RCPE Medicine of the Older Person Symposium

Thursday 19 March 2020

Heart Failure – Dr Diane Barker

Q. Who needs an echocardiogram?

A. Echocardiogram helps determine the mechanism and aetiology of heart failure. The treatments for HFrEF and HFpEF are quite different. In addition, valve disease, right ventricular impairment, pulmonary hypertension or amyloid heart disease may require further modifications to treatment. Echocardiography is recommended for all heart failure patients, unless they are for symptomatic and supportive care only due to other advanced comorbidities.

To work out who needs an echocardiogram, it may help to consider how the results may influence management:

1. To determine whether the mechanism of the clinical diagnosis of the heart failure syndrome is due to preserved or reduced left ventricular ejection fraction

2. To determine whether there is a structural (e.g. cardiomyopathy) or valvular cause or consequence of the clinical heart failure syndrome and to quantify its severity to determine prognostic features

3. To monitor structural and valvular problems in patients in whom a defined future clinical and or echocardiographic end-point would act (in isolation or as a synthesis of clinical features) as a trigger for either palliation or intervention

4. To screen family members for structural heart disease in isolation or as part of a recurrent screening strategy based on the genetics of the index inherited condition

5. To diagnose the cause of cardiac murmurs in patients in whom the diagnosis would make a difference to their management

6. As part of the diagnostic work up for endocarditis

7. To demonstrate, monitor and assess the haemodynamic effects of pericardial disease

8. To demonstrate and monitor the presence and impact of cardiac masses

9. To demonstrate the presence of right heart strain in primary pulmonary pathology when the presence of right heart dysfunction or the likelihood of high pulmonary pressures will make a difference in the further investigation or treatment of the patient e.g. thrombolysis for a significant PE, investigation of pulmonary arterial hypertension.

10. For detailed haemodynamics in patients in whom these assessment would make a difference to their management – constrictive pericardial disease, hypertrophic cardiomyopathy, amyloidosis

If patients fit into these 10 broad categories, then they may be benefit from echocardiography.

Q. How long after the switch from IV to oral can you send a patient home if they are well?

A. The NICE guidelines recommend the patient's condition is stable for 48 hours after switching from IV to oral diuretics before home. However if you have a suitable rapid review clinic where patients can be seen shortly after discharge then this may be a suitable pragmatic alternative to an additional 48 hrs of hospitalisation in otherwise stable patients. https://www.nice.org.uk/guidance/cg187

Q. Other than diastolic failure, are there other situations when prognosis-improving drugs might not be used?

A. It is important to recognise that diastolic failure and systolic heart failure are not diagnoses – they are merely arbitrary ejection fraction thresholds for the efficacy of some medications e.g. EF of 40% and below. The 'prognostic' medications have been studied in chronic heart failure with reduced EF.

Beta blockers should only be started or uptitrated once patients are stable and euvolaemic, even when indicated due to reduced ejection fraction.

Contraindications, cautions, interactions, allergy (including angioedema) and co-morbidities should be considered for each patient and each 'prognostic'medication i.e. treatment should be individualised. Some patients may be reaching end of life and so comfort care rather than prognosis-improving drugs may be the priority.

Q. Do you need an IV bolus before using a continuous subcutaneous infusion (CSCI) of furosemide? (NB: This is a very useful tool in Hospital at Home (H@H) and in similar community teams looking after subacute deterioration in congestive cardiac failure (CCF)).

A. No, an IV bolus is not necessary. Subcutaneous infusions can be very useful to avoid unwanted hospitalisations. Generally, subcutaneous infusions of furosemide would be used after IV diuretics as step-down treatment, or after increased oral diuretics, as step-up treatment. So the threshold concentration to invoke natriuresis will already have been reached.

Q. Should we treat everyone with raised proBNP?

A. NT-pro BNP is a marker of heart strain due to stretch. It is not diagnostic as to the aetiology of this 'heart strain'. There are many causes of an elevated NT-pro BNP including increased age, atrial fibrillation, infection, renal impairment and pulmonary diseases.

It is a diagnostic and prognostic marker whether it is raised from a primary cardiac pathology e.g. AF, MI, or due to secondary action on the heart e.g. PE, infection etc.

The stronger the clinical presentation of heart failure and the higher the N-pro BNP, the more likely the patient is to have heart failure. [European Journal of Heart Failure (2019) 21, 715–731]

Q. Do any trials have outcomes other than cardiovascular mortality Rather than all cause (we think they may mean Do any trials use the outcome of cardiovascular mortality rather than all-cause mortality?)

A. Both are relevant and many papers contain both. Examples include the recent PARADIGM and DAPA HF studies

In the Candesartan in Heart Failure Reduction in Mortality and Morbidity (CHARM) trials, all-cause mortality was used as the primary endpoint. Out of 1831 deaths, 371 were non-cardiovascular and unlikely to be influenced by cardiovascular therapy. While the effect of candesartan on all-cause mortality was not statistically significant, the effect on cardiovascular mortality was significant. [CHARM-Overall programme. Lancet 2003;362:759–766.] Heart failure trials are now often designed with cause-specific composite endpoints such as cardiovascular mortality or heart failure hospitalisation. All-cause mortality is usually still reported for safety. [European Journal of Heart Failure 2016; 18:482–489]

Q. "All cause mortality can't be reduced forever" - can you comment on this in terms of how we evaluate trials?

A. In chronic conditions such as heart failure, mortality is not the only meaningful efficacy measure, since a patient may be alive but have a poor clinical status, functional capacity, or quality of life [European Journal of Heart Failure 2016; 18:482–489]. Improvement of quality of life is also important and there have been proposals of how to standardise methods to measure this for clinical trials [Eur J Heart Fail 2002; 4: 243–247].

