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CT coronary angiography in patients with suspected angina due to coronary heart disease (SCOT-HEART): an open-label, parallel-group, multicentre trial

The SCOT-HEART investigators*  

Summary  
Background The benefit of CT coronary angiography (CTCA) in patients presenting with stable chest pain has not been systematically studied. We aimed to assess the effect of CTCA on the diagnosis, management, and outcome of patients referred to the cardiology clinic with suspected angina due to coronary heart disease.

Methods In this prospective open-label, parallel-group, multicentre trial, we recruited patients aged 18–75 years referred for the assessment of suspected angina due to coronary heart disease from 12 cardiology chest pain clinics across Scotland. We randomly assigned (1:1) participants to standard care plus CTCA or standard care alone. Randomisation was done with a web-based service to ensure allocation concealment. The primary endpoint was certainty of the diagnosis of angina secondary to coronary heart disease at 6 weeks. All analyses were intended to treat, and patients were analysed in the group they were allocated to, irrespective of compliance with scanning. This study is registered with ClinicalTrials.gov, number NCT01149590.

Findings Between Nov 18, 2010, and Sept 24, 2014, we randomly assigned 4146 (42%) of 9849 patients who had been referred for assessment of suspected angina due to coronary heart disease. 47% of participants had a baseline clinical diagnosis of coronary heart disease and 36% had angina due to coronary heart disease. At 6 weeks, CTCA reclassified the diagnosis of coronary heart disease in 558 (27%) patients and the diagnosis of angina due to coronary heart disease in 481 (23%) patients (standard care 22 [1%] and 23 [1%]; p<0·0001). Although both the certainty (relative risk [RR] 2·56, 95% CI 2·33–2·79; p<0·0001) and frequency of coronary heart disease increased (1·09, 1·02–1·17; p=0·0172), the certainty increased (1·79, 1·62–1·96; p<0·0001) and frequency seemed to decrease (0·93, 0·85–1·02; p=0·1289) for the diagnosis of angina due to coronary heart disease. This changed planned investigations (15% vs 1%; p=0·0001) and treatments (23% vs 5%; p=0·0001) but did not affect 6-week symptom severity or subsequent admissions to hospital for chest pain. After 1·7 years, CTCA was associated with a 38% reduction in fatal and non-fatal myocardial infarction (26 vs 42, HR 0·62, 95% CI 0·38–1·01; p=0·0527), but this was not significant.

Interpretation In patients with suspected angina due to coronary heart disease, CTCA clarifies the diagnosis, enables targeting of interventions, and might reduce the future risk of myocardial infarction.

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Introduction Many patients with recent onset angina have a cardiac event within 1–2 years1 and will benefit from intervention.2 Although the rapid clinical assessment of patients with suspected angina due to coronary heart disease is successful at identifying high-risk individuals,3 many patients are still misdiagnosed. Patients diagnosed with non-cardiac chest pain account for a third of patients who subsequently die from cardiovascular disease or have an acute coronary syndrome during 5 years of follow-up.4 Improved diagnostic accuracy and risk stratification is needed, especially in younger patients.5

CT coronary angiography (CTCA) has a sensitivity of 89% and specificity of 96% for the detection of coronary heart disease.6 However, concerns have been raised about its generalisability in patients with cardiac disease, with the potential for poor image quality in those with obesity, coronary calcification, or arrhythmia, and the high levels of radiation exposure (about 15 mSv).7 However, the evolution of scanning technology has led to improved spatial and temporal resolution with lower radiation doses that should translate into a more effective and safer imaging strategy.

