The 39th Plenary meeting & 40 years Jubilee meeting of the Scandinavian Sarcoma Group

The 11th meeting of the Scandinavian Sarcoma Group for Nurses & Physiotherapists

May 8-10, 2019
Bergen

PROGRAM

www.ssg-org.net

Conference venue

Grand Hotel Terminus Bergen
Zander Kaaes gate 6, 5015 Bergen, Norway
Phone: +47 55 21 25 00
Web: Grand Hotel Terminus Bergen

Preface

We wish all members of the Scandinavian Sarcoma Group and external guests welcome to our 39th plenary meeting in Bergen, Norway, May 8-10, 2019. This meeting also coincides with the 40 years jubilee for our group. We are excited to announce the participation of several excellent invited speakers, from both European countries and other continents.

The meeting will begin Wednesday morning with a workshop on Li-Fraumeni Syndrome arranged by the Norwegian Working Group for Hereditary Cancer where SSG-meeting participants get the possibility and are encouraged to participate. Basic research, diagnostics and treatment of bone- and soft tissue sarcoma will be extensively covered throughout the meeting, and also how to handle lung metastases with surgery or radiotherapy. A session on abdominal sarcomas including the gynecological entities along with the assets of transatlantic collaboration will form a central part of the agenda. Additionally, novel design of clinical trials will be presented and hopefully motivate SSG to participate in more studies! The nurses- and physiotherapy program will focus on handling wounds, alternative treatment and rehabilitation.

Many of the sessions will be joint sessions between physicians, scientists, nurses and physiotherapists. A collaboration between these groups is important for the development of new treatment strategies for sarcoma patients. The Scandinavian Sarcoma Group subcommittee for Nurses & Physiotherapists was established 12 years ago and has demonstrated its importance in many fields.

Lastly, there will be free presentations, a poster session and a panel discussion of the SSG´s 40 year's journey through sarcoma diagnostics and treatment. We hope that all SSG members and other participants will find the conference inspiring and take the opportunity to meet and discuss collaborative work.

We wish you an enjoyable and memorable stay in Bergen!

Kirsten Sundby Hall
Past chairman of the Scandinavian Sarcoma Group
Nina Jebsen
Local coordinator for the meeting in Bergen
Akmal Safwat
Present chairman for the Scandinavian Sarcoma Group

Program responsible

SSG Board:
Kirsten Sundby Hall, Mikael Eriksson, Jukka Kanerva, Thomas Baad-Hansen, Fredrik Mertens, Hans Kristian Haugland and Eva-Mari Olofsson.

Local program committee: Nina Jebsen, Hans Kristian Haugland, Ola Myklebost, Kathrin Maria Pulverer, Anne-Lise Salbu and Rune Haaverstad.

Program committee for Nurses and Physiotherapists: Anne-Lise Salbu, Lotta Våde and Stine Næss.

Next SSG meetings:
24th Working Group Meeting of the SSG, December 2-3, 2019, Malmö
40th Plenary Meeting of the SSG will be determined.

Contents

Workshop Li-Fraumeni Syndrome

Wednesday 8 May, 2019

09.00-10.00	Registration
10.00-13.00	**Plenary session with the Norwegian Working Group for Hereditary Cancer and SSG-meeting participants** Room: Terminus Hall, Grand Hotel Terminus Chairman: *H. Høberg-Vetti, Bergen*
10.00	Welcome *H. Høberg-Vetti, Bergen*
10.05	Introduction to Li-Fraumeni syndrome. - history, genetics, cancer risk *L. Mæhle, Oslo*
10.25	The use of whole body MRI in surveillance of patients with germline *TP53* mutations. *M. Ballinger, Sydney*
10.55	MRI surveillance within the SWEP53-study of patients with germline *TP53* mutations in Sweden. *L. Blomqvist, Stockholm (TBC)*
11.25-11-40	*Short break*
11.40	Cancer treatment in patients with a germline *TP53* mutation. *H. P. Eikesdal, Bergen*
12.00	Raising patient awareness about sarcoma. *O-J. Norum, Oslo*
12.15	Psychosocial aspects of intensive cancer screening in Li-Fraumeni syndrome. *M. Ballinger, Sydney*

