QUEENSLAND COURTS

OFFICE OF THE STATE CORONER

FINDINGS OF INVESTIGATION

CITATION:	**Non-inquest findings into the death of KS**
TITLE OF COURT:	Coroner's Court
JURISDICTION:	Brisbane
DATE:	27 July 2015
FILE NO(s):	2013/4458
FINDINGS OF:	Ainslie Kirkegaard, Acting Coroner
CATCHWORDS:	CORONERS: Investigation, management of chest pain presentation at rural hospital, misdiagnosis of NSTEMI, complications of anticoagulation therapy, NSTEMI and NSTEACS Clinical Pathways

KS was a 69 year old man who died at an outer metropolitan hospital on 22 September 2013.

KS's death was reported to the coronial judicial registrar on 13 December 2013 by the hospital's executive after routine internal mortality review identified that the death may have been health care related.

KS's medical history

Review of KS's medical records from several hospitals shows he had a history including a previous heart attack several years ago, chronic atrial fibrillation treated with an anti-coagulant (Xarelto), high cholesterol, hypertension, and an infected disc in the spine secondary to a fracture that was complicated by osteomyelitis and a spinal abscess, for which he had taken the antibiotic flucloxacillin for a year.

KS's presentation to hospital on 21 September 2013

KS was brought in by ambulance to a rural hospital at 5:50pm on Saturday 21 September 2013 with increasing lethargy, general joint aches, pain in his right hip and difficulty mobilising since the previous Monday. He reported having a raised temperature over the previous 24 hours. He denied any chest pain, palpitations or any other specific symptoms. He had not taken his Xarelto (rivaroxaban) dose that day.

On examination, he had a temperature (38 degrees) and an increased heart rate (136 beats per minute) in atrial fibrillation. There were no other findings, including no neurological deficits. A chest x-ray was normal. An iSTAT troponin test showed that it was elevated (0.10).

The doctor who examined KS considered that given the past history of heart attack and ischaemic heart disease and as there was a raised troponin level (a biomarker for cardiac cell damage), she was treating KS's presentation as an acute coronary syndrome. The doctor did also consider that the troponin could also have been from a leak due to the increased heart rate.

She treated KS for a possible non-ST elevation myocardial infarct (NSTEMI). He was anti-coagulated with aspirin 300mg, clopidogrel 600mg and Clexane 90mg. He was also given intravenous antibiotics due to the possibility of an infection. Blood cultures were taken.

The rural hospital notes indicate the doctor spoke to a consultant from an outer metropolitan hospital emergency department about KS. He was then transferred to that outer metropolitan hospital for further management.

Unfortunately, the treating doctor's notes are not clear as to what was advised, apart from accepting KS to the outer metropolitan hospital.

KS arrived at the outer metropolitan hospital at 11:02pm. On review in the emergency department, the troponin rise was identified to be as a result of KS's increased temperature and history of septic infection, not cardiac related. KS was noted to be distracted when giving a history and it was queried whether he was hallucinating. KS did state that he had experienced vague chest tightness and

palpitations over the past few days with crampy abdominal pain. The clinical impression was that he may have a delirium secondary to the temperature, and a recurrence of the infected disc was considered. He was commenced on IV flucloxacillin in case. It was planned to admit him to the medical ward for further management.

About an hour later, while still in the emergency department, KS's conscious level deteriorated and he was intubated with the assistance of anaesthetic and intensive care staff. An urgent CT scan of the head showed a large intracerebral bleed which, after consultation with neurosurgeons, was considered inoperable and not survivable. After discussion with KS's family, active treatment was withdrawn. KS died at 9:56am on 22 September 2013.

The cause of death certificate issued by the treating team stated the cause of death as intracerebral haemorrhage due to or as a consequence of sepsis due to or as a consequence of NSTEMI, with atrial fibrillation, hypertension and ischaemic heart disease as other significant factors.

The treating team did not discuss the death with the coroner before issuing the cause of death certificate.

KS's death is reported to the coroner

KS's death was reviewed by the outer metropolitan hospital's routine death audit process. This review queried whether anti-coagulation therapy administered at the rural hospital may have increased the likelihood of intracerebral haemorrhage, particularly given KS's regular Xarelto medication. This concern prompted the hospital executive to formally report the death on 13 December 2013.

