Podcast Episode 1: Primary Biliary Cholangitis Highlights from EASL 2023

Brought to you by;
Dr Emma Culver, John Radcliffe Hospital, Oxford, UK
Prof. Gideon Hirschfield, Division of Gastroenterology, University of Toronto, Canada

Please note:
COR2ED podcasts are designed to be heard. If you are able, we encourage you to listen to the audio, which includes emotion and emphasis that is not so easily understood from the words on the page. Transcripts are edited for readability. Please check the corresponding audio before quoting in print.

This COR2ED programme is supported through an independent educational grant from Ipsen.

The views expressed within this podcast are the personal opinions of the authors. They do not necessarily represent the views of the author's academic institution.

Emma Culver
So welcome, and thank you for joining us to discuss the PBC highlights from EASL 2023. I'm Emma Culver, Consultant Hepatologist from John Radcliffe Hospital in Oxford, in the UK.

Gideon Hirschfield
I'm Gideon Hirschfield, I'm a Hepatologist in Toronto, and I'm really pleased to join Emma today to talk about some of the highlights from EASL 2023.

Emma Culver
So let's get started with our first selection, and that's a Swedish study on the association of PBC with cancer risks and outcomes presented by Axel Wester of the Karolinska Institute. Gideon, can you take us through the study, please?

Gideon Hirschfield
Yeah, of course. It's a frequent question in clinic to ask us, beyond PBC what will happen to me, and in particular, is there an elevated risk of me developing cancer? So what these investigators have done is use a Swedish national register of patients living with PBC. So they over approximately 17 years, they were able to identify just over 3000 patients with PBC and they have a reference population, a comparative population of around 27,000, and the follow up is on average between 5 and a half years for the PBC patients and seven years for the control cohort. Now the patients are on average 64 years old and obviously the majority of them are female.

And I think what this study finds [are] really two things, to my mind. First of all, there is an increased risk of cancer in people living with PBC, but my interpretation of the data is that it's strongly and nearly exclusively linked to hepatocellular carcinoma [HCC]. So this is not a surprise. We know that PBC, if treated only with urso [ursodeoxycholic acid], will lead to

cirrhosis in around 30% of patients. Although we hope in the future the rate of cirrhosis will go down. And we know that cirrhosis is the leading risk factor for HCC across all diseases. So this was an important observation, although I would just put it into context that the rate of HCC is likely to be falling.

Then they looked at other non HCC cancers and they did report some increase in cancers, particularly around GI and lymphoma. This small increase was not so much that I was particularly worried. It seemed to be linked to possibly colon cancer and possibly some other GI cancers and lymphoma. Of course our patients can get Sjögren's [syndrome] and this study can't work that one out. So Sjögren's is associated with lymphoma. Our patients can have celiac disease and celiac disease is associated more with GI cancers. So there was a small increase in some of these non HCC cancers.

But my take homes Emma, was that this is a big study, that it confirms that PBC is an important liver disease and development of cirrhosis is what we're trying to prevent. And one of the complications of cirrhosis is hepatocellular carcinoma, and that is increased in patients living with PBC. But I wouldn't use the study to change my practice as such, other than to say it reinforces the goals of being a hepatologist, which are the prevention of end stage liver disease.

Emma Culver
Yeah, Thanks, Gideon. I think that's a lovely summary and I think it does highlight the main points. I guess for me, I kind of went away thinking - so, you know, should I be screening all of my patients, irrespective of cirrhosis, for hepatocellular carcinoma? Because I didn't feel like there was data to say it was just those patients who got cirrhosis who got hepatocellular carcinoma. And we know that for some earlier studies, including some from Palak Trivedi and I think yourself, suggesting that actually in those patients who don't respond, so for example men who are non-responders and who are non-cirrhotic, there's an increased risk of hepatocellular carcinoma and obviously in females with cirrhosis, but also female non-responders as well without cirrhosis.

So I think it went away with me thinking about whether or not we should just be looking at those with cirrhosis in terms of cancer risk and also those who are non-cirrhotic who are not responding. What do you think? Do you think that would change the way that you would practice in terms of surveillance?

