The Second International Primary Lateral Sclerosis (PLS) Conference

Promoting Research in PLS: Current Knowledge and Future Challenges

May 3rd & 4th 2019
Philadelphia Airport Marriott Hotel

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Conference Sponsors:
ALS Association
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NINDS/NCATS
Spastic Paraplegia Foundation (SPF)

May 3rd (Friday) First Day

Welcome and Introduction - Hiroshi Mitsumoto
Hiroshi said that PLS was first described by Charcot in 1865; Beal & Richardson wrote about it in 1981; ALS Centers were opened in the 1980s where PLS was treated. In 2002 the SPF was founded. I did not get further notes on the history.

PLS: Cause unknown. Nosology unknown. Natural History: Limited. Clinical Features; Variable. Diagnosis: Uncertain. I did not get further notes on description.

Clinical Spectrum - Moderator: Michael Benatar, U Miami (all talks were limited to 5 minutes)
1. PLS Registry, Cross-sectional (Christina Fournier - Emory U)
233 patients at 20 NEALS sites
2. PLS in Netherlands (Leonard van den Berg, Utrecht U)
Some families have both ALS & PLS. He talked about how different groups of PLS lead to either ALS, HSP or FTD.
3. PLS in Germany (Albert Ludolph, U Ulm)
Albert said EMG is not very helpful in the diagnosis of PLS. He talked about how ALS staging applied to PLS. He said they discovered that cognition in PLS is not different from cognition in ALS. PLS genes he mentioned: SQSTM1, KIF5a, KIF1a
4. What is not HSP (John Fink, U Michigan)
John said this is a difficult question because the diagnosis of PLS is qualitative not Quantitative. It is like asking how many grains of sand equals a heap of sand? Lower extremity weakness & spasticity is a symptom of many rare diseases. Early HSP is hard to separate from cerebral palsy. SPG7 is both a gene for PLS & HSP. There are also common genes for both PLS and ALS. There is a vibration impairment in both HSP and PLS. HSP commonly has hyper flex in arms but not dexterity problems in hands.
5. What is not ALS? (Terry Heiman-Patterson, Temple U.)
PLS is a prolonged illness. ALS is usually much more progressive. PLS has stiffness in 47% of cases and in ALS it is in 4% of cases. There is no atrophy in 2% of PLS. No fasciculations, normal sensation, UMN presentation.
6. What is Mills variant? (Martin Turner, Oxford U)
Charles Karsner Mills 1845-1891; unilateral PLS. Gastaut & Bartol. Hemiparetic PLS: revisiting Mills Syndrome.
Discussion, Q&A, 45 minutes
Again it was said that EMG was not good for diagnosis and showed no change. Teepu Siddique said all neurological degeneration is asymmetrical. PLS is a pathological term.

There was a long discussion of defining PLS based on characteristics that are often variable. John Fink said dorsal column impairment can appear in PLS.
Coffee Break
Cognitive Impairment (Moderator: Mary Kay Fleeter, NINDS)
1. Cognitive and behavioral impairment in PLS (Jennifer Murphy, UCSF)
Since the 1990s they have been measuring cognitive impairment in PLS. There is evidence from MRI, PET, SPECT (frontal region). Piquard (2006) studied 20 PLS patients. Similar to ALS. Executive function. Canu: 10 of 21 had it. Agarwal: “globally impaired”. 39% of PLS and 31% of ALS are cognitively impaired. The PLS group had more behavioral change than other groups.
2. Similarities and differences from other types of cognitive/behavioral impairment (AD, PD, ALS, HD, ET) (Edward Huey, Columbia U).
Social cognition is largely affected.
Discussion: 45 minutes.
Siddique was strongly critical of the controls used in the study. He said that the controls were probably college students and professors and so it biased the study. Other topics of discussion mentioned the pseudo bulbar effect (PBA), depression versus grieving, predominantly executive cognition, the prevalence and nature is the same in both PLS and ALS. Mary Kay Fleeter admitted that they may have had a selection bias because her PLS subjects had to travel to her location for the study. The FTD studies are biased because they are largely dependent on the reports from caregivers and caregivers are under a big burden.