Consequently, there was no opportunity for a post-mortem examination to be performed as part of the coronial investigation.

Independent clinical review

I arranged for an independent doctor from the Department of Health Clinical Forensic Medicine Unit to review the medical records and advise whether KS's death may have been preventable.

The reviewing doctor advised that Xarelto is an anti-coagulant that works by directly inhibiting one of the clotting factors (Factor Xa) in the clotting pathway. Clopidogrel is also an anti-coagulant that works in a different way by inhibiting the aggregation of platelets, the cells in the blood that aggregate together to stop bleeding. Aspirin is also a platelet inhibitor. Clexane is an anti-coagulant that acts at a number of sites along the coagulation pathway.

The combination of Clexane, clopidogrel and aspirin has been used in the acute management of NSTEMI. The risk is always one of bleeding, such as gut or brain haemorrhages. Xarelto would have increased that risk to some degree. It was noted at the rural hospital that KS had not taken his Xarelto that day, so the treating doctor may have been reassured that its residual effects were not such as to outweigh the treatment of what was considered to be a heart attack (NSTEMI).

The reviewing doctor considered that KS appeared to be primarily suffering from the effects of an infection. This had probably increased his heart rate and led to the troponin leak; he already had ischaemic heart disease and the increased oxygen demands this created would have further compromised the heart muscle. The likelihood of infection was recognised by the rural hospital doctor who commenced IV antibiotics. Blood cultures were taken as well. The senior doctor at the outer metropolitan hospital had queried whether KS's backbone infection had re-emerged and prescribed an antibiotic specific for this type of infection (flucloxacillin for staphylococcal infection).

When considering the appropriateness of the cause of death diagnosis certified by the outer metropolitan hospital treating team, the reviewing doctor commented that it was possible that with the increased heart rate from the infection and the heart in atrial fibrillation, a clot may have formed and travelled to the brain causing an ischaemic stroke, which then transformed into a haemorrhagic stroke. This would indeed mean that the infection caused the stroke, although the haemorrhage would likely have been worse with the anti-coagulants than without them.

This prompted the reviewing doctor to review the blood cultures and other tests taken to check if there was indeed evidence of a bacteraemia.

Review of the blood culture result confirmed it does show a bacteraemia, a serious one in the form of Staphylococcus aureus. This would have been the cause of KS's sepsis, which was the primary diagnosis. This likely stressed an ischaemic heart already in atrial fibrillation that led to a troponin leak.

The reviewing doctor commented that the rural hospital doctor formed the view that KS's presentation was a non-ST elevation myocardial infarct (NSTEMI) and treated this condition accordingly with the anti-coagulants Clexane, clopidogrel and aspirin prior to transfer to the outer metropolitan hospital. It was not clear if the treating doctor did this independently or received advice to this effect from the outer metropolitan hospital. However these anti-coagulants were given on a background of KS already being on another anti-coagulant, Xarelto, albeit this not having been taken by him on that day.

The reviewing doctor considered it was reasonable to state that the sepsis and the increased rate of atrial fibrillation could have led to a clot forming resulting in an ischaemic stroke that became haemorrhagic. However, the anti-coagulant therapy would then have contributed to the death, in that the haemorrhage would have likely been greater than it otherwise would have been, though would not have directly caused it.

The reviewing doctor noted that recent years have seen the emergence of anti-coagulants that act at different points along the coagulation pathway and recommended that a clinical protocol be developed by the relevant Hospital & Health Service for the management of acute coronary syndrome in the rural setting, including diagnostic criteria and when to instigate appropriate anti-coagulant therapy.

The National Heart Foundation of Australia and the Cardiac Society of Australia

Guidelines for the Management of Acute Coronary Syndromes (ACS) (2006) with a Guideline Addendum in 2011 were identified as good resources on which to base such guidelines.

The reviewing doctor subsequently considered the outer metropolitan hospital Emergency Department Risk Stratification pathway for NSTEMI relied upon by the rural hospital doctor to treat KS.

The reviewing doctor noted that this clinical pathway essentially followed the 2006 'Guidelines for the Management of Acute Coronary Syndromes' as it related to NSTEMI, referred to in that publication as NSTEACS. This latter term is used to encompass cardiac conditions that are not myocardial infarctions, such as angina. It has been found that separating STEMI from NSTEACS is useful because emergency reperfusion is not necessary in the NSTEACS (unless ST elevation occurs later).