Gideon Hirschfield
So the truth is, I didn't see this as changing how I practice. Already I screen, essentially, only patients with cirrhosis and even that I recognise in PBC may not meet the threshold for other kinds of cancer surveillance. I think this is one of the challenges. I'm not a stats man, as you know, but yes they can show an incredibly high hazard ratio for HCC compared to completely healthy people. But what does that mean about absolute risk and what does that mean about a stratified population? These are predominately women who already have less metabolic risks and lower rates of HCC and should, now and in the future, have very effective treatments for their PBC.

So my take home is that I'm not going to change my practice, I'm not going to look more than I look already. I'm going to survey those patients who have overt cirrhosis. And in that group of patients, perfectly reasonable to do an ultrasound every six months. And if you wish to do an AFP [alpha fetoprotein] and I think when we were trying to find a time to meet in your busy schedule you were telling me that you were coming from one of your HCC meetings and I genuinely don't believe in your professional experience that PBC is a big contributor to your HCC meetings on a weekly basis.

Emma Culver
No, no, I think you're right entirely. We don't see so much HCC in PBC in the overall group of HCC individuals. We see viral, NAFLD [non-alcoholic fatty-liver disease] and other aetiologies being more predominant. And I think in terms of screening you have to have something that's cost effective without increased risk of complications and with good evidence of benefit down the line and this data is great, but it hasn't quite met that yet.

Emma Culver
Fantastic. Can I ask just one last question in the cancer field? So I was quite surprised or maybe not. Obviously when we think about the different kinds of cancers, obviously we know that PSC [primary sclerosing cholangitis] is associated with cholangiocarcinoma and there was some nice work on the next day that was presented looking at kind of biomarkers to detect cholangiocarcinoma in the context of PSC. Obviously PBC didn't associate at all with cholangiocarcinoma. Were you surprised about that or do you think there was a logical explanation for that?

Gideon Hirschfield
I'm not surprised. I mean, cholangiocarcinoma is rising, intrahepatic cholangiocarcinoma is associated with cirrhosis, but largely that rise has been seen in viral and fatty liver disease. I'm not surprised because actually the biliary epithelium is so different and whilst we don't understand, you know, PSC is a large bile duct disease, PBC a small bile duct disease and something about the mucosal surfaces is relevant to cancer generation. And there's been some biology looked into that, we haven't solved it by any means, but it didn't surprise me.

Yes, do my patients have overlap with fatty liver disease and metabolic syndrome? Of course they do. Will we therefore see incidental associations? Naturally, we will. You know, obesity is going to be such a big impact on all of our practice. But fundamentally, biologically, these are very distinct diseases. And remember, genetics has always told us that they're distinct diseases and genetics hasn't helped us to understand why one's small bile duct, and one's large bile duct. I imagine that's got more to do with local mucosal immune responses between the two processes. But I think that was a great discussion.

So the other abstract, which really people were waiting for. Let's just change sort of tack slightly, still on PBC, very important disease, common, rare, but this is an abstract about how do we actually use the tools in our clinic about prognosis, the prognostic significance of liver stiffness progression in primary biliary cholangitis.

This was an abstract presented by Christophe Corpechot on behalf of the Global PBC Study Group. But Emma, can you summarise what you took away from Dr. Corpechot's presentation around liver stiffness progression in PBC?

Emma Culver
Yes, thanks, Gideon. The background to this very much is that PBC, we know is a chronic liver disease with an increased risk of complications, transplantation and death and whilst we've known for some time about the prognostic value of point measurements of liver stiffness in PBC, what we didn't know anything about was the relationship over a period of time with regards to outcome and occurrence of serious clinical events. And I suppose that was the real aim of this study, to look over it longitudinally in what was a really huge international retrospective cohort. There were 24 centres in 13 different countries represented and I think in total actually looked at about 2,200 patients. And those patients that were included were those who had compensated PBC, who were on ursodeoxycholic acid. They had to have at least one reliable FibroScan measurement with a follow-up of at least 12 months. And they got rid of those patients who had overlap and those who went on to second-line therapy, even though they included that in a sub-analysis and also those patients who had unreliable liver stiffness measurements. With the primary outcome really been looking to see whether or not you develop these serious clinical events and over what time that actually occurred.

So if you actually look at the data and what they included, most of them again were females at the age mean of about 60. 25% had advanced liver disease, which they defined as a liver stiffness of greater than 10. And the follow-up period was okay, it was around about four years, and at least, the median number of FibroScans was around about three with an interval of around about two years between scans. So, you know, they did cover that really well and that primary outcome was met in 10% of patients. So 10% had a significant clinical event.