Most clinical trials have focused on assessing the accuracy and comparability of CTCA for the identification of coronary heart disease.8–10 In one study,11 investigators assessed the effect of CTCA on the management of low-risk patients presenting to the emergency department with acute chest pain. Data from the study suggested improved decision making but an increase in further downstream testing and health-care costs without any
For the ASSIGN score see http://assign-score.com

Research in context

Evidence before this study
Between 2007 and 2010, we searched PubMed for reports published in English with the search terms “computed tomography”, “coronary angiography”, “angina pectoris”, and “coronary heart disease”. We identified a high-quality health technology assessment comprehensive systematic review that assessed the role of 64-multidetector CT coronary angiography (CTCA). In keeping with previous analyses from the European Society of Cardiology, the review confirmed the diagnostic utility of CTCA in the identification of coronary heart disease. However, this systemic review highlighted several areas that need further research and in particular highlighted the need to assess the usefulness of multidetector CTCA in patients with suspected coronary heart disease. Shortly afterwards, the National Institute for Health and Care Excellence (NICE) outlined recommendations for the assessment and investigation of recent onset chest pain or discomfort of suspected cardiac origin. These guidelines specifically called for research into the clinical and cost-effectiveness of CTCA in the diagnosis of angina pectoris due to coronary heart disease. One study has assessed the effect of CTCA on the management of low-risk patients presenting to the emergency department with acute chest pain. The data suggested improved decision making but an increase in further downstream testing and health-care costs without any effect on cardiovascular outcomes. By contrast, the benefit of implementing CTCA for patients presenting to the cardiology clinic with stable chest pain has not been systematically assessed.

Added value of this study
We provide new information about the effect of CTCA on the diagnosis, management, and outcome of patients referred to the cardiology clinic with suspected angina due to coronary heart disease.

Implications of all the available evidence
The addition of CTCA to standard clinical care markedly clarifies the diagnosis of angina due to coronary heart disease. This method reduces the need for further stress testing, increases the use of invasive coronary angiography, and results in more focused treatment regimes that are associated with an apparent reduction in fatal and non-fatal myocardial infarction. Further longer term follow-up and health economic assessment are needed to establish the clinical and cost-effectiveness of CTCA before adoption into routine clinical practice.

effect on cardiovascular outcomes. By contrast, the benefit of implementation of CTCA for patients presenting to the cardiology clinic with stable chest pain has not been systematically assessed.

Methods

Study design
In this open-label, parallel-group, randomised controlled trial, we recruited patients from 12 cardiology chest pain clinics across Scotland. The study design is described in detail elsewhere. The study was done with the approval of the research ethics committee.

Participants
Patients aged 18–75 years and referred by a primary-care physician to a dedicated cardiology chest pain clinic with stable suspected angina due to coronary heart disease were eligible for inclusion. Patients with acute chest pain are not seen in such clinics but are referred directly to the emergency department. Exclusion criteria were inability to undergo CT scanning, renal failure (serum creatinine >250 µmol/L or estimated glomerular filtration rate <30 mL/min), previous recruitment to the trial, major allergy to iodinated contrast media, inability to give informed consent, known pregnancy, and acute coronary syndrome within 3 months.

All patients underwent routine clinic assessment including, if deemed appropriate, symptom-limited exercise electrocardiography with the standard Bruce protocol. Symptoms (typical, atypical, or non-anginal chest pain according to the National Institute for Health and Care Excellence [NICE] definition), diagnosis, investigations, and treatment strategy were documented at the end of the clinic attendance, before randomisation. This included categorising (no, unlikely, probable, or yes) the likelihood of the diagnosis of coronary heart disease and angina due to coronary heart disease and documentation of the need for additional stress imaging, such as stress echocardiography, and radionuclide or magnetic resonance myocardial perfusion imaging, and invasive coronary angiography. Patients gave written informed consent.

Randomisation and masking
After recruitment, patients were randomly assigned (1:1) to standard care plus coronary calcium score and CTCA, or to standard care alone, by use of a web-based randomisation service to ensure allocation concealment. Randomisation used minimisation to ensure balance for age, sex, BMI, diabetes, history of coronary heart disease, atrial fibrillation, and baseline diagnosis of angina due to coronary heart disease.