This means that further investigation is required in NSTEACS to stratify the patient's risk to determine the most suitable treatment; this is in terms of high risk, intermediate risk and low risk NSTEACS. Risk stratification is determined by the various features listed in the publication and is repeated in the outer metropolitan hospital Risk Stratification Pathway document, along with the recommended treatment.

The rural hospital doctor diagnosed NSTEMI /NSTEACS in KS due to his past cardiac history and his elevated troponin, which is a biomarker for cardiac cell damage, and which appears in the High Risk features section of the Risk Stratification Pathway document, under the heading 'elevated level of at least one cardiac biomarker'. Hence making such a diagnosis is understandable in the circumstances. She then treated him according to the protocol.

However in the 2011 'Addendum to the … Guidelines for the Management of Acute Coronary Syndrome' the use of troponin in the diagnosis is further refined to recommend that while a positive troponin identifies patients at increased risk, it does not of itself provide definitive evidence of myocardial infarction in the absence of diagnostic ECG changes. In this situation a positive troponin should, it recommends, be considered within the entire clinical context, with further investigations directed to all plausible diagnoses. Numerous non heart attack conditions are listed as to potentially causing elevated troponin levels, including pulmonary embolus, sepsis and aortic dissection.

The Addendum recommends that in the absence of diagnostic ECG changes, a troponin should be done at presentation to hospital and if positive, a repeat troponin should be done six hours after that initial troponin. If after the six hours there is a significant change in the troponin level (20%) then myocardial infarction is considered likely; if there is no change in the troponin level after this time then it is considered myocardial infarction is unlikely and to look for other causes of an elevated troponin.

However, if the clinical context is such that myocardial infarction is considered likely by the treating doctor in any event, then the Addendum algorithm states that

management decisions should not be delayed for the repeat troponin testing in six hours.

The reviewing doctor recommended that the review of the then current protocol include discussion of the newer anti-coagulants and also include clarifying the use of troponin testing and interpretation in the diagnosis of NSTEACS in light of the Addendum recommendations.

Relevant Hospital & Health Service (RHHS) response

I provided the RHHS an opportunity to respond to the issues raised and suggestions made by the independent clinical review.

The Executive Director, Clinical Governance Education & Research provided additional information to clarify the sequence of events leading to the decision to treat KS with anti-coagulant therapy. After discussion with the rural hospital doctor and the senior doctor consulted at the outer metropolitan hospital, it is now clear that the rural hospital doctor assessed KS as high risk from a cardiac point of view and treated him with the combination of aspirin, clopidogrel and clexane in accordance with the outer metropolitan hospital NSTEMI protocol. The treating doctor was familiar with this protocol having worked previously at the outer metropolitan hospital. She then had a discussion with the senior medical officer on duty on the outer metropolitan hospital emergency department.

As at February 2014, the outer metropolitan hospital emergency department clinical pathway for NSTEMI was being reviewed by two senior emergency department clinicians to include discussion of the newer anti-coagulants. It was intended to circulate the reviewed clinical pathway to the RHHS rural hospitals.

As at late September 2014, the RHHS advised that cardiologists and emergency medicine specialists at RHHS, together with cardiologists and haematologists from another Hospital & Health Service had been involved in developing an Acute Coronary Syndrome Pathway to take account of the new anti-coagulants and the use of troponin testing. The new pathway was expected to be completed over the 'next few months'.

Root cause analysis outcomes

The RHHS subsequently commissioned a root cause analysis (RCA) of the care KS received during his admission. This is a systemic analysis of what happened and why and is designed to make recommendations to prevent adverse health outcomes from happening again, rather than to apportion blame or determine liability or investigate an individual clinician's professional competence. It is conducted by a review team who had no involvement in the patient's care.

I received the final RCA report on 29 January 2015. I note that RCA took a year to complete.

The RCA report contains a detailed discussion of the sequence of events from the time KS arrived at the rural hospital. Key observations include the following:

- KS was reviewed by a registered nurse at the time of presentation (5:50pm) and

the medical officer was notified of his arrival.

- The medical officer did in fact see him at the time of presentation though her entry in the clinical record is for 6:10pm.