And I think the real thing that we took from this was that, actually, any increase of liver stiffness measurement over time was associated with an increased risk of a clinical event. And that initial increased risk was actually independent of initial age, of biochemical markers, or of response to ursodeoxycholic acid. And if you turn that on its head, therefore actually decreases in liver stiffness actually is associated with an improved prognosis.

There was a link that they describe between initial liver stiffness measurement and liver stiffness measurement over time, particularly in those patients who were less than 45 years old and in those who had an inadequate response to ursodeoxycholic acid.

I think this is a really important study with the key message that actually this is, you know, a true value that can be used not only for clinical trials and research, but actually in clinical practice. What did you think?

Gideon Hirschfield
No, I agree. And, you know, I thought it was a very impactful study because we're looking for better ways to understand our patients' journeys, better ways to look for which patients

benefit from treatment, better ways to look at what treatments are demonstrating benefit beyond just using biochemistry. So I think it meets all of those aspects. I suppose what I'm interested to know is, will it change your practice? I don't know how easy it is for your real population of patients to get their FibroScan. So what do you think the impact is on your day to day practice?

Emma Culver
I think we moved a number of years ago actually to try and, at least in the John Radcliffe Hospital, to do annual FibroScans of our PBC patients. I guess you're right, in the slightly more district hospitals and peripheral hospitals, that's not always feasible. And it was interesting to see, obviously, there was the mean interval here with two years between scans and so this was found at various different time points. So it's not, maybe necessarily that you have to do an annual FibroScan, even though obviously the annual FibroScan will probably give you the better detail. But I think it will start to give people an idea that actually that whenever they do that FibroScan, they can start to prognosticate, they can start to think about how this might affect their patients and they can start to potentially personalise things and get them into trials in the longer term.

Gideon Hirschfield
I agree. And I think also we'll be looking to see our second-line therapies are over time. I'm not sure you can expect FibroScan to go down immediately. You give a new drug, but over time to stabilise and to stay in a certain risk category.

And I think what I would say is that how I use FibroScan is very different to how it's being promoted in the community. I look at trends. I look at broad risk categorisation. I don't think about absolute numbers. I realise that there's a real push to have a number and a cut-off. But I'm looking at, is it low, medium or high risk? I'm looking at, is it stable over time? And, you know, I think that's important in how I interpret the information.

The other little sort of nuance is rising use of shear wave elastography. It may mean that it's got better access because lots of, if all ultrasound machines can do a shear wave, that means where patients live and they're having their ultrasound will have that shear wave information. And I have nothing to believe it wouldn't give the same information, but it may just be slightly different cut-offs. Also, you know, ultimately, like you said you want to individualize care. I don't think it's bad care if you don't do it every year and that's important to also get the message out there. And you're going to look at this result and you look at their biochemistry and you look at their symptoms for people living with PBC, and that's how you're going to choose your second and third line treatments.

What will also be helpful is to factor in the CAP score, which gives you some idea of fatty liver. And again, that's that individualisation process for trying to ultimately do everything we can to prevent progression to end stage liver disease.

Emma Culver

And I think you define a really important point there with regards to the CAP score as well, because I do think it has to be used in association with that given the overlap that we know that's occurring with metabolic related liver diseases.

Gideon Hirschfield
And one last comment is, although there's a little bit of discussion amongst people interested in managing PBC, what this data doesn't really help you with is how to use the first FibroScan value. Now I am sure that FibroScan is always prognostic, but in practice, it's much better to start your clock one year after treating with UDCA [ursodeoxycholic acid] because that's the time you have the biggest drop in liver stiffness from ameliorating the cholestatic component of the disease.

So liver stiffness will measure inflammation, cholestasis and fibrosis as you ameliorate that with anti-cholestatic therapies, you would expect that to drop in the first 6 to 12 months. And that's not because you've got rid of fibrosis, it's because you've dealt with some of the cholestasis associated liver stiffness. So I do try and counsel my patients to wait until the second reading to give them a better idea. Now clearly if your FibroScan is in the 20s... But this is the patients who present with FibroScans between 5 to 10, and where I don't want them to go away unnecessarily thinking that we are on a pathway to more complications.

