

# SASCI-Mayo Clinic Fellows webinar: Managing bleeding complications in acute coronary syndrome

H Weich<sup>1</sup>, A Mazaza<sup>1</sup>, SS Gharacholou<sup>2</sup>, GW Barsness<sup>3</sup>,  
S Khan<sup>4</sup> and D Holmes<sup>5</sup>

<sup>1</sup> Division of Cardiology, Department of Medicine, Tygerberg Hospital, Stellenbosch University, South Africa

<sup>2</sup> Department of Cardiovascular Medicine, Section of Interventional Cardiology, Mayo Clinic Florida, United States of America

<sup>3</sup> Divisions of Interventional and Critical Care Cardiology, Department of Cardiovascular Medicine, Mayo Clinic Rochester, United States of America

<sup>4</sup> Department of Cardiology, School of Clinical Medicine, Inkosi Albert Luthuli Central Hospital, University of KwaZulu-Natal, South Africa

<sup>5</sup> Department of Cardiovascular Medicine, Mayo Graduate School of Medicine, Rochester, United States of America

## Address for correspondence:

Prof. Hellmuth Weich  
Division of Cardiology  
Tygerberg Hospital  
Parow  
7505  
Cape Town  
South Africa

## Email:

hweich@sun.ac.za

**Case presenter:** Dr Arlene Mazaza [Tygerberg Hospital] - AM

**Mayo Clinic Faculty:** Dr Michael Gharacholou - MG

Dr Gregory Barsness - GB

**Local SASCI Faculty:** Prof. Hellmuth Weich - HW

Prof. Sajidah Khan - SK

**Discussants are three fellows from different Universities in South Africa.**

**AM:** Case presentation

H Weich  <https://orcid.org/0000-0002-4283-0198>

A Mazaza  <https://orcid.org/0000-0002-9173-3503>

SM Gharacholou  <https://orcid.org/0000-0002-6903-1708>

GW Barsness  <https://orcid.org/0000-0002-6353-6780>

S Khan  <https://orcid.org/0000-0003-2206-8658>

D Holmes  <https://orcid.org/0000-0002-0037-0373>

DOI: <https://doi.org/10.24170/22-03-7641>

Creative Commons License - CC BY-NC-ND 4.0

Mr E.W. is a 67-year-old gentleman who, aside from a history of tobacco use, is otherwise healthy. He presented to his local clinic with a two-hour history of ischaemic chest pain and was diagnosed with an acute inferior ST-segment elevation myocardial infarction (STEMI) – Figure 1.

## ABSTRACT

Education remains a core purpose of the South African Society of Cardiovascular Intervention (SASCI), with an emphasis on engaging cardiology fellows in training through regular, structured educational initiatives. In support of this mission, SASCI has partnered with two of our past "Visiting Professors", David Holmes and Gregory Barsness of the Mayo Clinic in Rochester Minnesota, to host quarterly, case-based Fellows Webinars. The webinars are designed to foster critical thinking and clinical decision-making through interactive, case-based discussions and have thus far been very well received, with an average of 70 participants across Southern Africa and beyond.

