CARDIOLOGY RELATED COMPLAINTS

A Case of acute coronary syndrome

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Background and Chronology:

Mrs W (Born in February 1947) was documented to have been independently mobile and had a medical history of only osteoporosis, for which she was taking regular tablets of Alendronic acid, calcium and vitamin D. She was 67 years of age when she attended the A&E department of her local Hospital NHS Trust (the Trust) at 21:55 on Wednesday 12th November 2014, following a referral from her GP. She was then complaining of symptoms of central chest pain/tightness that was documented to have started at 12 am on Sunday (presumably 9th November 2014) lasting most of that day and then settled. The symptoms returned at 10 am on Monday (presumably 10th November 2014). The GP had already performed a Troponin blood test which revealed a significantly high level of 15,641 ng/L (normal value < 40 ng/L). Mrs W had no documented associated symptoms such as nausea, sweating, palpitations, dizziness, loss of consciousness. She was also documented to have been experiencing non-productive cough for the previous several weeks. Clinical observations (heart rate, blood pressure, breathing rate, temperature and oxygen saturation level) were largely unremarkable. Clinical examination was also unremarkable. Chest X ray revealed clear lung fields and her ECG demonstrated no significant changes to indicate a STEMI that could require emergency PCI. The full blood count and kidney function were shown to have been normal on blood tests.

Given the history of chest pain and the significantly raised Troponin blood level, Mrs W was diagnosed and treated as

NSTEMI. She was reviewed by the on-call consultant physician (12th November 2014 at 23:00) who upheld the diagnosis of NSTEMI and documented that Mrs W was clinically stable, had no further chest pain, and that she had no clinical signs of heart failure. It was documented that she was treated with Aspirin, Clopidogrel, beta blocker and statin therapy. Cardiac monitoring, along with transfer to the Cardiac Care Unit (CCU), was instructed.

Later (12th November 2014, no time specified), Mrs W was reviewed by the Cardiology team who agreed with the diagnosis of NSTEMI and the management initiated by the medical team. A diagnostic coronary angiogram procedure was arranged to be performed during Mrs W's inpatient stay.

On 13th November 2014, Mrs W underwent an echocardiogram scan, which reported normal function of all heart chambers and valves. On that same day, she was transferred to the Regional University

Hospital where she underwent a coronary angiogram procedure (performed via the right wrist by Dr N, Consultant Cardiologist), which revealed entirely normal coronary arteries. The angiogram report requested that Mrs W be investigated for other causes of her raised Troponin (other than a heart attack) given her normal echocardiogram and coronary angiogram results. Mrs W was transferred back to the Trust on the same evening.

The observation chart entries confirm that Mrs W continued to remain clinically stable until she suddenly collapsed with cardiac arrest between 8:24 am on 14th November 2014. Cardio-Pulmonary Resuscitation (CPR) was commenced and two DC shocks were given for VF before the heart rhythm then changed to PEA. Several adrenaline doses were given as part of the resuscitation protocol. A working diagnosis of PE was made (presumed to be a massive PE given the extremely critical clinical situation) and Mrs W was therefore commenced on Alteplase infusion at 8:45 am. A bedside echocardiogram scan was subsequently performed almost immediately, and revealed good function of the heart but also evidence of pericardial effusion, which was attributed to the chest compressions of the CPR. No evidence of cardiac tamponade was documented to have been seen on that scan. At 8:47 am, Mrs W's heart started beating in response to the then continuing resuscitation attempts, at which time the Alteplase infusion was stopped. The blood pressure was documented to have been very low at the time measuring 85/56 mmHg (normal range: 100-140/60-85 mmHg) and Mrs W was therefore commenced on adrenaline infusion, so as to increase her blood pressure, and was transferred to the Intensive Care Unit (ICU).

Upon arrival at ICU, at 9:14 am on 14th November 2014, Mrs W had a further cardiac arrest and CPR was restarted. However, resuscitation attempts were unsuccessful after 10 minutes and a decision was made for CPR to be stopped. The decision was documented to have been jointly made by Dr W (Consultant Anesthetist) and Dr I (Consultant Cardiologist). Mrs W was therefore pronounced dead thereafter.

A post mortem examination, undertaken on 17th November 2014, concluded that Mrs W's cause of death was haemopericardium and haemothorax due to a tear of the ascending aorta (referred to as: aortic dissection). This means that Mrs W suffered a tear affecting the lining of the aorta, which led to bleeding around the heart and around the right lung. Aortic dissection was neither suspected by the clinical team nor revealed by any of the tests undertaken by Mrs W during her admission.