Emma Culver
Yeah, really important points. So I think that brings us nicely on to our final two selections, which are really focused on sort of second-line therapy. And in the first of these, there's an interim biomarker data from a multi-centre randomised control trial, which was presented from Frederik Nevens in Leuven, Belgium. And I wonder Gideon if you could talk us through what was a long awaited but really kind of excellent study.

Gideon Hirschfield
I think this is a really nice study for you to have chosen to discuss, because in practice, we have seen second-line therapy. We have one conditionally approved drug, obeticholic acid and in many parts of the world, there's this off label use of bezafibrate. And at the same time we know that there are significant phase three clinical trials of new drugs that target PPARs [peroxisome proliferator-activated receptors] del-PARs [PPAR-delta] and drugs that have alpha and delta activity.

In this study, what we're now looking at is an extension of clinical practice. So we are looking at [an] industry sponsored study from Intercept, where they are making a combined therapy of obeticholic acid and bezafibrate. Highly logical, synergistic biology, FXR [farnesoid X receptor] and PPAR. And clearly from a patient perspective, the idea that you've got one tablet rather than two starts to be of interest when you're adding on therapy. And it's also logical because it builds on what we're doing already. 11% of my clinic, of the last 450 patients that I looked after are already taking OCA [obeticholic acid] and bezafibrate together. So having some randomised controlled data where the investigators have compared different does of bezafibrate against combination therapies is very impactful and very interesting.

And this interim analysis is of a population of PBC, 75% of whom have got a ALK phos greater than 1.67 who are randomised into four arms: bezafibrate 200, bezafibrate 400, OCA 5 to 10 plus bezafibrate 200, OCA 5 to 10 plus bezafibrate 400. We didn't get all of the nuances of the data. You never do in one of these oral sessions.

There's 62 patients in this analysis, but they look like patients who need second line treatment. Their ALP somewhere between 250 to 320 with a reasonable range. And as I said, 75% of them have got an ALK phos greater than 1.67. That magic Toronto number that has been adapted into practice saying, you know, we need second line treatment. And the difference here in the study is that previously we looked at an end point where we dropped the ALK phos less than 1.67 kept their bilirubin normal and saw a 15% decline between two readings. Where are we now? We are looking at normalisation of alkaline phosphatase. Why are we doing that? Because data from the global PBC Study Group says if your tests are normal, you do the best, normal ALK phos, normal bilirubin.

This is not rocket science Emma. This is just what we do in other liver disease. But normalisation is the new target and in this interim analysis, short term treatment, albeit but within you know, at week 12, you know, there are reporting that in the arm with bezafibrate 400 and OCA 5 to 10 that they're up to 58% of normalisation. Now, that's great. This really confirms what we're doing in practice. It tells us that we're moving the field forward.

What are my caveats? Well normalisation is always relative to where you started. And that's obviously the same for everyone doing these kind of studies. So, you know, it does depend on how high your ALK phos is. And also, this is at 12 weeks and we don't know, ALK phos does continue to drop a bit over time. But regardless, this is really exciting, a combination therapy with two drugs which are synergistic in one tablet where we can see rapid improvements in liver tests by week 12 is very, very exciting and is moving the field of PBC into a new era, if this can be shown to be safe over the long term and we can see what the optimised dose is.

Adverse events are very important. One of the Achilles heels of obeticholic acid is pruritus. One of the opportunities of bezafibrate and new therapies that are coming, other PPARs, is their predicted antipruritic effect. And this was clearly demonstrated again in the study, although I'm not sure we saw the exact itch scores, but generally pruritus was not an issue. So this is also something that I think is really appealing.

So this was, you know, important. It's a hard study to do, to find patients. Yes, there's relatively small numbers, but this is concept generating in terms of randomised data. It's taking our real world experience and it's done it in clinical trials, which is what we need if we're going to have labelled therapies. And it shows that bringing together these two important pathways therapeutically, obeticholic acid a FXR agonist and bezafibrate a pan-PPAR agonist, bringing them together into one tablet is an effective way to get rapid, potent and efficient anticholestatic and anti-inflammatory impacts for our patients, which we predict but we'll need to prove will be of benefit to their long term outcome and to all the points we mentioned, will, we predict, reduce the chance of cirrhosis and HCC. Will, we

predict, change their FibroScan over time. So building on some of the other data. So that was my take.