Procedures
Cardiovascular risk was calculated with the ASSIGN score, a validated Scottish cardiovascular risk score that also incorporates social deprivation and family history of
cardiovascular disease. CT scans were done with 64 detector row scanner (Brilliance 64, Philips Medical Systems, Netherlands, and Biograph mCT, Siemens, Germany) and 320 detector row scanner (Aquilion ONE, Toshiba Medical Systems, Japan) at three imaging sites. All CT coronary angiograms were assessed by at least two accredited assessors (a cardiologist and a radiologist) and the first 40 scans from each imaging site underwent independent central validation to ensure consistency of approach. Obstructive coronary artery disease was defined as a luminal stenosis more than 70% in one or more major epicardial vessel or more than 50% in the left main stem. Luminal cross-sectional area stenoses were classified as normal (<10%), mild non-obstructive (10–49%), moderate non-obstructive (50–70%), or obstructive (>70%). Assessors reported the presence of coronary heart disease with excellent intra-observer agreement of 95% and inter-observer agreement of 91%.

Outcomes
The primary endpoint of the study was the proportion of patients diagnosed with angina pectoris secondary to coronary heart disease at 6 weeks. Long-term outcomes were death, myocardial infarction, coronary revascularisation procedures, admittance to hospital for chest pain episodes, cerebrovascular disease, and peripheral vascular disease, and were identified with data from the Information and Statistics Division of the National Health Service (NHS) Scotland and, when appropriate, confirmed by review of patient health records. Categorisation for analysis was done masked to randomised allocation.

At 6 weeks, the attendant clinician was asked to review their patients' diagnosis and management plan in view of all available information including the CTCA report (standard care plus CTCA) or the ASSIGN score (standard care alone). The attending clinician documented all alterations in the diagnosis, investigations (eg, further stress testing or invasive coronary angiography), or treatments (eg, preventive and antianginal treatments). Like at baseline, patients' anginal symptoms were assessed by a self-administered Seattle Angina Questionnaire, with telephone follow-up for non-responders after two mailings two weeks apart.

For safety outcomes, radiation doses were recorded as the dose-length product and the effective radiation dose calculated (0.014 mSv/mGy·cm conversion factor). We noted adverse reactions to the scanning procedure—eg, contrast reaction, renal impairment or vasovagal response. We recorded the presence of incidental findings and whether they could provide an alternative diagnosis for the presenting chest pain.

Statistical analysis
For 80% power at a two-sided p value of 0.05, we aimed to recruit 2069 patients per group to detect an absolute change of 4% in the diagnosis of angina.

The diagnoses of coronary heart disease, and angina due to coronary heart disease (primary endpoint) were assessed for certainty (yes/no vs unlikely/probable in the primary analysis) and frequency (yes/probable vs
unlikely/no). These categories were analysed with logistic regression and adjusted for centre and minimisation variables excluding the baseline diagnosis. Odds ratios were converted into relative risks for presentation. Secondary binary outcomes were analysed in a similar way. All analyses were intention to treat, and Kaplan-Meier plots were constructed. Data were presented as mean±standard deviation, median (IQR), and relative risk or hazard ratio (95% CI) as appropriate. Statistical significance was taken as a two-sided p value of less than 0.05.

Role of the funding source
The funder had no role in the trial conduct including data collection, analysis, interpretation, writing of the manuscript, and the decision to submit. The data were analysed by Edinburgh Clinical Trials Unit. The Trial Steering Committee and the Chief Investigator were responsible for the decision to submit the manuscript. Toshiba, Siemens, and Philips had no role in the trial conduct.

Results
Between Nov 18, 2010, and Sept 24, 2014, we randomly assigned 4146 (42%) of 9849 patients who had been referred for assessment of suspected angina due to coronary heart disease at 12 cardiology centres across Scotland (figure I). Table 1 shows baseline characteristics. Few participants had a history of cardiovascular disease, but there was a high prevalence of cardiovascular risk factors and preventive treatments associated with a high 10-year risk of coronary heart disease events. At the initial clinic assessment, most participants had anginal chest pain with limiting symptoms and most underwent exercise electrocardiography stress testing (table 1). At baseline, the attending clinician established that half the participants had coronary heart disease and a third had angina due to coronary heart disease.

After clinic consultation, 2073 participants were randomly assigned to CTCA, of whom 295 defaulted or did not complete the scan (appendix). Participants who defaulted were less likely to have typical anginal chest pain (58 [23%] vs 686 [39%]; p<0.0001) or have a diagnosis of angina due to coronary heart disease (50 [20%] vs 692 [38%]; p<0.0001).