Electrophysiology (Moderator: Richard Barohn, U Kansas)
1. Cortical excitability (Matthew Kierman, U Sydney)
He showed a graph comparing cortical excitability of PLS, ALS and HSP. The graph showed PLS with about twice as much as ALS and HSP showed no cortical excitability.
2. CMCT by TMS (Seth Pullman, Columbia U).
TMS is Transcranial Magnetic Stimulation. TMS is an objective marker of UMN disfunction in PLS. TMS quantifies corticospinal tract involvement with PLS.
3. Intermuscular Coherence for PLS? (Kourosh Rezania, U Chicago)
Assessment of shared neural drive - very strong correlation.
4. What EMG changes constitute “no LMN involvement” in PLS? (Zachary Simmons, Penn State).
PLS do not lack LMN involvement entirely. PLS does not mean that EMG is normal. Motor Unit Number Estimation (MUNE). Mean MUNE in PLS was greater than ALS, mean MUNE in PLS was less than controls.
Discussion and Q&A 45 minutes
EMG is highly variable. John Fink asked if what the patients are using for control of spasticity (baclofen) was considered. The answer was that they had to be off of baclofen.

LUNCH

Neuroimaging (Moderator: Erik Pioro, Cleveland Clinic)
1. Background to PLS neuroimaging and functional insights (Martin Turner, Oxford U)

Martin showed many images of PLS brain damage that are not found in ALS.
2. Overlapping and distinctive imaging signatures in PLS and ALS. (Peter Bede, Trinity C.)
More brain images were shown. I did not get notes from this speaker.
3. Glial activation in patients with PLS assessed with [11C]-PBR28 PET (Sabrina Paganoni, MGH)
There is increased glial activation which is greater in ALS than in PLS. Deep white matter forming corticospinal tracts. She closed by thanking SPF.
4. Motor Neuron Subsets in ALS and PLS: From development to degeneration (Georg Haase, INSERM)
Light sheet microscopy of motor neurons. Cerebral spinal involvement.
Discussion Q & A:
John Fink said corpus callosum involvement is common in SPG4 HSP.

Measuring PLS Disease Progression (Moderator: Mamede de Carvalho, U Lisbon)
1. Novel PLSFRS (Madison Gilmore, Columbia U)
They developed a 68 point PLS progression measurement PLSFRS Functional Rating Scale.
2. Quantitative UMN scale (Lauren Elman, U Penn and Suma Babu, MGH)
They mentioned the drug Udextra for controlling bulbar dysfunction. They said the Penn Upper Neuron Score is a binary scale. 15 patients, 33% female, 20% have bulbar onset. median age: 62. Median ALSFRS. A lot of other numbers were listed that I couldn't get. PUMNA uses to date: no correlation with age, no correlation with disease duration. Negative correlation with ALSFRS-R (R=.45, p = .007)
3. Quantitative Motor Performance (Pat Andres, Formerly MGH)
He described at length the pathology of motor neuron disease, described UMN spasticity, dysregulation of innervated muscles, etc etc. Contextual Framework of WHO (1980). They measured quality of life. (this might be something we could try to measure); Another measurement is Timed Up and Go (TUG), Speech test, Purdue pegboard test, finger foot tapping,
Discussion Q&A:
Someone asked if these tests are ever used to predict when a patient will need a walker or wheelchair and the answer was no. Michael Bulbar asked why speed of movement was not measured for upper motor disease progression.
Coffee Break

Biological Biomarkers (Moderator: John Ravits, UC San Diego)
1. Mitochodrial bioenergetics (Giovanni Manfredi, Weill-Cornell U)
Primary skin fibroblasts, 177 sporadic ALS, 34 sporadic PLS, 12 C90rf72, 94 ctrl. Mitochodria are hyper energized. Hightened energy expenditures lead to higher stress. 25 algorithms & used AI to find a pattern. Great discriminator between ALS & PLS.
2. Lipidomics (Estella Area-Gomez, Columbia U)
Signature of 22 Lipids. Triglicerides not from diet are higher in both ALS & PLS. Cholesterol trafficking is reduced in ALS & PLS (significant). Increased proportion of VLDL & LDL vs HDL in ALS & PLS. There are changes in many categories of lipids. Mitochondria Associated ER Membranes (MAM) is disrupted in ALS. Lipomics analysis