12.45 Discussion

13.00–14.00 *Lunch*

14.00-16.30 **Workshop Li-Fraumeni syndrome**
Room: Group room, Grand Hotel Terminus
Chairman: *L. Mæhle, Oslo*

14.00 Surveillance of patients with germline *TP53* mutations in Denmark.
K. Wadt, Copenhagen

14.15 Surveillance of patients with germline *TP53* mutations in Norway.
L. F. Engebretsen, Trondheim

14.30 The challenges in establishing a national surveillance study – experiences from SWEP53.
S. Bajalica-Lagercrantz, Stockholm

15.00-15.15 *Short break*

15.15 Case presentations
By participants

15.45 ERN Genturis guidelines for Li-Fraumeni syndrome
S. Bajalica-Lagercrantz, Stockholm

16.00 The next steps. Scandinavian collaboration?

16.30 move to…

Joint session with SSG Plenary meeting

16.40 L1 Bone sarcoma - Genetics and oncological therapy
Room: Terminus Hall, Grand Hotel Terminus
D. Thomas, Sydney

SSG Plenary Meeting

Wednesday 8 May, 2019

12.00-14.00 Registration
Exhibition stand & Poster exhibition (all day)

13.00-14.00 *Lunch buffet*

Joint session with physicians, scientists, nurses and physiotherapists

14.00-14.10 Welcome by C. Trovik.
Introduction by N. Jebsen and A. Safwat.
Room: Terminus Hall

14.10-17.10 Session I: Challenges in Bone Sarcoma
Chairmen: *P. Bergh, Gothenburg, O. Myklebost, Bergen*

14.10 L2 Bone sarcoma - Molecular biology/pathology.
H. K. Haugland, Bergen

14.30 L3 Bone sarcoma - Surgery.
R. Grimer, Birmingham

15.00 L4 Targeting the genomic chaos in osteosarcoma with PARP inhibitors.
O. Myklebost, Oslo

15.10 L5 Bone sarcoma - Oncology.
Ø. Bruland, Oslo

15.30-16.00 *Coffee break*

16.00 L6 Morphologically distinct cell population in patient-derived Ewing sarcoma cells display variation in drug sensitivity .
A. Papakonstantinou, Stockholm

16.10	L7	Particle therapy for bone sarcoma. *J. E. Dale, Bergen*
16.30	L8	Off-label use of pazopanib for patients with metastatic bone sarcoma. *N. Aggerholm-Pedersen, Aarhus*
16.40	L1	Bone sarcoma - Genetics and oncological therapy. *D. Thomas, Sydney*
18.30		Departure for social program (see separate section).

SSG Plenary Meeting

Thursday 9 May, 2019

08.00-08.30		Registration Exhibition stand & Poster exhibition (all day)
08.30–10.40		**Session II: Challenges in Soft Tissue Sarcoma** Room: Terminus Hall Chairmen: *K. Boye, Oslo, T. Baad-Hansen, Copenhagen*
08.30	L9	Monitoring disease evolution by liquid biopsies. *L. Meza-Zepeda, Oslo*
08.50	L10	Radiology, strategy for optimal biopsy. *O. Angenete, Trondheim*
09.10	L11	Molecular biology and pathology, strategy for optimal biopsy and diagnosis. *F. Puls, Gothenburg*
09.30	L12	Treatment of low-grade fibromyxoid sarcoma: Systematic review and experience of a tertiary cancer center *B. Engelmann, London*
09.40	L13	Soft Tissue Sarcoma - Surgery *M. Mørk-Petersen, Copenhagen*
10.00	L14	Fertility preserving efforts in oncology. *C. Rechnitzer, Copenhagen*
10.20	L15	Early diagnosis of liposarcoma progression by a 3-gene-signature. *A. Serguienko, Oslo*
10.30-11.00		*Coffee break*

11.00-12.30 **Session III: Lung metastasis and local therapy**
Room: Terminus Hall
Chairmen: *R. Haaverstad, Bergen, M. Sloth, Lund*