Mrs W's family were unhappy with the care provided to her by the Trust and made an official complaint. No local resolution was reached and the compliant was therefore referred to the PHSO for investigation.

Before answering the specific questions raised below, I would like to state some facts about aortic dissection for the purpose of proper understanding and contextualization:

Acute aortic dissection is an extremely rare yet serious medical condition that requires urgent surgical intervention. Initial clinical suspicion is key to a quick diagnosis The European Society of Cardiology (ESC) Guidance document on the diagnosis and management of aortic diseases states (quote):

"The incidence of aortic dissection is estimated at six per hundred thousand persons per year (0.006% or 60 people in 1 million per year). This incidence is higher in men than in women and increases with age. The prognosis is poorer in women, as a result of atypical presentation and delayed diagnosis" - page 2890 of the guidance document.

Chest pain of specific characteristics is the main presenting symptom in acute aortic dissection. The diagnosis should be suspected if the chest pain is described as abrupt & severe, sharp, ripping, tearing or knife-like. The location and radiation of the pain are less specific features and can't be relied upon in forming the clinical suspicion. Please see the below quote from the ESC guidance document (reference 23):

"Chest pain is the most frequent symptom of acute aortic dissection. Abrupt onset of severe chest and/or back pain is the most typical feature. The pain may be sharp, ripping, tearing, knife-like, and typically different from other causes of chest pain; the abruptness of its onset is the most specific characteristic. The most common site of pain is the chest (80%), while back and abdominal pain are experienced in 40% and 25% of patients, respectively" - page 2890 of reference 23.

Chest pain is also the main clinical symptom of NSTEMI, a much more common condition than acute aortic dissection. The annual incidence of NSTEMI is three per one thousand persons (0.3% or 3000 people in 1 million) , making NSTEMI 50 times more common than acute aortic dissection. In NSTEMI, the chest pain is described as heaviness, indigestion-like or a gripping sensation across the chest, with a relatively gradual onset and typical radiation to the left arm and jaw.

If a patient presents with chest pain suggestive of acute aortic dissection, an urgent CT scan of the aorta would be indicated to confirm the diagnosis, and urgent aortic repair surgery would subsequently be undertaken/considered. It must be emphasised that the prognosis is extremely poor without surgical intervention, and it still remains significant – although comparatively much better - after surgical intervention. Please see the quoted statement below from page 2895 of reference 23:

"Surgery is the treatment of choice. Acute Type-A aortic dissection – which is what Mrs W had - has a mortality of 50% within the first 48 hours if not operated. Despite improvements in surgical and anaesthetic techniques, perioperative mortality (25%) and neurological complications (18%) remain high. However, surgery reduces 1-month mortality from 90% to 30%."

Conversely, if a patient presents with chest pain suggestive of NSTEMI, an urgent coronary angiogram would be indicated, and PCI (reference 4) or CABG would subsequently be undertaken if found to be required. An urgent CT scan of the aorta isn't routinely performed in patients whose chest pain is deemed to be suggestive of NSTEMI. i.e. there is no clinical guidance that promotes the routine ruling-out of acute aortic dissection (via an urgent CT scan of the aorta) in patients who are suspected to be experiencing NSTEMI.

In addition to the above, an abnormal Troponin result is found in all NSTEMI cases (100%) as opposed to only ¼ of acute

aortic dissection cases (25%). i.e. the annual incidence of acute aortic dissection with an associated abnormal Troponin result is only 0.0015% or 15 people in 1 million, which is 200 times less common than NSTEMI. Below is a statement from the ESC guidance referenced 23 (page 2890):

"If systematically assessed, Troponin elevation may be found in up to 25% of patients admitted with Type-A aortic dissection. Both Troponin elevation and ECG abnormalities, which may fluctuate over time, may mislead the physician to the diagnosis of acute coronary syndromes (meaning NSTEMI) and delay proper diagnosis and management of acute Aortic Dissection."

Moreover, Mrs W's chest pain was described as 'central chest tightness' that had lasted for several hours for each of the two days leading to the admission, thereby implicitly indicating that the pain onset must have been insidious rather than severe/abrupt, for otherwise she would have presented to A&E much sooner after the pain onset. This means that Mrs W's chest pain was more suggestive of NSTEMI, particularly given the accompanying significantly abnormal Troponin result that was available to the admitting team on her arrival at the hospital. In other words, while Mrs W turned out to have suffered from acute aortic dissection, she seemed to have presented with chest pain whose description was more suggestive of NSTEMI. Given the associated significantly abnormal Troponin result, it was reasonable of the team not to have suspect aortic dissection, and to have diagnosed and treated NSTEMI instead.