of serum discriminates ALS & PLS. PLS is a slow progression of ALS. She thinks people with PLS have some sort of protective factor that protects them from the worst aspects of ALS.
That evening, David Marren sponsored a dinner at the Hall of Flags, Houston Hall (3417 Spruce St. Downtown) It was a large room with a marble floor and wood walls that looked very historic with many flags hanging from the ceiling. Two buses took everyone to the dinner. I sat with David Marren on the bus and we had a great conversation. Hiroshi arranged a debate in front on a stage between Jinsy Andrews, Mamede de Cavalho, Mary Kay Floeter, Orca Hardiman and Erik Pioro. The debate question was whether PLS is a separate disease or whether it is really a form of ALS. Mark Kay Floeter was sitting at the same table as I and she had refrained from drinking any wine because she wanted to be ready for the debate. Erik Pioro was also at my table and he did not refrain so adamantly. The first debaters were of the opinion that PLS is of the same phenotype as ALS. They said that PLS is a form of ALS from a phenotypic view. Genetic PLS is an ALS phenotype. The cognitive changes are the same. There are more commonalities than differences. Mary Kay Floeter argued that they are different diseases. She said that for 120 years scientists have been arguing that they are different. 1/4 of PLS patients after 15 years turned out to have something else. Erik Pioro said while PLS has a much slower progression, there is an overlap in lower neuron changes in PLS. The question is what is protecting the lower neurons. Imaging shows a lot of commonality. BRAC region is the same. The process is just more slow in PLS. It is a similar disease - just a different degree - similar pathogenesis - something is preventing lower motor development. Everyone had a great time. I thanked David Marren and his family for sponsoring this event.

Second Day

Genetics (Moderator: Vincenzo Silani, U Milan)
1. Sporadic PLS and familial PLS (Teepu Siddique, North Western U)
Teepu talked about genetic pleiotropy and pathologic pleiotropy which was first described by Jean-Martin Charcot 1825-1893.
2. PLS in familial ALS (Phillippe Corcia, U Tours)
Phillippe works for ENCALS or European Network to Cure ALS.
3. Gene for familial PLS (Guy Rouleau, McGill University)
TMEM175 also involved in Parkinson's disease.
4. Exome and genome sequence study (Matt Harms, Columbia U)
C9ORF72 (also ALS) PSENI VUS SPG7 etc.
GTAC study PLS 6%, GTAC cosmos NYGC (sources of genes)
Looked for 289 genes. C9ORF72 expansions 2.8% (7% in ALS)
Many genes were listed that I could not write all of them down. I noticed SPG78, SPG49 and SPG52.
Discussion Q&A
There was a good discussion on the hypothesis that people with PLS have some sort of protective genes that protect them from full blown ALS.

Keynote Speaker: Neuropathological and Biological View on PLS, ALS and FTD - Dr. Ian Mackenzie (U British Columbia)
Ian showed many pictures of the neuropathology of ALS. Pictures of brains and spinal cord were shown. He also showed the spectrum of ALS and FTD. Bumina bodies are unique for ALS. He talked about ubiquitin IHC. He showed how ALS and FTD overlap. TDP-43 in the ALS motor cortex not found in aging. There are no Bunia bodies in PLS. 14/20 FTLD-TDP had motor cortex & CST depredation (PLS). PLS - TDP43 scarce. PLS with nonspecific pyramidal system degradation. FTLD-TDP with No LMN pathology. PLS with minimal TDP and LMN pathology. Most PLS has TDP pathology. Similar to ALS. Associated with FTD. ALS & PLS represent an MND-TDP spectrum, not a continuum. Hypothesis: PLS has some factor of protection.
Discussion Q&A:
Erik ???? asked about protective factors. No real answer. There was a question about the word “spectrum” vs “continuum”. A spectrum is an array of things progressively aligned while a continuum is a solid column progressively aligned.