11.00 L16 Molecular imaging with PET in sarcoma.
M. E Revheim, Oslo

11.20 L17 Thoracic surgeon's perspectives.
P. Ryom, Copenhagen

11.50 L18 Radiotherapy and lung metastases.
J. Engellau, Lund

12.10 L19 CT-guided percutaneous radiofrequency ablation of pulmonary malignancies.
P. Kandiah, Bergen

12.30–13.30 *Lunch*

13.30–15.00 **Oral Poster-presentations (page 51-61)**
Room: Terminus Hall
Chairmen: *J. Engellau, Lund, J. Kanerva, Helsinki, O-J. Norum, Oslo*

15.00–15.30 *Coffee break*

15.30-16.45 **40 years of SSG – panel discussions**
Room: Terminus Hall
Chairmen: *O. Brosjö, Stockholm, O-J. Norum, Oslo*

- Surgery: H. Bauer, C. Trovik. J. Åhlén
- Oncology: K. Sundby Hall, M. Eriksson, P. Lindholm, A. Safwat
- Radiology: M. Sloth, I. Taksdal
- Pathology: B. Bjerkehagen, P. Rissler
- Nursing in SSG: L. Våde

18.45 Departure for social program (see separate section)

SSG Plenary Meeting

Friday 10 May, 2019

08.00–08.30		Registration Exhibition stand & Poster exhibition
08.30–15.00		**Joint session with physicians, scientists, nurses and physiotherapists** Room: Terminus Hall
08.30-10.25		**Session IV: Soft (ware) aspects in sarcoma care** Chairmen: *M. Eriksson, Lund, L. Våde, Oslo*
08.30	L20	Mentor program to re-socialize sarcoma patients. *L. Våde, Oslo*
08.50	L21	Mobile application for sarcoma patients and their relatives. *M. Karlsen Ødegaard, K. Brunvoll, Oslo*
09.00	L22	Oncology, advances in systemic therapy and inter-patient differences in treatment. *R. Jones, London*
09.30	L23	Novel design of clinical trials *M. Sydes, London*
10.00	L24	Perioperative care in cancer patients with special emphasis on correction of Anaemia. *I. Gögenur, Copenhagen*
10.30-11.00		*Coffee break*

11.00-15.00 **Session V: Abdominal sarcoma/gynaecological sarcoma**
Chairmen: *K. Øvrebø, Bergen, A. Safwat, Aarhus*

11.00 L25 Surgery in abdominal sarcoma: transatlantic collaboration.
C. Swallow, Toronto

11.30 L26 Personalized medicine in soft tissue pathology.
A. Lazar, Houston, Texas

12.00 L27 Uterine sarcomas - Current approaches and emerging therapeutic options.
A. G. Zahl Eriksson, Oslo

12.20–13.20 *Lunch*

13.20 L28 Radiology in abdominal sarcoma: The surgeon´s expectations.
A M. Wiedswang, Oslo

13.40 L29 Prognostic significance of peritoneal tumor penetration in gastrointestinal stromal tumor (GIST).
T. Hølmebakk, Oslo

13.50 L30 Surgical challenges in sarcomas of the torso.
A. Gronchi, Milan

14.20 L31 Oncological challenges in abdominal sarcoma.
S. Bauer, Essen

14.50-15.00 **Closing remarks and announcing of the winners of posters and free presentations.**
SSG chair and evaluation committee

SSG Nurses & physiotherapists

Wednesday 8 May, 2019

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12.00-14.00		Registration Exhibition stand & Poster exhibition (all day)
13.00-14.00		*Lunch buffet*

Joint session with physicians, scientists, nurses and physiotherapists

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14.10	L2	Bone sarcoma - Molecular biology/pathology. *H. K. Haugland, Bergen*
14.30	L3	Bone sarcoma - Surgery. *R. Grimer, Birmingham*
15.00	L4	Targeting the genomic chaos in osteosarcoma with PARP inhibitors. *O. Myklebost, Oslo*
15.10	L5	Bone sarcoma - Oncology. *Ø. Bruland, Oslo*
15.30-16.00		*Coffee break*

16.00	L6	Morphologically distinct cell population in patient-derived Ewing sarcoma cells display variation in drug sensitivity . *A. Papakonstantinou, Stockholm*
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16.40	L1	Bone sarcoma - Genetics and oncological therapy. *D. Thomas, Sydney*
18.30		Departure for social program (see separate section).