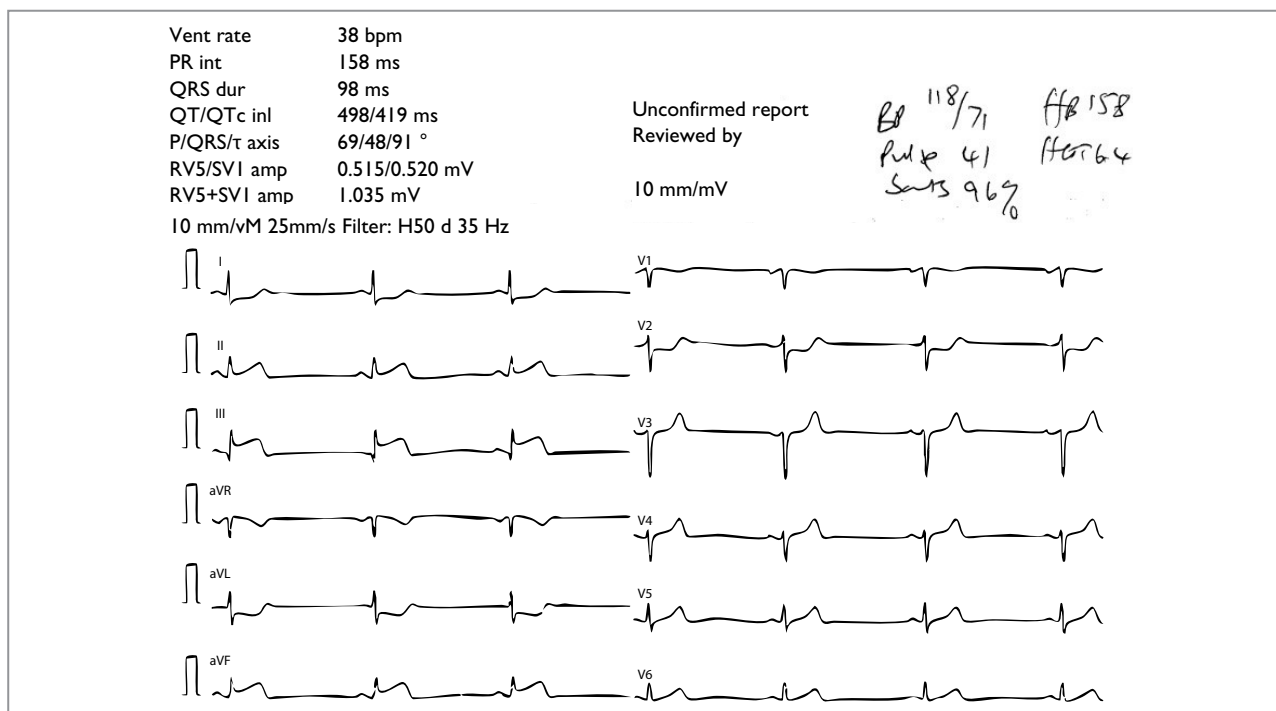
Specific topics are selected by the SASCI faculty in collaboration with the Mayo cardiologists. Each webinar session has a structured agenda, starting with a clinical case presentation by a cardiology fellow. This is followed by a concise, focused lecture by a Mayo Clinic expert, to provide context and evidence-based guidance. A robust discussion then follows, moderated jointly by the SASCI and Mayo faculty, where active participation by fellows is encouraged. The session concludes with the case presenter sharing a brief follow-up, detailing patient outcomes and the rationale behind management decisions.

All cases are anonymised to protect patient confidentiality. Each webinar is recorded and made available online at <https://form.jotform.com/251685088627570>. Access is restricted to verified healthcare professionals.

In line with our commitment to ongoing academic contribution, each webinar is intended to culminate in a peer-reviewed manuscript for publication in the *South African Heart Journal*. As part of the webinar series, the current manuscript focuses on uncommon bleeding complications in patients with acute coronary syndromes, a clinically relevant yet often under-appreciated challenge in contemporary interventional cardiology.

SA Heart® 2025;22:191-195

On admission, his pulse rate was recorded as 41 beats per minute and his blood pressure at 118/71 mmHg. He was given stat doses of oral Aspirin 300 mg, Clopidogrel 300 mg and subcutaneous Enoxaparin of 80 mg. After excluding any major contraindications, systemic thrombolysis with 100 mg Alteplase was administered. Laboratory investigations showed no major



**FIGURE 1. A 12-lead ECG showing an acute inferior STEMI.**

abnormalities. There was a good clinical response with resolution of his chest pain and ST-segment elevation. As part of the local clinical practice, he was referred to Tygerberg Hospital on the Friday evening to await angiography the following Monday.

Twelve hours after arrival, he developed a right hemiplegia with expressive aphasia. An urgent CT brain scan demonstrated a left occipito-parietal haematoma. Instructions for immediate cessation of all antiplatelet and anticoagulant therapy were issued and an urgent neurosurgical opinion was sought. The initial plan was for conservative management but a follow-up CT brain scan done after 24 hours showed expansion of the haematoma volume from 40 ml to 68 ml – Figure 2.