Of those 1778 participants who underwent CTCA, 31 (2%) had an adverse event related to the CTCA (13 contrast reactions, seven contrast extravasations, four vasovagal reactions, four headaches, and three other reactions). All adverse events were mild and self-limiting with no cases of anaphylaxis or renal failure. The median radiation dose was 4.1 (IQR 3.0–5.6) mSv, (dose-length product of anaphylaxis or renal failure. The median radiation dose was 4·1 (IQR 3·0–5·6) mSv, (dose-length product 291 [216–397] mGy · cm); more than a third (37%) of the dose was attributable to the measurement of the coronary artery calcium score. CTCA were mainly done with the 320 detector row scanner (n=1343) and overall diagnostic quality was achieved in 95%. Most participants (63%) had evidence of coronary heart disease with a quarter having obstructive disease (table 2). In the opinion of the clinicians reporting the CTCA, this finding markedly
increased the certainty (relative risk [RR] 3·76, 95% CI 3·61–3·89; p<0·0001) but reduced the frequency (0·78, 0·70–0·86; p<0·0001), of the diagnosis of angina due to coronary heart disease. We noted several incidental findings that were clinically important or identified non-coronary causes of chest pain (table 2).

The attending clinician reported that, compared with standard care, CTCA increased the certainty (RR 2·56, 95% CI 2·33–2·79; p<0·0001) and frequency (1·09, 1·02–1·17; p=0·0172) of the diagnosis of coronary heart disease at 6 weeks (table 3). For the primary endpoint, this translated into an increased certainty (1·79, 1·62–1·96; p<0·0001) but no effect on frequency (0·93, 0·85–1·02; p=0·1289) of the diagnosis of angina due to coronary heart disease. Overall, the 6-week diagnosis of coronary heart disease changed in 27% of participants assigned CTCA compared with 1% assigned to standard care and the 6-week diagnosis of angina due to coronary heart disease in 23% of participants assigned CTCA compared with 1% assigned to standard care (p<0·001 for both).

Changes in the diagnosis were associated with changes in planned investigations (15% vs 1% respectively; p=0·001; table 4). Specifically, the use of CTCA was associated with the cancellation of 121 functional stress tests and 29 invasive coronary angiograms. Conversely, CTCA was associated with 94 further invasive coronary angiograms (table 4). These changes were mainly the result of the exclusion or identification of obstructive coronary heart disease (appendix).

The changes in diagnoses and investigations were associated with changes in the subsequent recommendations for preventive (18% vs 4% respectively;
p<0·0001) and antianginal (9% vs 1% respectively; p<0·0001) treatments (table 4). Although the use of antianginal treatment was reduced, CTCA was not associated with an increase in the proportion of coronary revascularisation (11·2 vs 9·7%; p=0·0611; table 5, figure 2, and appendix).

Results of the Seattle Angina Questionnaire showed that treatment satisfaction was excellent at the baseline clinic attendance (92/100). Compared with baseline, angina stability and frequency markedly improved at 6 weeks in patients undergoing CTCA (n=640, 44±28 to 62±24 at baseline; p<0·0001; and n=655, 68±22 to 79±23; p<0·0001, respectively) and in those assigned standard care (n=651, 44±28 to 61±24; p<0·0001 at baseline; and n=653, 68±22 to 80±23; p<0·0001, respectively). We noted no differences in the improvements in angina stability and frequency between the randomised groups (between group difference, 1·02±0·84 [p=0·2234] and −0·87±0·70 [p=0·2147]). Furthermore, we recorded no differences in subsequent numbers of admittance to hospital for chest pain (table 5 and appendix).

Patients were followed up for a median of 1·7 (range 0·1–4·1) years. CTCA was associated with 38% reduction in coronary heart disease death and non-fatal myocardial infarction (figure 2) although, for the prespecified analysis, this fell just short of statistical significance (adjusted HR 0·62, 95% CI 0·38–1·01; p=0·0527).

**Discussion**

In this large multicentre randomised clinical trial, the addition of CTCA to standard clinical care clarified the diagnosis of angina due to coronary heart disease. This reduced the need for further stress testing, increased the use of invasive coronary angiography, and changed treatment regimes that might be associated with a reduction in fatal and non-fatal myocardial infarction.