SSG Nurses

Thursday 9 May, 2019

07.45-08.20 Registration
Exhibition stand & Poster exhibition (all day)

08.20–12.15 **Nurses session**
Room: 707

08.20 Welcome!
L. Våde, Oslo

08.30-10.30 **Session I – Wound**
Chairmen: *H. Fuglø, Copenhagen, A. Pettersson, Umeå*

08.30 L32 Hyperbaric oxygen treatment for wounds in irradiated tissues.
G. Vaagbø, Bergen

09.15 L33 Regional hyperthermia in advanced solid tumors.
T. Nordberg, Bergen

09.45 L34 A patients wound narrative after removal of sarcoma.
L. Lernevall, Bergen

10.30–10.50 *Coffee break*

10.50-12.15 **Session II – Alternative treatment**
Chairmen: *H. Bøgseth, Oslo*

10.50 L35 Controlling pain with cannabis for medical use.
J. Thau, Copenhagen

11.10 L36 Mindfulness.
S. Bremerthun, Bergen

11.30	L37	Finding balance with Yoga and Ayurveda. *G. Foldnes, J. Winderl, Oslo*
11.50-12.15		Annual meeting
12.15-13.00		*Lunch*
13.00-15.15		**Joint session with physiotherapists** **Session III – Rehabilitation** Chairmen: *E. Frisk, Stockholm, L. Ingeman Petersen, Copenhagen*
13.00	L38	Rehabilitation at Haukeland University Hospital. *B. Dyngeland, Bergen*
13.20	L39	Communicating with seriously ill patients; how can we improve informing sarcoma patients of the complex and dangerous treatment we offer? *J. Engellau, Lund*
13.40	L40	General and sarcoma specific late effects in cancer patients. *C. Johansen, Copenhagen*
14.00	L41	An explanation of the variation in patient's adaption to rehabilitation? *T. Farmen Nerli, Stavern*
14.20	L42	Exercise oncology. *T. S. Nilsen, Oslo*
14.40	L43	Use of a simple form to facilitate communication on long-term consequences of treatment in sarcoma survivors. *L. Fauske, Oslo*

15.30-16.45 **40 years of SSG – panel discussions**
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- Surgery: H. Bauer, C. Trovik. J. Åhlén
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SSG Physiotherapists

Thursday 9 May, 2019

07.45-08.20 Registration
Exhibition stand & Poster exhibition (all day)

08.30–12.15 Physiotherapists session
Room: 711

08.30 Welcome!
M.L. Johansen, Oslo

08.45-10.30 Session I – Physiotherapists

08.45 L44 Physiotherapists´ clinical practice facing the newly amputated patient.
S. Ludvigsen, Oslo

09.30 L45 Three cases with osteosarcoma in children.
E. Frisk, Stockholm

10.00 L46 Psychosocial support to AYA with cancer.
H. Bøgseth, Oslo

10.30–10.50 Coffee break

10.50-12.15 Session II – Physiotherapists

10.50 L47 Creating an information app for sarcoma patients.
S. Ludvigsen, Oslo

11.30 L48 Evaluation of muscle strength, balance and gait function with patients with tumor prosthetics in the knee joint – a pilot study (MAGUS)
S. Nilsen, M. L. Johansen, Oslo

12.15–13.00 *Lunch*

13.00-15.15 **Joint session with nurses**
Session III – Rehabilitation
Chairmen: *E. Frisk, Stockholm, L. Ingeman Petersen, Copenhagen*

13.00 L38 Rehabilitation at Haukeland University Hospital.
B. Dyngeland, Bergen

13.20 L39 Communicating with seriously ill patients; how can we improve informing sarcoma patients of the complex and dangerous treatment we offer?
J. Engellau, Lund

13.40 L40 General and sarcoma specific late effects in cancer patients.
C. Johansen, Copenhagen

14.00 L41 A case report that illustrates the most important aspects in rehabilitation process.
T. Farmen Nerli, Stavern

14.20 L42 Exercise oncology.
T. S. Nilsen, Oslo

14.40 L43 Use of a simple form to facilitate communication on long-term consequences of treatment in sarcoma survivors.
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S. Bauer, Essen

14.50-15.00 **Closing remarks and announcing of the winners of posters and free presentations.**
SSG chair and evaluation committee

Lectures (L)

L1 Bone sarcoma - Genetics and oncological therapy.