He subsequently underwent emergency evacuation of the haematoma with platelet and cryoprecipitate transfusions. He was referred for intense inpatient rehabilitation, and he had no recurrence of chest pain within the first week of his admission. In summary, we had a patient who presented with a high-risk acute coronary syndrome, without any clear risk factors for life-threatening bleeding complications. He had clinically successful thrombolysis but unfortunately this was complicated by an intracranial haemorrhage. The next options are of major importance and include the questions

- At this point, would he still need invasive coronary angiography, and if so, when?
- Or should we let sleeping dogs lie because of any bleeding risk that comes with coronary intervention?
- Do we need to know his LV function?



**FIGURE 2. A CT brain image showing the large intracerebral haematoma.**

**Discussant 1:** The patient had a BARC 3C bleed which is significant and will impact post intervention antiplatelet therapy. I would let the neurosurgical management take preference and let them decide when coronary intervention is safe.

**Discussant 2:** The patient has had a life-threatening cerebral bleed, and the infarct is 2 days old with established Q-waves on the ECG so I think there is little need for angiography at this stage.

**HW:** What about just doing a diagnostic angiogram with no intervention? Could that not help your decision-making?

**Discussant 2:** I don't see much benefit in that. One can consider non-invasive ischaemia testing but unless he becomes haemodynamically compromised or has ongoing angina, I would prefer to let him recover from his intracranial bleed first.

**GB:** Excellent case. Although strokes and bleeds are rare (< 1%), they are uniformly fatal so it's rare that we get a chance to discuss a case like this. I agree with your management plan and ultimately, we are going to want to see his coronary anatomy, but not at this stage. He has preserved left ventricular function, so there appears to be viability in the inferior wall. So, what if he has a sudden re-occlusion? Or develops acute ischaemic mitral regurgitation or haemodynamic compromise? How would you manage that?

**Discussant 1:** I think very short-term dual antiplatelet therapy may be an option but at high risk but I'm not sure what I would do.

**Discussant 3:** If we really have a life-threatening coronary event, one can have a look and try only ballooning to buy time but I would prefer not to stent.

**Discussant 2:** I think lytic is out of the question, but I would discuss risks with the patient to make an informed decision.

**GB:** Just so I'm clear, he just had an evacuation procedure to clear blood and not a vascular intervention, so the risk of bleeding is still there. Even if you just balloon a coronary, you will need to give anticoagulants for the intervention which would place him at risk of additional bleeding. It was a trick question really because I don't think there is a good answer. You'll only know the right answer retrospectively.

**MG:** Those are all good points and I agree that the time sensitivity of angiography has passed and I think the patient has reperfused and the only indication to go to the lab would be clinical. My first question to the surgeons would be when I can start low dose aspirin. I would wait on the idea of interrogating the coronaries.

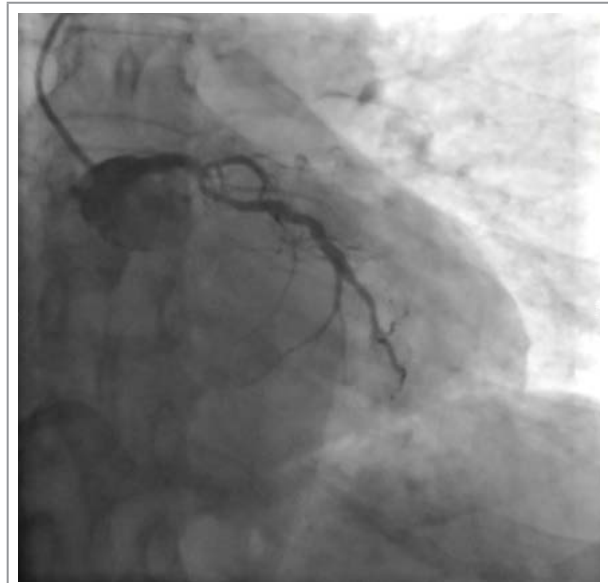
**HW:** I would like to sketch the opposite scenario to what Greg did: if the patient remains stable, is it necessary, let's say in two weeks, to do an angiogram at all.

**Discussant 1:** Although CT coronary angiography is rarely indicated in infarcts, it may be useful to non-invasively exclude left main and significant proximal disease. This may be useful to plan further management without the risk of blood thinners.