Reducing mortality is interpreted to mean postponing death. How meaningful this is depends on the time to benefit as shown in the clinical studies, in addition to individual patient factors such as age and comorbidities.

Q. Does frailty increase the risk for heart failure?

A. Yes. Elderly patients with heart failure are at increased risk of developing frailty, and frail older adults are more likely to develop new-onset heart failure [Am Heart J 2013; 166: .doi:10.1016/j.ahj.2013.07.032.] Managing frailty may help improve quality of life and have a substantial impact on prognosis in heart failure [European Journal of Heart Failure 2019; 21: 1299–1305]

Q. Can New York Heart Association (NYHA) Functional Classification be used when other comorbidities limit activity?

A. This can make use of NYHA classification difficult. NYHA is a simple, easily applied classification system for everyday clinical practice, but has limitations and can be very subjective.

Q. What are the advantages of nebivolol over carvedilol?

A. There have not been any randomised trials comparing carvedilol with nebivolol. Nebivolol has been studied in a randomised controlled trial specifically in the older population (mean age 76 years). Nebivolol was well-tolerated and reduced mortality and morbidity compared to placebo. [SENIORS European Heart Journal (2005) 26, 215–225.] A trial comparing carvedilol and bisoprolol found more pulmonary side effects with carvedilol and more bradycardia with bisoprolol. [CIBIS-ELD European Journal of Heart Failure (2011) 13, 670–680.] In clinical practice, bisoprolol and carvedilol are more commonly prescribed for HFrEF than nebivolol.

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Diabetes - Mark Strachan

Q. Does the United Kingdom (UK) produce its own insulin or do we still import insulin?

A. All insulin is imported from US and Europe

Q. Are DPP-4 inhibitors (gliptins) effective for all patients with type 2 diabetes mellitus (T2DM)?

A. DP4 inhibitors overall have weak glucose lowering ability. Some individuals will respond better than others, but as a class they are less potent than other anti-diabetic agents

Rheumatoid arthritis - Hema Bhat

Q. What are the lab tests that should be within normal limits before prescribing conventional disease-modifying anti-rheumatic drugs (DMARDs)?

A. That would depend on the choice of DMARD. Usual blood tests would be a full blood count, LFTs and UE.

Here is the guidance on specific DMARD-
https://academic.oup.com/rheumatology/article/56/6/865/3053478#.XnybkkHC89Y.email

Q. What measures should be taken in a patient with rheumatoid arthritis with dengue haemorrhagic fever who has already taken some doses of non-steroidal anti-inflammatory drugs (NSAIDs) at home?

A. I presume, your question is to do with COVID-19. If it is so then there is no evidence to suggest at this point that NSAID use worsens COVID infection.

https://www.gov.uk/government/news/ibuprofen-use-and-covid19coronavirus

Q. How common are bilateral eye signs in new giant cell arteritis (GCA)?

A. Would be very unusual to have bilateral involvement. Unfortunately I don't have much information on the exact incidence of this.

Q. How common are fleeting eye symptoms (lasting seconds) in giant cell arteritis (GCA)?

A. Fleeting eye signs are a high risk for ophthalmic involvement in GCA and should not be taken lightly.

https://doi.org/10.1093/rheumatology/kex428

Q. Should we dose-adjust prednisolone for giant cell arteritis (GCA) according to patient weight?

A. No. Prednisolone dose of 40mg for all and higher dose of 60mg for ophthalmic involvement. The earlier BSR guidance did talk about a weight dependent dose, however there is not much literarture on it and it has been removed from the most recent guidance.

https://academic.oup.com/rheumatology/article/59/3/487/5714025

Perioperative management - Magda Sbai

Q. What is the current advice on anti-coagulation, post-DHS (dynamic hip screw) for neck of femur fracture, for patients on warfarin or direct oral anticoagulants (DOACs) pre-surgery?

A. Restart once surgeons happy haemostasis achieved, usually 24-48 hrs post surgery, however LMWH can be given 6-12 hrs post if worried about potential bleeding risk to bridge

Q. Would you discontinue disease-modifying anti-rheumatic drugs (DMARDs) in the peri-operative period? When would be the ideal time to restart?

A. DMARD therapy should not routinely be stopped in the perioperative period, however if Patient factors (age, frailty, multi-comorbidities, smoker) and surgical factors (surgical time >60 minutes, dirty procedures, Class 3 or 4 procedure) constitute significant perioperative infection risk stop DMARD 2 weeks prior to surgery and restart once wound healing satisfactory

In the Community: The Silver City Project - Robert Caslake

Q. Do you have access to urgent investigations?

A. I have access to the investigations you would usually expect to be requestable from a geriatrics clinic, so outpatient radiology including MRI and CT, echocardiograms, etc. I would usually try to see go out to see people before requesting any complex tests, rather than just ordering them on the basis of a discussion at one of the meetings.

I work closely with our Community Geriatrics Nurses (CGNs), who do ECGs and have 72 hour ECG monitors that they can put, and who do lying and standing BPs, Dix-Hallpike maneouvres for investigation of falls and dizziness.

Blood tests are harder than at clinic- I usually have to ask the GP practice to arrange them but will sometimes ask the CGNs to do them if its anything especially urgent or complex. We haven't figured out a good way of doing short synacthen tests or measuring parathyroid hormone levels just yet, which is sometimes a problem, but we have an excellent endocrine investigation unit locally who are happy to help out if people can manage to get there.

Epilepsy - Richard Davenport

Q. Many of our patients have an 'inconclusive' EEG result. What would be your approach in these patients?

A. ignore it, EEG rarely helpful in this age group and never diagnose epilepsy on an inconclusive EEG, trust your clinical instincts instead.