David Tomas
Garvan Institute of Medical Research, Sydney, Australia

L2 Bone sarcoma - Molecular biology/pathology.

Hans Kristian Haugland
Haukeland University Hospital, Bergen, Norway

L3 Bone sarcoma - Surgery.

Robert Grimer
Birmingham, UK

40 years ago, when the SSG was formed, surgery for sarcomas was ‘haphazard’. It was carried out by a variety of different specialities and amputation was the most common procedure still for many bone sarcomas. The transformation that happened over the next few years was fueled by better imaging, effective chemotherapy, the advent of reliable implants and surgical ingenuity, combined with the centralisation of surgical specialisation.
Limb salvage is now the norm for most bone sarcomas but there are still many challenges ahead, some of them disease specific and some site specific.
Updated results will be presented from recent trials and consensus meetings in osteosarcoma, Ewing’s sarcoma, chondrosarcoma and chordoma. The long term results (30 years+) of limb salvage by endoprosthetic replacement will be used to show the success (and failings) of limb salvage and recent advances that will hopefully reduce surgical complications in the future, addressing particularly the challenges of pelvic sarcoma surgery and limb salvage in young children.

L4 Targeting the genomic chaos in osteosarcoma with PARP inhibitors.

Ola Myklebost[7,1,4], Eva W Stratford[1], Susanne Lorenz[1,2,4], Mona M Lindeberg[1], Else Munthe[1], Kjetil Boye[1,3], Olga Zaikova[3], Serena Nik-Zainal[5], Patrick S. Tarpey[5], Peter Campbell[5], Adrienne Flanagan[6], Stein Waagene[1], Heidi A K Bjørhovde[1], Aqsa Mahmood[1] and Leonardo A. Meza-Zepeda[1,2,4]
Affiliations: [1]Dept of Tumor Biology, [2]Genomics Core Facility, and the [3]Clinic for cancer, Oslo University Hospital, Norwegian Radium Hospital, Oslo, Norway, [4]Norwegian Cancer Genomics Consortium, Norway, [5]Wellcome Trust Sanger Inst., Hinxton, UK, [6]Royal National Orthopaedic Hospital, Middlesex, and UCL Cancer Inst., University College London, London, UK, [7]Dept. for Clinical Science, Faculty of Medicine, University of Bergen, Bergen, Norway.

Background: It has been reported that osteosarcomas have mutation profiles similar to BRCA-mutated breast and ovarian cancers, and that this might provide a sensitivity to PARP inhibitors.

Material and Methods: We have determined the efficacy of PARP inhibitor BMN 673 on a panel of osteosarcoma models. This includes proliferation and apoptosis assays performed using the live cell imaging as well as viability and colony formation assays on 12 osteosarcoma cell lines characterised for a number of possible biomarkers. Efficacy *in vivo* was also tested treating patient-derived OS xenografts in NOD/SCID mice.

Results and Discussion: Osteosarcoma cell lines respond variably to PARP inhibitors. We have performed a range of preclinical assays and ranked the 12 osteosarcoma cell lines based on the therapeutic responses *in vitro*, and confirmed efficacy to PARP inhibitor *in vivo*. The cell lines were investigated for a number of suggested biomarkers for response to PARPi, but none of these could reliably predict response *in vitro*.

Interpretation: PARP inhibitors shows potential for treatment of osteosarcoma, but responses vary. These findings suggest that clinical trials should be pursued, with a strong translational project to identify predictive biomarkers.

L5 Bone sarcoma - Oncology.

