CASE REPORT

Atherothrombotic non-ST segment elevation myocardial infarction in a 42-year-old white female with no known cardiovascular risk factors

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SUMMARY

Coronary artery disease (CAD) in the absence of known cardiovascular risk factors is rare. This is the case report of a 42-year-old white female who was admitted with a non-ST segment elevation myocardial infarction (NSTEMI). The patient had seen her general practitioner (GP) in the 2 weeks prior to this with exertional chest pain. She had no known risk factors for cardiovascular disease and other possible underlying aetiologies were ruled out. The patient underwent percutaneous coronary intervention (PCI) to the culprit lesion in the left anterior descending artery. She was discharged on guideline-directed medical therapy and the Dianette contraceptive pill she was taking was stopped. This case serves to remind us that CAD can occur in a variety of patients including premenopausal females and in the absence of known risk factors. It also emphasises that patients presenting with a convincing history of typical angina require prompt investigations, regardless of their apparent level of risk.

BACKGROUND

Coronary artery disease (CAD) is rare in premenopausal women, particularly in the absence of cardiovascular risk factors.¹ The incidence of acute myocardial infarction in women aged 40–44 with no risk factors is estimated at 2.13 events per 100 000 women years.² This case demonstrates the most severe form of CAD, acute myocardial infarction, in a 42-year-old white female with no traditional risk factors.

There are reports in the literature of acute myocardial infarction in premenopausal women in the presence of cardiovascular risk factors as well as due to rarer causes such as hypercoagulable states, illicit drug use, hyperhomocysteinaemia and spontaneous coronary artery dissection.^{3–8} However, to the best of my knowledge, this is the first individual case report of its kind: an atherothrombotic myocardial infarction in a premenopausal woman in the absence of traditional risk factors and without a rarer underlying aetiology.

CASE PRESENTATION

A 42-year-old white female presented to the accident and emergency (A&E) department with sudden onset chest pain at rest. This had been present for approximately an hour. It was described as a severe heaviness in the centre of the chest which radiated into the neck and left jaw. She also reported mild shortness of breath and nausea.

The patient had several episodes of intermittent chest pain in the 2 weeks prior to presenting to A and E. These pains were brought on by ~30 min of walking and relieved by rest. She consulted her general practitioner (GP) at this stage and was prescribed omeprazole for suspected reflux. This produced no subsequent improvement in her symptoms. An ECG was not performed at this stage.

The patient had no medical history and took no regular medications other than the Dianette contraceptive pill. There was no family history of coronary artery disease, hypercholesterolaemia or sudden death.

The patient worked as a prison officer and had never smoked. There were no preceding work-related stressors or bereavements. She reported drinking ~4 units/week and denied any recreational drug use. Her body mass index was 19 kg/m² and she exercised regularly.

On examination, the patient had a blood pressure of 134/72 mm Hg and a regular pulse of 86 bpm. Heart sounds were clearly audible with no added sounds. The internal jugular vein was not raised, there was no peripheral oedema and there were audible vesicular breath sounds with good air entry at the lung bases.

INVESTIGATIONS

12-lead ECG ([figure 1](#))

Full blood count—no abnormalities detected (NAD)

Urea and electrolytes—NAD

Liver function tests—NAD

Total cholesterol (mmol/L)—4.2 (<5)

Total high-density lipoprotein (HDL)(mmol/L)—1.6 (>1)

Total low-density lipoprotein (mmol/L)—2.6 (<3)

Total cholesterol: HDL ratio (mmol/L)—2.6 (<4)

Troponin T (ng/mL)—6 hours: 89, 12 hours: 112 (<0.01)

Blood glucose (non-fasting sample) (mmol/L)—6.2 (<11.1)

Thyroid-stimulating hormone (mU/mL)—2.3 (0.4–4.5)

Follicle-stimulating hormone (mU/mL)—7.6 (4.7–21.5)

Coronary angiography ([figure 2](#)).

Echocardiogram – mild anterior wall hypokinesis.

No left ventricular systolic dysfunction

DIFFERENTIAL DIAGNOSIS

The history of chest pain that the patient gave was highly suggestive of myocardial ischaemia. Initially, when the patient consulted her GP, the pain was characteristic of that caused by a stable coronary artery

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Figure 1 ECG on admission to accident and emergency department demonstrating T wave inversion in leads 3 and AVF and T wave flattening in V4–V6, findings consistent with myocardial ischaemia.

plaque: a heaviness in the chest with predictable onset which disappeared with rest. By the time the patient presented to A and E, the pain had worsened significantly; it was more severe, present at rest and of an hour's duration. This suggested progression of disease with plaque rupture and resulting acute coronary syndrome (ACS).