This trial has five major strengths. First, we included a broad and large population of patients who were representative of those referred to the cardiology clinic for assessment of suspected angina due to coronary heart disease. Second, we specifically included patients who had been excluded from previous studies of CTCA such as those with obesity, high calcium scores, and atrial fibrillation. Despite their inclusion, we obtained diagnostic information in 99% of patients. Third, we allowed unrestricted use of further stress imaging in keeping with clinician choice and routine practice because we wanted to explore the effect of the addition of CTCA as opposed to doing a head-to-head comparison with other diagnostic approaches. Fourth, we focused on the effect of CTCA on patient-centred and clinician-centred outcomes rather than comparing diagnostic accuracy between imaging modalities or anatomic versus functional testing. Finally, we assessed the effect of this intervention on both short-term and long-term outcomes to define the impact of this additional imaging intervention to routine clinical practice.
In our study, 36% of patients were diagnosed with angina due to coronary heart disease in the clinic. We noted that although the certainty and frequency of the diagnosis of coronary heart disease increased, the overall diagnostic rate of angina due to coronary heart disease did not change or seemed to fall with the introduction of CTCA. This finding suggests clinicians tend to overdiagnose angina due to coronary heart disease, probably for fear of undertreatment. However, CTCA did diagnose many patients with angina due to coronary heart disease who had been misclassified in the clinic. Overall reclassification happened in one in four patients and this is clearly important for the subsequent investigation and treatment of these patients.

In view of its strong negative predictive value, the cancellation of invasive coronary angiography with the use of CTCA would be anticipated. However, CTCA caused a modest net increase in use of invasive coronary angiography. These extra invasive coronary angiograms showed obstructive coronary heart disease in most patients, including those with severe triple vessel disease. Indeed CTCA was associated with an apparent increase in coronary revascularisation, although this fell just short of statistical significance.

CTCA undoubtedly increased the identification of both obstructive and non-obstructive coronary atherosclerosis. This finding led to changes in preventive treatments despite a high prevalence of such treatments at baseline. In keeping with the changes in diagnoses, overall preventive treatment was increased and antianginal treatment was decreased. One in four patients had changes to their treatment.

Treatment satisfaction at the clinic was high and symptoms improved markedly by 6 weeks, but we recorded no short-term differences in the frequency and severity of symptoms between the allocated groups. In view of the marked improvement in symptoms with standard care and the reduction of antianginal treatment with CTCA, it is perhaps not surprising that there was no effect on 6-week symptom severity. It will be interesting to see whether differences can be seen with longer follow-up at 6 months when the potential effect of coronary revascularisation will have had chance to take effect. However, CTCA did not seem to prevent subsequent admittance of patients to hospital with chest pain, suggesting admission with chest pain is not dependent on whether there is uncertainty regarding the presence of coronary heart disease.

We had anticipated that a significant early change in overall clinical outcome would be unlikely because most patients had normal coronary arteries or mild coronary heart disease. Indeed, the overall absolute event rate during a median of 1.7 years of follow-up was low at 2%. However, we recorded a 38% reduction in myocardial

![Figure 2: Kaplan-Meier curves for CHD death and myocardial infarction (A), CHD death, myocardial infarction, and stroke (B), and coronary revascularisation (C) in patients assigned to CTCA (blue) and standard care (red).](http://www.thelancet.com)
In conclusion, in patients with suspected angina due to coronary heart disease, CTCA clarifies the diagnosis and leads to major alterations in investigations and treatments. There is a suggestion that this finding is associated with apparent improvements in fatal and non-fatal coronary events, but this needs to be confirmed by further long-term follow-up.

Contributors
The SCOT-HEART investigators contributed to the conception or design of the work, or the acquisition, analysis, or interpretation of data for the work; were involved in drafting the manuscript and revising it, and have given final approval of the version to be published; and are accountable for the work.

Declaration of interests
DN, EvB, GR, and GMcK have received honoraria and consultancy from Toshiba Medical Systems. GR has received honoraria from companies (Bracco, Bayer-Schering, GE Healthcare and Guerbet) producing contrast media.

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