Øyvind S Bruland
Institute for Clinical Medicine, University of Oslo and Dept. of Oncology, Norwegian Radium Hospital – Oslo University Hospital, Oslo, Norway

The lecture will address selected challenges related to oncological management in five primary bone sarcomas (BS); i.e. osteosarcomas (OS), Ewing sarcomas (ES), chondrosarcomas (CS), chordomas (CD), as well as spindle-cell pleomorphic non-OS bone sarcomas (SCS). In the EUROCARE database BS are accounting for < 0.2% of malignant neoplasms [1]. Different BS has quite different clinical presentation and biological behavior. Patterns of incidence are so that none has no more than 0.3 incident cases reported per 100 000 per year [2]. Clinical Practice Guidelines for diagnosis, treatment and follow-up of BS have recently been published [3].

Challenges related to use of radiotherapy (RT) as the sole and/or postoperative local treatment modality will be presented for CS, CD and ES with the options for dose-escalation related to recent technological developments in conventional radiotherapy. Also particle therapies will briefly be mentioned, with focus on patients with BS located in the axial skeleton.

Challenges related to adjuvant chemotherapy (CT) cover those of bone-marrow ablative CT in poor-prognostic ES, poor histological responders among OS, as well as drug used and impact of CT in patients with SCS. In OS the challenge of synchronous versus the time-line for occurring metachronous metastases will be addressed.
For relapsing patients with OS the role of second line CT is not well defined.

Briefly, the lack of evidence for treatment effect exploring “druggable targets”, as well as experiences with selected immunotherapies in BS will be mentioned.

1. Stiller CA et al.: Descriptive epidemiology of sarcomas in Europe: report from the RARECARE project. Eur J Cancer 2013; 49:684–695.
2. Whelan J et al.: Incidence and survival of malignant bone sarcomas in England 1979-2007. Int J Cancer 2012; 131: E508–E517.
3. Casali PG et al.: Bone sarcomas: ESMO-PaedCan-EURACAN Clinical Practice Guidelines for diagnosis, treatment and follow-up. Ann Oncol. 2018 Oct ;29(Supplement_4) :iv79-iv95.

L6 Morphologically distinct cell populations in patient-derived Ewing sarcoma cells display variations in drug sensitivity.

Antroula Papakonstantinou[1], Christina Linder-Stragliotto[1], Carolina Brodin[2], Swapnil Potdar[3] and Bertha Brodin[2].

[1]Department of Oncology and Pathology, Karolinska Institutet, and Theme Cancer, Section for Breast Cancer, Endocrine Tumours and Sarcoma, Karolinska University Hospital, Stockholm, Sweden.

[2]Department of Microbiology Tumor and Cell Biology, MTC, Karolinska Institutet, Stockholm,

[3]Institute for Molecular Medicine, University of Helsinki, Finland

Background and purpose: Recurrent or primary metastatic Ewing sarcoma has a poor prognosis and, in the lack of effective agents, identifying new cancer pathways is of major importance patient-derived sarcoma cells (PDC) from patients referred at the Sarcoma Centre at Karolinska University Hospital are used for drug screens and gene panel sequencing as means for personalizing treatment. Hereby we report a case of a patient with treatment refractory Ewing sarcoma with morphologically different cell populations grown *ex-vivo* and distinct sensitivity to oncology drugs.

Methods: Ewing sarcoma cells were obtained through fine needle aspiration from a patient with recurrent tumour in the lumbar region. The cells were grown and expanded *in vitro* for approximately 30 days, hereby referred to as Ewing Sarcoma-Patient-Derived Cells (ES-PDC). The content of Ewing cells in the ES-PDC was examined by the expression of the EWS-FLI1 fusion protein using Proximity Ligation Assay (PLA). Drug sensitivity testing was performed using a library of 525 anti-cancer agents at 5 different concentrations. Drug sensitivity was evaluated using a customized program and expressed as drug sensitivity score (DSS). Sequencing of a custom panel of 16 sarcoma-associated genes was performed using the Haloplex target enrichment system and Next Seq (Illumina). Expression of 180 cancer genes was evaluated using Q-RT-PCR arrays.