Other non-cardiac causes to consider for this patient's chest pain were gastro-oesophageal reflux disease (GORD) and a pulmonary embolism (PE). GORD and stable angina can be hard to distinguish as GORD can also produce central chest pain. However, GORD is more often described as a burning pain rather than a heaviness in the chest and is unlikely to be so closely correlated with exertion. An improvement with omeprazole might also have been expected if GORD was the cause of this chest pain. Furthermore, GORD is unlikely to progress acutely to severe chest pain at rest, with radiation to the neck and jaw and associated shortness of breath.

PE is a diagnosis worth considering, particularly in a female patient >40 taking the combined oral contraceptive pill (COCP). A PE could account for the presentation at A and E: acute onset of chest pain at rest with accompanying shortness of breath. However, the pain was non-pleuritic and radiated into the neck and jaw, which would make a PE less likely than ACS.

Clinical examination would not help differentiate between these differentials further. The history on presentation to A and E was fairly convincing of ACS and basic investigations were therefore performed to confirm the diagnosis (see Investigations). The ECG findings and troponin levels on admission to A and E confirmed that the cause of the chest pain was a non-ST segment elevation myocardial infarction (NSTEMI).

TREATMENT

The patient was given aspirin 300 mg, clopidogrel 300 mg and glyceryl trinitrate spray before being transferred to the cardiology unit. A total of 8000 units of intravenous heparin were administered and a 6 F diagnostic catheter was inserted via the right radial artery, revealing a 100% occlusion of the proximal left anterior descending artery (figure 2). The remainder of the coronary vessels were normal. Direct stenting to the lesion with a 3.0×15.0 drug eluting stent (figure 3) was performed and the proximal edge was post dilated with a 3.0 non-compliant balloon. This produced a good result (figure 4).

OUTCOME AND FOLLOW-UP

The patient was discharged from the cardiology unit on guideline directed medical therapy with no complications post percutaneous coronary intervention.¹

Figure 2 Angiography demonstrating 100% occlusion of the proximal left anterior descending (LAD) artery.

Figure 3 Stenting of the proximal left anterior descending (LAD) lesion with a 3.0×15.0 drug eluting stent.

Figure 4 Left anterior descending (LAD) flow restored following stenting.

She was seen in the cardiology clinic at 6 weeks postdischarge where she reported full compliance with her medications and no further episodes of chest pain.

Blood tests were performed to rule out autoimmune pathology, hyperhomocysteinaemia, vasculitides and thrombophilia. The results of these were all normal.

DISCUSSION

This patient presented to her GP with chest pain in the 2 weeks prior to this NSTEMI. The history of this chest pain was in line with the National Institute for Health and Care Excellence's (NICE's) description of typical angina: a constricting discomfort in the chest precipitated by physical exertion and relieved by rest.⁹ The GP treated this with omeprazole for suspected reflux without further investigations for a cardiac cause. NICE's table (figure 5) estimates that 10–20% of patients of the same age, sex and lack of risk factors presenting with this history would have CAD.⁹

Figure 5 The National Institute for Health and Care Excellence estimates of percentage of people estimated to have coronary artery disease (CAD) according to typicality of symptoms, age, sex and risk factors.

Age	Non-anginal chest pain				Atypical angina				Typical angina			
	Men		Women		Men		Women		Men		Women	
	Lo	Hi	Lo	Hi	Lo	Hi	Lo	Hi	Lo	Hi	Lo	Hi
35	3	35	1	19	8	59	2	39	30	88	10	78
45	9	47	2	22	21	70	5	43	51	92	20	79
55	23	59	4	25	45	79	10	47	80	95	38	82
65	49	69	9	29	71	86	20	51	93	97	56	84

For men older than 70 with atypical or typical symptoms, assume an estimate >90%.

For women older than 70, assume an estimate of 61–90% EXCEPT women at high risk AND with typical symptoms where a risk of >90% should be assumed.

Values are % of people at each mid-decade age with significant coronary artery disease (CAD).

Hi = high risk = diabetes, smoking and hyperlipidaemia (total cholesterol >6.47 mmol/L).

Lo = low risk = none of these three.

The shaded area represents people with symptoms of non-anginal chest pain, who would not be investigated for stable angina routinely.

Note: These results are likely to overestimate CAD in primary care populations.