Emma Culver
It really is, you know, an excellent study and small numbers, as you said. I think obviously some of the things that were pointed out immediately after the presentation in the subgroup analysis, the dose of ursodeoxycholic acid was a little bit lower than we'd expect, definitely not meeting the 13 to 15 milligrams per kilogram. And I do wonder if that's just, you know, data crunching. But obviously that would be a concern if patients weren't on effective or optimal therapy. And maybe some of those patients were not on therapy with ursodeoxycholic acid, but that didn't really come out in the presentation.

I guess the other thing is, is, you know, I was quite surprised by the low rates of things like myopathy that often we find in patients with fibrates. But I actually think that was probably related to everyone stopping their statin. And I know necessarily in clinical practice, not everyone who goes on bezafibrate will necessarily stop their statin. So I think that was a little bit of a message to me and maybe to others as well that actually, much lower rates of myositis and myopathy described in this small clinical trial with regards to numbers than you necessarily see in clinical practice.

And no evidence of renal dysfunction either, which was another really important take home message. And actually if you looked at the kind of LDL and HDL cholesterol levels and the overall cholesterol levels, again, there seems to be a degree of improvement. And I know that cholesterol and cardiovascular outcomes are not necessarily synergistic in the context of PBC, but I guess that's relatively reassuring for those patients who are having to stop their statin and go on a fibrate.

The other thing I guess as well in that study is they also took out, if I recall from actually doing that study, they actually took out patients who had gallstones or any who'd had a previous cholecystectomy. So again, I think we're going to have to think about those patient groups that we give this combination therapy to in real life. And so that leaves the question really about a phase three. Do you think, given the fact that you've commented already that 11% of your patients are already on combined therapy and the challenges that there were being able to recruit to this study, that it would be possible to do a phase three combination dose study given the way that actually we may incorporate this in clinical practice?

Gideon Hirschfield
I think the answer is yes. Nothing's easy in clinical trials, whatever disease you work on. But what we're trying to do here is think about the global management of PBC, for everyone with PBC, regardless of where they live and to increase the uniformity of care. And labeled therapy increases uniformity of care. Not everyone gets a chance to go to a clinic where someone is really focused on PBC and to have a clinician who thinks about all those nuances, the statins, the lipids, the interactions.

And that's where we do need licensed therapies. It makes it much more linear, it makes it much more predictable, it makes it much easier to write clear guidelines. So yes, it will be a

challenge to recruit and it will have to have global investigators. But it will be a worthwhile effort and I think will ultimately reach our goal, which is to improve liver treatment and to aim for normal tests, not for, you know, just suppression.

We need to move on to the last abstract, Emma. But this is actually a great abstract to finish this really enjoyable conversation because this is now trying to help people who are using second-line treatment know whether those second-line treatments are working. And this is the development and validation of a score predicting response to obeticholic acid in PBC, the OCA response score. And I'd really be interested to know on this abstract presented by De Vincentis from the Italian group, some of your high level summaries about this abstract and why you thought this was newsworthy.

Emma Culver
Yeah. So again, I really like this and actually I was quite interested in the kind of concept of trying to develop tools to predict treatment response and thinking about actually personalising treatment strategies. And if indeed that's even possible with the data we have available. And I guess this was data that was extracted from the Italian RECAPITULATE study, which was presented as a separate oral presentation earlier on in the day. And that's real world data, retrospective, looking at obeticholic acid therapy initially to look at efficacy and safety in Italy over a good geographical distribution. And in this particular analysis they were only included if you knew their alkaline phosphatase at the start of OCA treatment and they had a minimum of six months' observation.

They did have to choose their values quite carefully and they chose the candidate variables of which there was 12, based on the fact that they had to exclude those with missing variables. So one of the caveats is that they had to exclude things like platelets or albumin or INR [international normalised ratio] because they were more than 20% of missing values, but they were able to develop a response score and then actually a response score plus, using these 12 variables.

So the OCR plus, just to mention it, is basically where they find these predictive models and then they added the six-month response of alkaline phosphatase and also bilirubin to that. And their main outcomes where the three outcomes we've been talking about before. One of them obviously being the ideal, which is normalising the alkaline phosphatase, the ALT and bilirubin, one of them being that the standard alkaline phosphatase less than 1.67 times the upper limit of normal, and the other one is the POISE criteria, which was this alkaline phosphatase and also the normalised bilirubin.