Results: ES-PDCs grew in 2 morphologically distinct patterns; 1 population as spheroids (sES-PDC) and 1 as adherent cells (aES-PDC). Both populations expressed the EWS-FLI1 protein in nearly all cells. Drug sensitivity to anti-cancer agents was similar between the 2 populations to 80%, however distinct

for 20% of the agents. A closer analysis of the latter group of agents showed that spheroid growing ES-PDC were more sensitive to differentiating drugs and epigenetic modifiers than ES-PDCs growing as adherent cells. The expression of cancer-driver genes was also different within both populations.

Interpretation: Our study supports previous observations of intratumour heterogeneity in ES, and shows that distinct ES cell populations derived from patient biopsies can be characterized both genetically and in drug screens to provide information of potential vulnerabilities that can be exploited to improve the treatment of patient with recurrent ES.

L7 Particle radiotherapy for bone sarcoma.

Jon Espen Dale
Dep. of Oncology and Medical Physics, Haukeland University Hospital, Bergen, Norway

At present there is no high-quality clinical evidence proving that proton radiotherapy (PRT) is superior to modern photon RT in any cancer or disease site. However, PRT offer superior dose conformity [1], which probably will lead to a reduction in acute and long term side effects, including secondary malignancies [2]. As a consequence, PRT should be considered for most pediatric patients requiring RT.
For adults, PRT is safe and effective for bone sarcomas of the skull base [3, 4] and spine [5], sites in which the tumor's close proximity to vital organs often prevent both adequate surgery and sufficient dose when using photon RT. For radioresistant sarcomas, RT using carbon ions show promising results.
In many situations the obvious dosimetric benefit of PRT would make it unethical to perform traditional RCTs. Therefore, a new approach is to perform comparative planning, and subsequently select patients to PRT rather than photon RT only if a significant reduction in risk of relapse or toxicity is predicted. Rigorous collection of prospective outcome data and long-term follow-up will be mandatory to better define the indications for PRT.
In the setting of postoperative RT for spine sarcomas, traditional titanium fixation devices induce artifacts on MRI and CT images, which hamper delineation of RT target volumes, induce significant uncertainties in the dose distribution, and may prevent early detection of tumor relapse. Carbon fiber reinforced polyetherehterketone (CK/PEEK) fixation devices should therefore

be considered for patients where the need for postoperative RT is likely.

1. Palm, A. and K.A. Johansson, *A review of the impact of photon and proton external beam radiotherapy treatment modalities on the dose distribution in field and out-of-field; implications for the long-term morbidity of cancer survivors.* Acta Oncol, 2007. **46**(4): p. 462-73.
2. Eaton, B.R., et al., *Secondary Malignancy Risk Following Proton Radiation Therapy.* Front Oncol, 2015. **5**: p. 261.
3. Fossati, P., et al., *Review of photon and proton radiotherapy for skull base tumours.* Rep Pract Oncol Radiother, 2016. **21**(4): p. 336-55.
4. Mercado, C.E., et al., *Proton therapy for skull base tumors: A review of clinical outcomes for chordomas and chondrosarcomas.* Head Neck, 2019. **41**(2): p. 536-541.
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L8 Off-label use of pazopanib for patients with metastatic bone sarcoma.

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Background and purpose: Many patients develop distant failure after primary curative treatment of localized bone sarcomas. The effect of chemotherapy in metastatic bone sarcomas is poor and the condition is invariably fatal. New treatment modalities are therefore badly needed. Pazopanib is a selective multi-targeted tyrosine kinase inhibitor that has proved to be effective in the treatment of metastatic soft tissue sarcomas. The objective of this study was to evaluate the off-label use of pazopanib in patients with metastatic bone sarcomas who failed standard chemotherapy.

Patients and methods: All patients with metastatic bone sarcomas treated with pazopanib between October 1st 2011 and October 1st 2017 at the Department of Oncology, Aarhus University Hospital, were evaluated. Demographics, treatment and survival outcomes were collected and analyzed.

Results: 19 patients were identified. The median age was 38 years (range 18-62). Half of the patients were diagnosed with osteosarcoma. All patients had documented disease progression at the time of starting pazopanib. The median overall survival (OS) was 11 months. Median progression free survival (PFS) was 5.4 months. 13 out of 19 patients had either partial response or stable disease. In 5 patients the dose of pazopanib was reduced due to toxicity.