- The medical entry notes that KS had no neurological deficits. A chest x-ray was ordered which was noted as 'no abnormal deficits'. Blood tests were collected including blood cultures and a full blood count. A ward urine test was unable to be collected as KS did not pass urine. An Istat troponin test was taken and showed that the level was elevated to 0.10.

- The medical officer considered that because KS's past medical history of myocardial infarction, ischaemic heart disease and atrial fibrillation with an increased troponin, acute coronary syndrome with NSTEMI was the primary diagnosis.

- The medical officer ordered broad spectrum antibiotics for possible infection due to the elevated temperature.

- The rural hospital medication chart has medications (anti-coagulants and antibiotics) ordered and signed for by the medical officer and a different signature for being given but no time is recorded for their administration. A retrospective entry to the medical records by the registered nurse two days after KS's presentation notes the medications were administered to KS at the rural hospital over a two hour period and did not commence until 7:30pm. This was after the treating doctor had consulted with the emergency department consultant at the outer metropolitan hospital during which conversation it was advised that the anti-coagulants and antibiotics had been given. The reason for the delay in administering antibiotics was not ascertained as the registered nurse was unable to be interviewed by the RCA team.

- The ambulance was called for KS's transfer at 9:09pm, two hours after he was accepted for transfer by the outer metropolitan hospital emergency department. The hospital notes indicate that KS and his family took some time in deciding their preferred location for transfer and were considering a metropolitan private hospital. Once the decision was made, the ambulance was ordered and then took a further 45 minutes to arrive at the rural hospital.

- KS arrived at the outer metropolitan hospital emergency department at 11:00pm and was triaged as a category 3 rather than category 2 patient which is the recommended category for a patient with NSTEMI. KS was examined by an emergency registrar within 45 minutes of presentation which is outside the recommended triage timeframe. Following examination a diagnosis of possible sepsis was made with referral to the medical team for admission.

- A medical registrar reviewed KS at 2:30am on 22 September noting possible hallucinations. The clinical impression was delirium secondary to febrile episode due to possible recurrence of discitis. A differential diagnosis of encephalitis was made and KS was noted as possibly requiring an intensive care admission.

- The medical registrar noted that blood cultures had been taken at the rural hospital and listed a plan for blood cultures to be repeated in the morning and if febrile greater than 38 degrees. Intravenous Flucloxicillin 1g was ordered, however there is no signature to note if it was administered by either medical or nursing staff.

- Medical imaging of KS's back was planned for an MRI, and a CT head scan and chest x-ray. KS was ordered to have a nursing special and a telemetry bed. A clarification of KS's past Rivaroxaban dose was highlighted to occur prior to him having a lumbar puncture.

- During the medical consultation, the registered nurse noted a deterioration in KS's conscious level and he is reported to have dry retched two to three times. A decision was made to move him to a resuscitation bay in the emergency department and assistance was sought from both anaesthetics and the intensive care unit for a rapid sequence intubation and insertion of an arterial line at 3:30am.

- An urgent CT head scan was performed following a chest x-ray to confirm placement of the endotracheal tube (airway is the highest priority). The CT head scan confirmed a significant left frontal intracerebral haemorrhage with extension in ventricles causing hydrocephalus. KS's pupils were fixed and non-reactive.

- Consultation with neurosurgery at a tertiary Brisbane Hospital identified the cerebral haemorrhage was inoperable and it was recommended that KS be palliated in the emergency department. A family conference was held and the decision was made with the family and the medical team to extubate KS with the understanding he would die following extubation. He was extubated at 9:40am and died at 9:56am.

The RCA report contains a detailed discussion of the causal factors identified in respect of KS's death:

1. *Need to consider positive troponin within the entire clinical context*

The RCA team considered that the presenting clinical signs and symptoms of increased troponin with a temperature of 38, increased heart rate and history of septic infection in the absence of any cardiac biomarkers such as chest pain, that a primary diagnosis of a recurrence of septic infection should have been made. With this diagnosis, Flucloxacillin 2g IV should have been ordered and not the broad spectrum antibiotics and anti-coagulant therapy that was provided.

The RCA team acknowledged the coronial investigation had identified the issue that knowledge of an increase in troponin in the absence of any other cardiac biomarkers should not trigger urgent treatment of acute cardiac syndrome and that as per the 2011 Addendum to the Guidelines for the Management of Acute Coronary Syndrome 2006, sepsis must be considered.