So they created these 12 candidate variables in a study or a group that were mainly females, again, of 60 years, median duration of around about seven years. They didn't exclude their overlapped patients, so the overlapped patients got included in this, but they had to officially have a stable dose of immunosuppression over six months and be a biopsy diagnosis. And the third of the group was cirrhotic. A third of the group were itching at baseline and they had 24 months mean follow up on obeticholic acid.

And I think actually, as we expected in terms of how many people met the 12-month outcome actually in terms of normal range on these on the obeticholic acid only that was only 10%. If you use the ALP less than 1.67 times the upper limit of normal, it was 60%. And if you use the POISE criteria, it was around about 40%.

But actually at the end of all of that, they came up with a score and then an advance score that was able, they felt, to predict those patients who would need second-line therapy. Not necessarily predict those patients who wouldn't respond, but predict those patients who would meet need for obeticholic acid. And the main sort of factors that we're including that were patients age, their pruritus, specifically their symptoms and the biochemical values we know, which is of alkaline phosphatase, ALT and the bilirubin.

And there was good discriminatory values and there was also good calibration scores. And they say that these scores didn't differ based on gender, on cirrhosis, on higher ALT at baseline, even though they did acknowledge there was a small portion who weren't on ursodeoxycholic acid and there was a small proportion who went on to fibrates a little bit later in the study.

But I think it's quite nice to have a baseline score that you could potentially use. And I guess the real question is, you know, would you use this in clinical practice?

Gideon Hirschfield
A good question. I don't actually see much clinical utility in this. What I think is relevant, just conceptually, is that we do need better understanding of who responds to what. And when I was sitting in the audience thinking, you know, I don't always know why one person responds to OCA rather than fibrates, and the other person seems more fibrate sensitive and less OCA. And that's relevant when I'm trying to understand mechanisms and synergy in the context of joining drugs together. I think this is a bit self-fulfilling, if I'm honest. And I couldn't quite immediately see how I would use it until it's been validated a bit more.

But it's important to do because we do need to tease away at who responds and why, so that we can more effectively use treatments. And this will become more relevant when our choice of therapies is improved in the coming years to have a family of second-line treatments which have got conditional approval and which reach towards our goal of normalising tests and improving symptoms.

But we may need to just draw this really exciting discussion to an end Emma. I might just ask you to spend just one minute of summarising your take on EASL 2023.

Emma Culver
I think it was an excellent conference and I think I started this by saying that. I guess the key things that I have taken from this is, firstly that we really should be starting to look towards normalising alkaline phosphatase and not necessarily settling for a higher alkaline phosphatase. I suppose it depends on the group of patients that you choose to do that or whether that should be everyone in the system that we have currently. And also that we should be thinking about how we get and move forward with regards to our second-line

therapies, our clinical trials that will help to actually look prognostically at fibrosis markers, not just necessarily biochemical response and serious clinical events, which we really care about in these groups of patients. And lastly, how we can incorporate liver stiffness into this. How about you?

Gideon Hirschfield
I agree totally. I think you've summarised it very well. It was a great conference. We got the momentum going for PBC and other autoimmune liver diseases. But what's even more exciting, if you think about ASLD 2023 in November in Boston, we predict from what we heard, that there will be two phase three clinical trials presented on the future treatments of PBC, which is staggering when you think about it, to be hoping to hear for our patients and our colleagues phase three data of new molecules being developed for PBC. So I think there's more excitement coming.

So Emma, it was really a great pleasure as ever to chat to you and to record this podcast and I hope people find it interesting. Thank you. And I hope everyone has a good day.

Presentation list:

Presentation ID OS-048-YI
Risk of cancer and subsequent mortality in primary biliary cholangitis: a population-based cohort study of 3,052 patients
Oral Presentation: Axel Wester (Stockholm, Sweden)

Presentation ID OS-073
Prognostic significance of liver stiffness progression in primary biliary cholangitis
Oral Presentation: Christophe Corpechot (Paris, France)

Presentation ID OS-046
Results from a planned interim analysis of a randomized, double-blind, active-controlled trial evaluating the effects of obeticholic acid and bezafibrate on serum biomarkers in primary biliary cholangitis
Oral Presentation: Frederik Nevens (Leuven, Belgium)

Presentation ID OS-070
Development and validation of a score predicting response to obeticholic acid in primary biliary cholangitis: the OCA response score (ORS)
Oral Presentation: Antonio De Vincentis (Roma, Italy)