If there are resting ECG ST-T changes or Q-waves, the likelihood of CAD is higher in each cell of the table.

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It is impossible for us to predict which 10–20% of these patients actually have CAD, which patients will go on to develop more severe complications of their CAD such as an acute MI, and indeed over what time span these complications may occur. However, studies have shown a history of typical angina to be the best indicator of significant CAD when compared to other parts of the history such as age, sex, prior MI and cardiovascular risk factors, for example, hypertension and hypercholesterolaemia.^{10–12} Furthermore, the presence of traditional cardiovascular risk factors alone is an insufficient way of assessing risk of CAD in women.¹³ Different risk factors have different impacts on CAD risk in women depending on age and menopausal status also has an important role. There may also be other risk factors for CAD which we are yet to get a handle on which act independently, or interact with traditional risk factors.¹³

The lesson here therefore is that a modified approach in women is required. A history of typical angina should prompt investigation (including an ECG and blood tests) and referral, regardless of the patient's lack of risk factors. In addition, young women with a smoking history, or postmenopausal women with elevated total cholesterol may be at greater risk of plaque erosion or rupture and subsequent coronary thrombosis.¹³ Recognition of these points with appropriate early investigations will allow timely intervention of any underlying CAD and prevention of further complications such as acute MI.

This patient had been taking Dianette since the age of 26 for long-standing acne. Dianette is a COCP containing 2 mg of cyproterone acetate and 35 mg of ethinyl oestradiol.¹⁴ I wondered whether Dianette may have been implicated in this patient's MI; there is no evidence to suggest that this may be the case.¹⁴ Other COCPs, however, have been shown to carry a small increase in risk of MI, with those containing >30 µg conferring the greatest increase in risk.^{15 16}

It therefore seems sensible, particularly in women >40 where the background risk of MI may be higher,² to err on the side of caution and prescribe an oral contraceptive pill with a low risk of MI. Progestogen only pills carry no significant increase in MI risk.¹⁵ Of the combined pills, those containing a low ethinyl

Patient's perspective

- ▶ It was only after being discharged from hospital the seriousness of what had occurred hit me and I struggled to comprehend how this could have happened to me when I had always been so health conscious after my mother sadly died from cancer aged 53. I have never smoked, drink only in moderation, have never been overweight, exercise regularly and have no family history of heart disease. This made it all the more difficult to accept. I had always been a confident, outgoing person but following this I became withdrawn, depressed and lost all my confidence. I also began to suffer with health anxiety.
- ▶ I felt it was important that I accepted all help and support offered to me at this time. I attended the 16 sessions of cardiac rehabilitation which improved my confidence enormously as initially I was terrified of attempting any physical exertion in case my symptoms returned. Keeping fit has always been an important part of my life and I was worried I would not be able to continue this.
- ▶ I was also attending counselling at this time, as despite my family and friends being incredibly supportive, I was reluctant to let them know how I was actually feeling. I knew they were worried enough about the physical consequences and did not want to burden them any further. I found it difficult to sleep for the first few weeks after being discharged from hospital and found this the loneliest time when everything was quiet, everyone was asleep and I only had my own thoughts for company.
- ▶ I am extremely grateful for the support I received throughout this time and I doubt I would have recovered so well without it.
- ▶ I returned to work 6 months after the heart attack on a phased return for 4 weeks. I have now returned to my full duties as a senior prison officer. I feel I have returned to the same level of fitness that I had prior to my heart attack and I feel I have come out of the other side of this experience a stronger person.'

Learning points

- ▶ Coronary artery disease (CAD), although rare, can occur in patients without risk factors.
- ▶ The presence of traditional cardiovascular risk factors alone is an insufficient way of assessing risk of CAD in women. Risk assessment in women should therefore be modified.¹³
- ▶ Regardless of a lack of traditional cardiovascular risk factors, a history of typical angina should prompt referral and appropriate investigation.
- ▶ There is a small increase in risk of MI with some oral contraceptive pills. In women >40 where background risk of MI is higher,² the use of pills with a lower risk of MI should be advocated.
- ▶ Contraceptive pills with little or no increase in risk of MI are progestogen only pills and combined pills containing 20 µg of ethinyl oestradiol.⁸

oestradiol content (20 µg) confer the smallest increase in risk. The difference in MI risk according to progestogen type is not significant.^{15 16} An older progestogen such as levonorgestrel, however, is preferable due to the lower overall combined risk of venous and arterial thrombosis.^{16–19}

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