The RCA recommended that the Department of Health Patient Safety Unit be asked

to develop and issue a State-wide Patient Safety Alert regarding recognition of sepsis in a patient with elevated troponin in the context of a presentation with fever, lethargy, known history of long term infection and antibiotic use and/or in the absence of any other ischaemic cardiac symptoms.

As at July 2015, the Patient Safety Unit advised it was in the final stages of completing a State-wide Patient Safety Communique regarding the recognition of sepsis in a patient with elevated troponin. This work was expected to be finalised by end July 2015.

2. *Medical officer understanding of the pharmaceutical actions of new oral anti-coagulants*

The RCA team identified that the knowledge of the rural hospital doctor as to the pharmaceutical actions of the new oral anti-coagulants was limited due to little previous experience with these medications. KS was taking rivaroxaban as prescribed by his private cardiologist. The increase in the Istat troponin level in conjunction with his cardiac history lead the treating doctor to treat him as a NSTEMI and order the administration of further anti-coagulant therapy. The limited understanding of the actions of new oral anti-coagulants was identified as a contributing factor in the outcome for KS.

The RCA recommended the development of a Workplace Instruction for the RHHS outlining that patients on any existing anti-coagulants who present with increased troponin in the absence of any other ischaemic cardiac symptoms are not be prescribed and/or administered any additional anti-coagulant therapy without consultation with a cardiologist, emergency consultant and/or intensive care physician.

After discussion with a range of medical specialists (cardiology, haematology, emergency, intensive care and pathology), the RCA identified that the new oral anti-coagulants need to be identified in a specific area on the National Inpatient Medication Chart (such as the area currently dedicated to Warfarin) to facilitate easier identification by all staff as to the serious nature of this category of medication. This recognises the known increased risk of internal bleeding from the drug and the absence of any known effective reversal agent. Although the current format of the National Inpatient Medication Chart was not a direct contributing factor in the outcome for KS, the RCA team considered it appropriate to recommend that this issue be referred to the Department of Health Medicines Regulation Unit which participates on the National Inpatient Medication Chart Committee.

3. *Delay in availability of blood cultures and commencing effective antibiotic treatment*

The RCA identified that weekend pathology services for both the rural hospital and the outer metropolitan hospital are extremely limited – there is a pathology delivery twice (approximately 9:00am and 5:00pm) on a Saturday and once (approximately midday) on a Sunday to either one of the two tertiary hospital pathology services in Brisbane. At all other times, pathology specimens are sent in an ambulance only when a patient is being transferred, so can wait many hours.

The blood cultures taken from KS at 6:50pm were not registered at a tertiary hospital pathology service until 11:25pm. Growth after 4.9 hours showed a Gram Positive Staphylococci infection. By this time, KS had already deteriorated and had not been given appropriate antibiotics (Flucloxacillan 2g) as per the CKN therapeutic guidelines for his condition. The RCA team considered that if the blood cultures had been available 4.9 hours after being taken, this would have had an impact on the outcome for KS as it would have enabled the infecting organism to be identified and the correct antibiotics to be commenced within an earlier timeframe.

The RCA recommended the establishment of a working group to review current weekend pathology arrangements and consider the increased patient acuity within the WMHHS and planned service development and expansion of clinical services.

The RCA team noted that KS's blood cultures were noted in his clinical record until 24 September 2014, three days after collection and two days KS died. The RCA recommended that RHHS investigate the use of an automated notification system to notify clinicians when clinical test results are available, such as that used by the Mater Health Service.

The RCA recommendations were accepted by the RHHS Chief Executive on 19 January 2015.

New Queensland Health Acute Coronary Syndrome Clinical Pathway

The RHHS Chief Executive also provided me with a copy of the Statewide Acute Coronary Syndrome Clinical Pathway being trialled in the HHS and advised this tool has been implemented across Queensland public hospitals and evaluation was planned for February 2015.

Further independent clinical advice

Noting that the trial Acute Coronary Syndrome clinical pathway provided to me did not address clinical decision making in the initial examination and assessment of patients presenting to non-tertiary hospitals, I arranged for the reviewing doctor to obtain further information about the outcome of the trial referred to by the RHHS Chief Executive.