Questions & Answers:

Question 1:

Overall, did the staff at the Trust take an appropriate history from Mrs W in line with established good practice and/or any applicable guidance?

Answer to Question 1:

YES.

As explained in the last paragraph of page 5 above, Mrs W's chest pain was described as 'chest tightness' and appeared to have pursued a prolonged insidious onset over several hours across two days. This is not typical of acute aortic dissection pain, which would typically be described as sharp, severe, abrupt, tearing and knife-like.

In short, Mrs W presented with chest pain that wasn't typical of acute aortic dissection, hence the erroneous diagnosis of NSTEMI, which was justified at the time given the clinical context. The possibility of such diagnostic errors in patients presenting with atypical symptoms is real, and is recognised as a poor prognostic marker by the ESC guidance quoted in fifth paragraph of page 4 above.

Question 2:

Should staff at the Trust have asked Mrs W specific questions about whether she had back pain and if her pain was moving anywhere else?

Answer to Question 2:

The staff at the Trust could - rather than should - have asked about pain radiation to the back. However, given the nature of Mrs W's pain in terms of its character (tightness) and onset (insidious), NSTEMI would have still been suspected as the main diagnosis even if the pain was radiating to the back. This is because NSTEMI chest pain can also radiate to the back, and because only 40% of acute aortic dissection patients experience back pain. i.e. the presence or absence of associated back pain shouldn't be used to differentiate between NSTEMI and aortic dissection. Rather, one should adhere to the pain features described in the ESC guidance quoted in paragraph 7 of page 4 above.

Therefore, the presence or absence of back pain wouldn't have helped the team diagnose Mrs W's aortic dissection.

Question 3:

Were appropriate investigations arranged for Mrs W, including whether she should have had a CT scan, in line with established good practice and/or any applicable guidance?
Answer to Question 3:
Given the atypical nature of Mrs W's chest pain and overwhelming indication that she was suffering from NSTEMI, it was reasonable that a CT of the aorta had not been performed. Please refer to the above summary of evidence-based factual statements on aortic dissection for details.
Question 4:
Was it in line with established good practice and/or any applicable guidance for staff to consider that Mrs W had had a heart attack rather than suspect an aortic dissection?
Answer to Question 4:
YES.
Please refer to the detailed explanation provided in page 5.
Question 5:
Should staff have considered more serious diagnoses, such as aortic dissection, following Mrs W's normal angiogram?
Answer to Question 5:
NO.
Patients do not routinely get investigated for the possibility of aortic dissection if their coronary angiogram reveals normal findings following an admission with suspected NSTEMI. This is because it is not uncommon for NSTEMI patients to have normal coronary angiogram results. In fact, a coronary angiogram is normal in about 15% of NSTEMI patients, as stated in page 3035 of the ESC guidance document referenced 24. Therefore, the incidence of normal coronary angiogram in NSTEMI patients is still much more common than the incidence of acute aortic dissection with an associated high Troponin result. In addition, Mrs W was constantly noted to have been stable and pain free during the vast majority of her hospital stay, which gave the reassuring impression that she was responding to her NSTEMI treatment.
Question 6:
If the Trust should have identified Mrs W's aortic dissection, could her death have been avoided? (Please respond to this question taking into account the balance of probabilities)Answer to Question 6:
NO.
Given the clinical context described in the Background and Chronology section above, it would have been extremely challenging for the Trust to have diagnosed acute aortic dissection. The clinical context at the time strongly indicated that Mrs W was suffering from NSTEMI, which was managed well and as per the ESC guidance. Most other clinical teams would have pursued a similar management course to that of the Trust's, and Mrs W would have sadly faced the same fate. It is difficult to foresee a scenario whereby Mrs W could have been diagnosed correctly early enough before her collapse and demise on 14th November 2014. Therefore, on the balance of probabilities, Mrs W's death was unavoidable.

Conclusion:

Mrs W presented with atypical chest pain for aortic dissection, which – together with her significantly raised Troponin result on presentation – made it extremely challenging to diagnose acute aortic dissection, and at the same time made it extremely probable that she was suffering from NSTEMI.

It was reasonable of the Trust to have diagnosed Mrs W and treated her as NSTEMI.

The diagnosis of acute aortic dissection could not have been foreseen during Mrs W's life given the clinical context.
On the balance of probabilities, Mrs W's death was unavoidable.