UNANSWERED QUESTIONS DURING THE LIVE WEBINAR

1. **How to classify non ischemic non infiltrative RV Myopathies?**
   isolated RV dilated cardiomyopathy is rare. First you should exclude ARVC using Task Force criteria.Second you should exclude secondary causes of right heart failure. in DCM most often there is associated LV dilation and dysfunction with variable degree of RV involvement.
2. **What measures have been done to check up athletes and many sportsmen and women to get them away of cardiomyopathy ?**
   Follow guidelines on sport medicine.
3. **As the definition states that the diagnosis of dilated cardiomypathy is made in the absence of coronary disease should we stop using the term 'ischaemic cardiomyopathy'?**
   Yes
4. **Is the post partum cardiolmyopathy is the classification of DCM ?**
   Yes
5. **What is the management of post-partum cardiomyopathy?**
   There is overlap with medical management of DCM. There are also consensus ESC papers on PPCM
6. **If DCM has been diagnosed when pregnancy is allowed?**
   We give some indications in our position paper. In addition please follow ESC guidelines on heart disease in pregnancy and on heart failure.
7. **In subjects with genotype for CDM the sports physical activity can contribute to development of phenotype of CDM and to the worsening of the clinical outcome?**
   indeed there are such assoications found however the data is not strong enough to draw firm conclusions , but we do reecommend: 1) no competitive high intensity sport for subjects who are affected 2) lower intensity sports (<80% of maximal, only endurance not interval not isometric forces) for affecteds 3) no real limits for completeley unaffected altough I limit LMNA mutation carriers as affected.

8. **Does it expensive gen-testing in your hospital?**
   around € 800,- for a panel
9. **Do you suggest that all patients with suspected myocarditis undergo endomyocardial biopsy or just those with active inflammation/arrhythmias in whom the yield is likely to be high?**
   In all patients with clinically suspected myocarditis according to ESC Task Force criteria. In particular all patients with non ischemic DCM regardless of MRI findings
10. **If we don't have evidence of improve outcomes with revascularitation en ischemic cardiomyopathy why would we have to screening for artery coronary disease?**
    Not pertinent to our webinar.
11. **Should we consider dilated left ventricle if EDD>57mm or should we correct this figure with the body surface area or BMI? Thanks**
    The EDD dimension in family studies has been corrected using the Henry's equation. LV volumes are corrected by BSA (see imaging guidelines)

12. **If Endomyocardial Biopsy would it be only considered in clinically suspected (acute) myocarditis we would miss a large propotion of chronic inflammatory cardiomyopathy etiology in patients presenting with chronic DCM.**
    Correct. In fact in our Position paper on myocarditis (Eur Heart J 2013) we suggest to perform EMB in all patients with non ischemic acute or chronic DCM that by Task Force definition have clinically suspected myocarditis.
13. **what about the use of speckle tracking and the early diagnosis?**
    It may be useful, little prospective data at present are available
14. **Does alcoholic CM have genetic background?**
    So far there are not enough data. It is clear that in true alcoholic DCM the process is reversible with abstinence, therefore there is a toxic effect of alcohol.
15. **Alida what do you think about significance of anti-heart antibodies in diagnosis of inflammatory cardiomyopathy?**
    If they are present they are markers of an autoimmune process. Most of the time EMB is negative for infectious agents.
16. **Why recommend family screening in all DCM cases when the family types might be only 1/3 of the cases?**
    Because family background may be underestimated.
17. **How important or is Strain analysis is more reliable test to assess function of LV.**
    No need of strain to assess LV function, some studies suggest that it may be an early marker of subclinical dysfunctio. So far there is a relative paucity of prospective data
18. **Is it possible - secondary noncompaction in patients with severe systolic dysfunction?**
    It is a very controversial issue, mainly because diagnostic criteria for LVNC are not very reproducible. In addition, some studies suggest that LVNC pattern may change with time. So far CMR is considered the gold standard.
19. **What do you think about the use of immunoadsortion in inflammatory cardiomyopathy?**
    A multicentre trial in ongoing in Germany. The results should be available soon. In principle it may be useful in autoantibody-mediated forms of DCM, since some of the anti-heart autoantibodies have been shown to have a direct functional role.
20. **Would you ask for immuno-histochemical investigation for desmosomal proteins in endomyocardial biopsy of a DCM patient?**
    Only if there is suspicion of biventricular ARVC
21. **When myocar-biopsy should be performed?**
    At diagnosis of a non-ischemic DCM
22. **CMR role in cardiomyopathy? How much specificity of CMR in cardiomyopathy?**
    The specificity depends on the cardiomyopathy type. In relation to DCM, CMR may show a non-ischemic LGE pattern, with or without oedema. It is not enough to rule in or rule out myocarditis especially in DCM (the clinical case presented at the webinar is a typical example).