**SK:** Valid point. May I ask what blood thinning therapy he was placed on?

**AM:** Nothing. What would your plan be in terms of recommencing antiplatelet therapy?

**Discussant 2:** I would be guided by the neurosurgeons, and they will likely prefer two weeks. I would then only start low dose aspirin.



**FIGURE 3. Angiographic view of the left coronary system showing an occluded LAD and diffusely diseased left circumflex artery.**

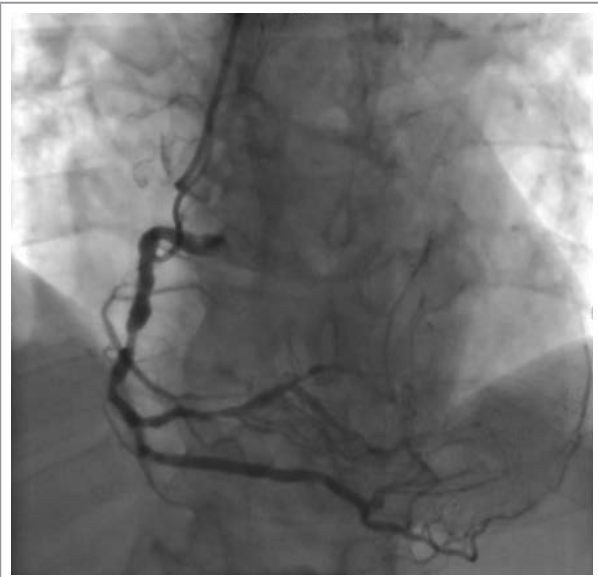
**MG:** To come back to the CT angiogram, although it is very unlikely to be normal, I think this is a great option to exclude proximal disease, although I think in this country, somebody who has had successful lysis, resolution of ST-elevation and no symptoms may be best treated by a functional test for ischaemia. It doesn't have to be angiography.

**HW:** That's an interesting thought, but ischaemia testing will not identify an angiographic unstable lesion in a proximal vessel if it is not flow limiting so one might combine the 2 to decide. Arlene, shall we proceed to your management of the case?

**AM:** The neurosurgeons were happy to say that it would probably be safe to reinstate antiplatelet therapy after 2 weeks. His neurological fallout showed mild improvement, and we decided to proceed with coronary angiography, with a plan to define the coronary anatomy.

In the first injection, we now see that the LAD is occluded – Figure 3. The circumflex is also diffusely diseased and one of the obtuse marginals is occluded. The right coronary artery is a very large system, and it is collateralising the left system – Figure 4. The culprit from the acute event is probably in the mid-right coronary artery and the patient has normal left ventricular systolic function.

We now had a patient 2 weeks post-infarct with evidence of triple vessel coronary artery disease, but with a culprit lesion that is actually a very important vessel as it is collateralising the LAD. Now that we've seen the coronary anatomy, we'll get some more opinions from the panel about what to do.



**FIGURE 4. The right coronary artery is a large vessel collateralising the left. There is a midvessel lesion which we deemed to be the culprit.**

**Discussant 1:** It's a very tricky case because even if you must do a bypass on him, unless you're going to do it off-pump, he's going to need heparinisation. An off-pump bypass may be an option, but it would be quite difficult, I suppose. I think he would get mortality benefit from revascularisation, but the risk is also very high. So, I think you'd have to put it to the patient, speak to his family. Also depending on what his recovery was like following his neurosurgical intervention, what his level of functioning is, was he mobile, was he bed bound?

**Discussant 2:** In my centre, this patient would be offered bypass surgery because he has triple vessel disease. Waiting time for bypass surgery in my hospital is about 3 to 6 months. This would give his brain time to heal, and you can control his risk factors. The LAD and RCA are both good targets.

**HW:** Two questions:

- The lesion in the RCA looks unstable, and I'm not sure we can trust it to settle over the next 3 months and if it does re-occlude, it may not be well tolerated.
- What if you work in the private sector and the surgeons say they can do the bypass tomorrow?

