's report.

While not excusing the missed STEMI diagnosis, the hospital again highlighted the particular circumstances of AD's presentation on 3 May 2017 that helped confound it, namely the busyness of the emergency department that morning; the atypical nature of his chest discomfort and relatively benign appearance for a man suffering a STEMI; the ECG factors including atypical ECG formatting on the first hospital ECG and the lack of appropriate highlighting of the abnormal finding by the ECG algorithm.

I am advised the tertiary hospital has since undertaken the following quality and safety improvement activities:

- submitting recommendations to the hospital's Safety & Quality Committee for a body of work relating to auditing ECG machines, interpretation of ECG report and clear escalation processes for abnormal ECG findings;
- audit and review of all ECG machines and algorithms with sensitive computerised diagnostic skills (underway as at March 2018) – the findings of this audit will inform an assessment of which ECG machines perform the best reporting formats; and
- initiating a new process in the emergency department to require the most senior medical officer on duty to review every ECG and sign off a stamped template on the ECG (requiring name and signature and a written explanation of the importance or otherwise of any critical abnormal findings made by the automated ECG report) – discussions are underway to determine whether a similar process can be initiated across the hospital.

Dr PS reiterated that the PCI performed on 3 May was very difficult and that attainment of antegrade flow, albeit suboptimal, was a "relative success in very trying circumstances". He did not accept Dr Garrahy's opinion that the moderate upstream left anterior descending artery disease required treatment in the context of such a complex PCI requiring multiple stents already.

I am advised the tertiary hospital undertook open disclosure with AD's family on 12 December 2017.

Findings required by *Coroners Act 2003* s. 45

Identity of the deceased: [de-identified for publication purposes]

How he died: AD died from complications of a large anterior myocardial infarction. Despite there being early clinical evidence of the evolving STEMI available to both emergency department and cardiology staff, there was a significant delay in both diagnosis and treatment of the STEMI with emergency percutaneous catheter intervention. These delays occurred in the context of a very busy emergency department, AD's atypical clinical presentation and an apparent failure by staff to carefully study and compare the pre-hospital ECGs (performed by the paramedics) with the first and second hospital ECGs which, in sequence, were diagnostic of an evolving STEMI. By the time AD was transferred to the coronary cath lab over 10 hours since the onset of his symptoms, he had sustained a very large anterior myocardial infarction, the complications of which caused his death despite emergency interventions. Having regard to independent interventional cardiology opinion, I consider these delays contributed significantly to the outcome for AD by exposing him to the much higher mortality rates associated with late reperfusion following infarction; given AD's swift presentation to hospital that morning after the onset of his symptoms, earlier diagnosis and transfer to the cath lab would have greatly improved his chances of surviving this event.

I am satisfied that the tertiary hospital has carefully reviewed the issues leading to the delayed STEMI diagnosis and identified opportunities to reinforce the need for review and timely escalation of abnormal ECG reports. However, the most significant learning arising from the circumstances of AD's death lies in the importance of all clinicians, both junior and senior no matter how busy, carefully studying all available ECG reports including available pre-hospital ECGs.

Place of death: A tertiary public hospital

Date of death: 06 May 2017

Cause of death: 1(a) Multi-organ Failure
1(b) Cardiogenic Shock
1(c) Anterior myocardial infarction

I close the investigation.

Ainslie Kirkegaard
Coronial Registrar
CORONERS COURT OF QUEENSLAND
09 July 2018