23. **When should we ask for a genetic test?**
    When you have red flags or when there are at least two first degree affected

24. **What is the difference between hypertrophic cardiomyopathy and hypokinetic non dilated cardiomyopathy?**
    In HCM there is LVH. However, some HCM patients may progress to a hypokinetic non dilated CMP at end-stage where LVH may regress. Therefore it is important to study the family to find out whether there are cases of HCM. In addition it is important to check whether in preceding years there was a diagnosis of HCM in the proband.
25. **Strain assessment would be of help in this case more sensitive than EF**
    To be seen. Little data (see previous replies)
26. **So when should we do genetic tests in this case?**
    When you have red flags or when there are at least two first degree affected
27. **What about post partum CM?**
    see previous reply on PPCM
28. **How many relatives we should screen (first second degree more?)**
    Start with first degree, if there are affected relatives go to second degree (cascade screening)
29. **Why not recommend MRI in all DCM patients to achieve the best measurements of volumes and EF as echo might have a very poor accuracy?**
    Echo may be very accurate if acoustic window is acceptable, In addition, nowadays 3-D echo is also available.
30. **In DCM when in OPT according to heart failure guidelines and in a stable condition (NYHA I-II) how long could we expect for further improvement ? What has been reported so far ?**
    It is variable from patient to patient and also in relation to aetiology. For instance autoimmune inflammatory cardiomyopathy may resolve completely, but relapses are also possible after many years. There are little data on etiology-specific DCM forms.
31. **Do we have substantial news on the overlap of the identical genetic mutations being associated to both hypertrophic or dilated cardiomyopathy?**
    Correct. There is overlap.
32. **What about peripartum cardiomyopathy ?**
    see previous reply on PPCM
33. **Would beta blocker worsen EF?**
    Beta-blockers are part of the chronic medical management of DCM, although according to CHF guidelines they should be introduced when the patient is hemodinamically
34. **What about pregnancy cardiomiopathy and follow up can be a minor form, is this for genetic influence?**
    see previous reply on PPCM
35. **If there was syncope in this patient and non-sustained tachycardia was detected - why not ICD?**
    the fall from a bike was not syncope: she simply slipped…

36. Would starting a beta-blocker in this patient not lead to bradycardia and potentially a pacing indication which may be counter-productive?
Yes indeed this also led to some questions by the referring cardiologist, with hindsight would have been better to get the genetics and then give him an ICD

37. is GLS is recommended in such pts?
For research purposes.

38. How important would you say that Global Longitudinal Strain is in this group of patients?
For research purposes.

39. What about the use of beta blocker and conduction abnormalities?
With great caution and while they are needed in overt DCM, this happens a lot so good caution is needed

40. Nuclear Radiologiy Imaging has any place diagnostically?
Do you mean Coronary TC? If so, it may have a role for patients at low risk of CAD. However, if you aim at an etiology-specific diagnosis in DCM you need EMB, therefore it.

41. What autoantibodies should we test and which methods are shown to be reliable and reproducible?
In the myocarditis position paper (Eur Heart J 2013) there is the list of all available tests and accuracy can be found in the original reference. The technique used at our institution (indirect IFL on human heart and skeletal muscle) is accurate cardiac and and disease-specific for inflammatory cardiomyopathy/DCM.

42. How disease specific are 'autoantibodies' in DCM? Are the detection tests commercially available and standardised?
See previous reply

43. What is the role of steroids as an anti-inflammatory and are NSAIDs contraindicated in fluid-overloaded heart failure?
NSAIDs are not used in myocarditis. Steroids are part of the most used immunosuppressive protocol in biopsy-proven autoimmune DCM and usually well tolerated (please go to myocarditis position paper for further info , Eur Heart J 2013)

44. What distinct auto-antibodies should we look for?
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45. What is the role of USING tissue doppler echo. in detecting early phase
For research purposes.

46. Endomyocardial biopsy for diagnostic reasons in DCM is not performed in North America! Why do we behave different in Europe?
I do not know why. In Europe we have a different NHS system (mainly public).

47. GLS in detecting early disease
For research purposes.

48. What is the yield of coronary angiography in patients with a non-ischaemic DCM appearance on CMR? Can these patients not be spared the

risk of invasive angiography?

Good question. However, if you aim at an ethiology-specific diagnosis in DCM you need EMB, therefore it seems preferable to perform coronary angiography and at the same time if the coronaries are normal, EMB and spare CMR.

49. **Please remind us of the associations between any congenital & inherited conditions - and cardiomyopathy > Any screening?**

    Congenital defects should be ruled out in DCM.

50. **Did this patient receive anticoagulation due to very low EF? How long to continue this therapy?**

    He did not receive anticoagulation (there was no endocavitary thrombus)

51. **Are the AHA and AIDA tests widely available? Do they have any additional value in case we have the possibility to do myocardial biopsy?**

    Aha and AIDa are available in the central lab at our centre and are part of the diagnostic tests within the NHS in Italy. They give additional information to EMB. They are also useful in symptom-free relatives of proven DCM, because they predict DCM development, similarly to other serum autoantibodies in extra-cardiac autoimmune disease.

52. **During the period between the ICD implantation and normalization of LVEF did the ICD discharge as a response to a ventricular arrhytmia? Thank you**

    No

53. **What will be Your behaviour if the endomyocardial biopsy did not support the diagnosis inflammatory cardiomyopathy?**

    Since no viruses were detected and antiheart autoantibodies were present, we might have considered to give immunosuppression anyway, after discussing with the patient, although with a lower level of evidence.

54. **Can ivabradine help DCM patients?**

    In selected patients, according to CHF guidelines

55. **The interobserver variabilty and sampling error are substantially hampering the validity of myocarditis diagnostics in endomyocardial biopsies. How to address these issues?**

    With current immunohistemical and molecular tools this is not an issue. This was an issue when only the histological criteria (Dallas criteria) were used.

56. **I'm a physician from Latvia.My patient of 42 with dilated cardiomyopathy has been now for 3 years on Thoratec Heart Mate II having now inflammatory process of the front abdomen wall and no prospectives of transplantation in Latvia.Could you give recommendations t?**

    I am afraid we do not have perspectives here. This is why we think it is ethical to reach an aetiological diagnosis in DCM as soon as possible and to use aetiology-specific therapy if indicated.

57. **Who paid for procedure whem we are talking about different countries?**

    I do not understand the question. In Italy DCM patients are covered by the NHS, as well as EU citizens.