**Discussant 2:** The current guidelines also recommend a hybrid approach so one could stent the RCA now and do a bypass to the LAD at a later stage. I would target the RCA because this is the simpler lesion to treat and can explain his presentation.

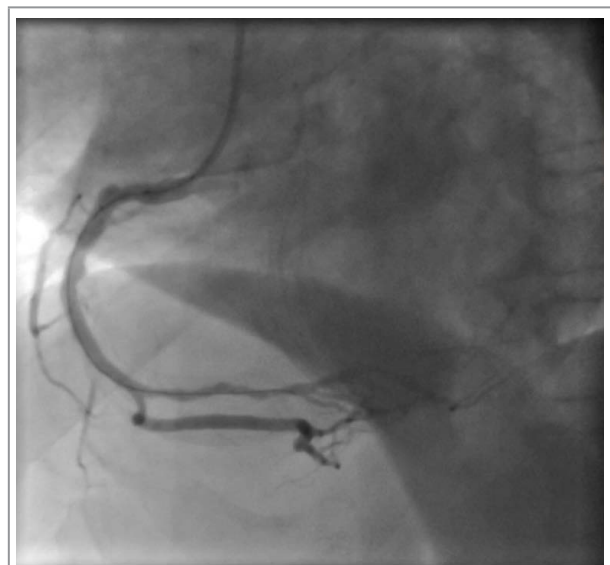
**MG:** I guess the question fundamentally is, do you think the RCA is stable? If you think the RCA is stable, you could nurse this patient along medically until they're more neurologically out of the woods. I think a recent head bleed is a no-go for many

surgeons. So, the idea that this patient's going to go for CABG anytime soon is probably not correct. Maybe, ultimately, CABG would be a good idea, but you've got to get that patient through the next several months. So really, is there anything here that could help us decide whether to just do the mid-right in this positively remodelled, ectatic vessel? Or do we just walk away and treat medically? And I think it's a tough decision. I think there's equipoise.

**GB:** I think it could go either way. I think some sort of hybrid procedure is probably worthwhile to consider in the very distant future. But I like the idea of stabilising the mid-right vessel. We have data that, in general, this single vessel intervention is likely going to be safe and effective at reducing reinfarction and improving prognosis. So, I think limited stenting of the mid-right certainly seems reasonable to me. I would avoid balloon angioplasty alone. I wouldn't over-treat, but I think managing what appears to be the offending lesion or lesions in the mid-right would be reasonable. Imaging might play a role here, although, again, with the caution that you want to mitigate risk by trying to avoid doing too much. Arlene, we are dying to hear what you did.

**AM:** There was a question of whether the LAD could be at play, so we opted to first wire interrogate the LAD and we were quite happy it was a CTO. We then proceeded to treat the mid-right and achieved a good result with no complications. We then offered the patient 1 month of dual antiplatelet therapy. See Figure 5.

He continued inpatient rehabilitation, and we aimed to address future revascularisation options once his neurological state stabilises. Depending on his symptoms and degree of neurological recovery, it would either be a LIMA to the LAD or a formal CTO procedure percutaneously.



**FIGURE 5. Right coronary artery after stenting the mid vessel lesion.**

**GB:** That was great, great, great management. Fantastic. Just amazing that the patient survived all of that. I mean, that's, that's really, really, really remarkable.

**HW:** Who thinks he's going have symptoms? Are we going to end up treating this LAD?

**GB:** So I think in answer to your question, it depends on his neurologic state. If he has improved neurologic status, then he may well have some symptoms. I think, especially with the LAD residual territory, if his prognosis is reasonable, there's going to be some sort of benefit to complete revascularisation, survival benefit even. But it depends on his wishes and his underlying state. But yeah, fascinating.

**MG:** Great case. I agree that time will tell for this case, I think, which way it's going to go. He may never really do very much to have angina, to have a lot of ischaemia. He may do well with this and medical therapy. If the patient becomes more active, he's a young person, then dealing with the LAD may become an issue. Although with some of these images, it's hard to tell how diffuse the disease is in the LAD. You know, he's probably going to come back to the cath lab at some point in the future

**SK:** Well, that was a fantastic discussion. Thanks everybody for participating and I look forward to seeing you soon for the next one.