The reviewing doctor clarified that the Department of Health has since implemented a suite of nine documents for 'non-interventional cardiac facilities' (essentially non-tertiary facilities) and the Clinical Pathways have been split into STEMI and Non-STEMI (i.e. NSTEACS - non-ST elevation acute coronary syndrome), meaning not all nine pathways are to be used for an individual patient - it depends on whether the diagnosis is STEMI or non STEMI (or neither).

The suite comprises:

1. Emergency Department Cardiac Chest Pain Risk Stratification Pathway (green) – the reviewed doctor noted this is a good and a clinically important document as it is directed at emergency doctors in non-tertiary hospital emergency departments to think about the cause of the chest pain from the

outset. On the back is a summary of a clinical journal article stratifying the risk for chest pain that is possibly cardiac.

2. Emergency Department Chest Pain Medical Assessment Tool – this is provided for the medical officer to document the history, examination, allergies, medications etc. and provides prompts that include other serious causes of chest pain (aortic dissection, AAA, pulmonary embolism) and whether the chest pain is in fact cardiovascular at all. This compliments the Risk Stratification document and arms the doctor with sufficient information to think about the cause of the chest pain and subsequent management.

3. The next two documents deal with the Intermediate Risk Chest Pain Clinical Pathway (with 'additional page') that prompts repeat troponin and ECG, as well as ongoing care requirements.

4. The 'STEMI Management Plan' is the next document and is used when the Risk Stratification Pathway has led to this diagnosis. On the second page it again summarises the clinical journal article which is very useful. On the first page it provides reperfusion guidelines, when thrombolysis is indicated and contraindicated and what to do, as well as monitoring and when to transfer for PCI.

5. The STEMI Clinical Pathway document (blue) is relevant to ongoing care of the STEMI patient.

6. The 'NSTEACS Management Plan' document (green) is used when the Risk Stratification Pathway has led to this diagnosis. Again it summarises the chest pain risk stratification journal article on the second page, and references the article. It provides the appropriate management guidelines for NSTEACS.

7. The NSTEACS Clinical Pathway (blue) alerts the clinician to the troponin testing (repeat in 6-8 hours) and to the aspirin, clopidogrel (or alternative) and to review the need for Clexane or heparin. The latter also refers to clinician to the NSTEACS Management Plan which details the doses of Clexane in patients with renal impairment. It then lists the pathway for the next 5 days. It also lists the variance codes (as does the 'STEMI Management Plan') that refer to actions to be taken in the event of further symptoms e.g. recurrent chest pain, arrhythmias, heart failure.

8. The STEMI/NSTEACS Pathways document (pale blue) is labelled 'additional page' and appears designed for further entries after 5 days, for either STEMI patients or NSTEACS.

The reviewing doctor particularly liked the initial stratification document and chest pain medical assessment tool as they direct the medical officer to make the most likely diagnosis in the circumstances - if it is not cardiac then the rest of the documents are not required and management is in accordance with whatever that diagnosis is. This relates in a way to the Workplace Instruction proposed by the RCA team to alert clinicians to other causes of troponin elevation, such as sepsis (and renal failure).

The reviewing doctor was satisfied that this is a comprehensive and appropriate response to the assessment and management of chest pain in non-tertiary hospitals.

Conclusion

KS died from haemorrhagic stroke arising as a complication of sepsis and exacerbated by anti-coagulant therapy administered in the context of a likely misdiagnosis of his initial presentation to the rural hospital as a non-ST elevation myocardial infarction. Although KS's death should at least have been discussed with a coroner before the cause of death certificate was issued (because the treating team at the outer metropolitan hospital were aware of the initial diagnosis and treatment at the rural hospital), his death was appropriately reported as soon as the local death audit process identified the clinical management issues at the rural hospital. I am satisfied that the relevant Hospital & Health Service has since undertaken a thorough investigation of these issues (clinical misdiagnosis, lack of clinician understanding of actions of newer anti-coagulants, weekend pathology service deficiencies) and is taking steps to implement appropriate action to address them in response to the circumstances of KS's death. That said, it is disappointing it has taken the RHHS nearly two years to achieve those outcomes. The subsequent implementation by the Department of Health of a suite of statewide clinical pathways for non-interventional cardiac facilities, particularly those guiding the risk stratification and medical assessment of chest pain, will provide medical officers with comprehensive and appropriate guidance to improve the initial assessment and subsequent management of patients presenting with possible cardiac signs and symptoms.

Ainslie Kirkegaard
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27 July 2015