Coffee Break

Critical Trials in PLS (Moderator: Merit Cudkowicz, MGH)
1. Symptomatic Treatment: Dalfampridine trial (Dale Lange, HSS/Jim Wymer, U Florida)

This drug has been very effective with MS. It increases walking speed and leads to an improved quality of life. Their goal is an increase of 20% in walking speed.
2. Disease-modifying treatment: Are we ready for a PLS clinical trial (Hiroshi Mitsumoto, Columbia U)
Qualities of PLS: Rare: Yes. Biology Understood: No. Medication available: Yes/No. Clinical Consortium established: Yes. Diagnosis well defined: Not yet. Clinical assessment developed: Yes. Etc Etc. They need sensitive scale & biomarker, a goal attainment scale, time to fail, a natural history study of PLS, multi site study, a patient registry, better diagnosis criteria.
Discussion Q&A:
Someone mentioned how the drug Spinraza was passed through a clinical trial for SMA. The answer was that they received a congressional mandate.

Trainees and Young Investigator Presentations (Moderator: Frank Davis, SPF)
I thanked Hiroshi Mitsumoto for including SPF in this very exciting and productive conference. I said that I am sure everyone agrees that the real hope and excitement for PLS research lies in the future and the future belongs to the young and strong neurologists of the future. I introduced them in alphabetical order. They each got up after being introduced and talked about their plans for their career. Most were going to concentrate on ALS and many mentioned PLS. No one mentioned HSP.

Then, Dr. John Fink introduced Dr. Debra Warden. She has PLS and was in attendance. I had had lunch with her and her husband the day before. She is a neurologist and in her career worked for the military specializing in head injury treatment. Her husband works for NIH. They then showed the film available on Youtube that was a patient’s

perspective on having PLS. It is a very moving and informative film about PLS. I asked Debbie if we could post this video on our website. She said yes and said she could send it from her computer to mine. I said I'm not sure there is any time for that and wondered if she could email it to me. She said it is too large to email but it is available on Youtube so I can put a link to that on our website. She later handed me a DVD with that film on it.

LUNCH I talked with someone that is going to email me soon. He was interested in getting DNA samples from our PLS members. I suggested that they set up a booth at our Annual Conference in San Antonio. After some discussion, he thought it might be more productive to send out the sample kit for saliva samples to everyone willing to participate in our PLS members. I also suggested to Michael Benatar with CReATe the same suggestion I made to Stephan Zuchner a couple of weeks ago that they set up a booth at our AC for HSP DNA samples. I thought that they could just use their San Antonio CReATe doctors to get the DNA samples. He said that he will sure think about it.

The First Panel Discussion: The PLS Diagnostic Criteria (Moderator: Martin Turner, U Oxford) Participants: Richard Barohn, U KS; Phillip Corcia, U Tours; Matthew Harms, Columbia U; Matthew Kiernan, U Sydney; Jeffrey Statland, U Kansas.
EMG is a critical part of the diagnosis of PLS.
Typical PLS: UMN signs in at least bulbar > 4Y
Probable PLS: UMN signs in arms and legs. > 4Y
Possible PLS: UMN signs in legs only >4Y. (This questionable to me as it is also HSP)
Suspected PLS: UMN signs in bulbar and/or spinal regions.
PLS Plus: Other neurological diseases such as FTD & Parkinsonism

pseudo bulbar palsy = PLS
upper motor neuron lesion
Spinal Myelopothy is other disease that looks like PLS.
"Basket Design" is what Sabrina Paganoni said is a clinical trial where more than one disease can be involved in the same trial at the same time.
Coffee Break

The Second Panel Discussion: International PLS Registry (Moderator: Pam Factor-Litvak, Columbia U) Participants: Orla Hardiman, Trinity C; Albert Ludolph, U Ulm; Mitsuya Morita, Jichi MC; Sabrina Paganoni, MGH; Teepu Siddique, Northwestern U; Leonard van den Berg, U Utrecht.)
A rare disease registry in Japan is Nanbyou for intractable diseases. They have about 100 PLS people in that registry. 47% of neurologists in Japan consider PLS a subtype of the ALS spectrum. Specimens are not collected. Teepu Siddique mentioned the Shamanad Criteria that was developed at the first PLS conference. He is going to email information about it to those in attendance. He also spoke about how many of these same issues were discussed at the first PLS meeting 15 years ago. There was a long discussion where Hiroshi was trying to get anyone to agree on the definition of PLS, how a study could take place, when, where - all of which no consensus was being

reached. Hiroshi seemed frustrated and acted like no one could go forward until a consensus could be reached.

The Final Summary: Where should we go from Here? (Moderator: Hiroshi Mitsumoto, Columbia U) Participants: Michael Benatar, CReATe; Robin Conwit, NINDS; Merit Cudkowicz, NEALS; Frank Davis, SPF, toby Ferguson, Biogen; Bryan Hill, MTP; John Ravits, WALS; Leonard van den Berg, ENCALS.
The WALS group was created earlier than NEALS but is still in existance. It stands for Western ALS group. There was talk about creating an organization from some of the people in attendance to promote treatment for PLS. There was a lot of discussion on whether different neurologists could combine their PLS patient information and they went around asking how many patients each neurologist had. I suggested that there is probably an enormous overlap as many PLS patients I know go to several different neurologists. I mentioned that SPF has over 950 people who claim to have PLS. As always there were a lot of questions about the validity of our SPF data. I said that I had taken out all people that are just PLS family or caregiver members or anyone that only suspected they had PLS. I said that I am sure that some of our people with PLS could have turned into ALS and not reported it to us. It could be that some of our PLS members could have died with a living spouse and the spouse has not reported it to us. I said that we could easily ask all of our PLS members to tell us if they were diagnosed by a board certified neurologist. Someone in the audience said that wouldn't be any good. Teepu Siddique seemed encouraged by our data and I asked him if he could email me with a list of sample questions, he would like to be on our survey to PLS members. He agreed.

When my turn came up to comment on "where should we go from here?" I changed my mind on what I was going to talk about, about 5 minutes before I started talking. I had become frustrated by the seeming lack of a possible start to getting much done because of a lack of consensus in the audience. I had planned to talk about the patient experience with PLS as Hank Chiuppi had described it to me but David Marren and Dr. Debra Warden had already done an excellent job of describing the patent perspective better than I could ever do.

I expressed my thanks for being asked to participate in this very exciting and promising conference. I described what SPF does, that we support about $600,000 in research every year. That, while $600,000 is a drop in the bucket compared to the Ice Bucket ALS Challenge, it is still a considerable amount and can do a lot for HSP and PLS research. We use half of our annual funds to support PLS research and half to support HSP research. I said we have no problem finding, through our S.A.B., great research for HSP but we have many years had a very difficult time finding good research to support for PLS. I said we have gotten around that problem by supporting educational grants for two people: Christina Fournier and Sabrina Paganoni which has obviously worked out to be a great investment. I asked everyone to give them both a round of applause. `I then said that I had planned on explaining to everyone the patient experience of PLS but David Marren and Dr. Debra Warden had already done an excellent job of doing that. I said that I have been asked to suggest where should we go from here and I am

concerned by how Teepu Siddique said that we are talking about a lot of the same issues that were discussed 15 years ago and that Hiroshi Mitsumoto seemed so frustrated because a universal consensus could not seem to be reached on so many aspects of PLS. I said that my career was as a business man for 30 years and so I would like to suggest a business person's and patient's perspective on where we go from here. I said no successful business would wait for all of their employees to agree on what their next new product would be before putting it on the market. I said while most people think the best approach is: "On your mark, get set, go", the business world is more successful when the attitude of "On your mark, go, get set" is applied. Action should be taken, even if there are still a lot of things unknown and then compensate as you go forward. People with PLS want action to take place as soon as effectively possible.

I then said that a lot of people with PLS go to neurologist after neurologist and they are not informed by any of their neurologists about SPF. They finally find SPF through an internet search and SPF is a great help to their quality of life. I asked every neurologist in the room to please notify their patients with PLS and HSP about SPF.

I was worried that I had offended Hiroshi Mitsumoto with my talk so, that night, I emailed him to thank him and congratulate him again for the excellent conference. I said that I hoped my closing comments did not seem unappreciative or disrespectful of him or his team. His response was that "on the contrary, your comments are so important!! I will not end my goal at the end of the conference. We have a lot of work to do. Thank you so much for your continuous support!"

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There was attached to the agenda, many scientific reports on different aspects of PLS. If anyone wants to read them, let me know and I'll scan them and send them to you.