Interpretation: Off-label use of pazopanib is effective in the treatment of metastatic bone sarcomas of different histology. Pazopanib was well tolerated in the treatment of patients with refractory bone sarcomas. Studies examining the effect of pazopanib alone or in combination with chemotherapy or other targeted therapies are needed.

L9 Monitoring disease evolution by liquid biopsies.

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Background Molecular analysis of circulating tumor DNA (ctDNA) has a large potential for clinical application by capturing mutations and heterogeneity through non-invasive sampling. The CircSarc studies aims to provide new insights into the clinical utility of liquid biopsies in soft tissue sarcomas (CircSarc STS) and gastrointestinal stromal tumours (CircSarc GIST).

Methods Plasma have been collected longitudinally for a cohort of 35 localized, high-grade STS and 30 metastatic GISTs and at time of diagnosis for 50 GISTs. Mutations in ctDNA are identified using ultra-low-pass WGS, targeted NGS or ultrasensitive-PCR[1]. The levels of somatic mutations in ctDNA are correlated with clinopathological features.

Results At time of surgery and/or progression, we detect ctDNA in 30% of the first STS samples with higher levels in larger or metastatic tumours. Longitudinal plasma samples are analyzed with more sensitive assays. In a case study, the ctDNA levels correlated with clinical manifestation of metastatic disease[2]. ctDNA were detected in 36% of treatment-naïve GIST patients including all patients with metastatic disease. Tumor burden was the most important detection determinant. Analysis of metastatic GISTs are ongoing, but repeated plasma samples in a progressing patient have demonstrated that clonal evolution can be monitored over time. ctDNA analysis of the patient revealed multiple resistance mutations, and these were spatially distributed in the primary

tumour[3].

Interpretation Detection of mutations in plasma is particularly feasible for patients with high tumor burden. Mutational analysis by use of liquid biopsies can capture the molecular heterogeneity of the tumour and guide treatment decisions during progression.

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L10 STS - Radiology, strategy for optimal biopsy.

Oskar Angenete
University Hospital Trondheim, Norway

L11 STS - Molecular biology and pathology, strategy for optimal biopsy and diagnosis.

Florian Puls
Sahlgrenska University Hospital, Gothenburg, Sweden

L12 Treatment of low-grade fibromyxoid sarcoma: Systematic review and experiance of a teriary cancer center.

Bodil Engelmann[1,2], Florence Chamberlain[1], Omar Al-Muderis[1], Christina Messiou[1], Khin Thway[1], Aisha Miah[1], Shane Zaidi[1], Charlotte Benson[1], Spyridon Gennatas[1], Robin L Jones[1]

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Background and purpose: Low-grade fibromyxoid sarcoma (LGFMS) is a rare sarcoma subtype with a generally indolent pattern of clinical behavior. Complete surgical resection with or without radiation is the mainstay of management for localised disease but treatments for advanced disease are limited. In this study we evaluated the outcome of patients with LGFMS and the efficacy and safety of non-surgical therapies.

Patients and methods: A literature search was performed as well as a retrospective review of a prospectively maintained database of patients treated at our institution from 1994-2018.

Results: 102 patients were identified. 94/102 (92%) had primary resection. Of these, 13/94 (14%) required re-excision for recurrence and 73/94 (78%) were treated with surgery alone. 7/102 (7%) were treated with palliative systemic therapy and 23/102 (23%) with radiotherapy. Median time from presentation to death or last follow up was as follows: surgical excision alone (n = 73) (median 52 months, interquartile range (IQR) 16 – 76 months), surgery and radiotherapy (n = 15) (median 35 months, IQR 21 – 46 months) and surgery and systemic therapy (n = 6) (median 33 months, IQR 9 – 112 months) respectively. For patients treated with best supportive care (n = 8) median time from presentation to death was 35 months (IQR 22 – 44 months).

Interpretation: Our data support the current opinion that neither chemotherapy nor radiotherapy improves survival in advanced LGFMS but surgical resection is effective for localised LGFMS. Further studies are required to determine if targeted treatments can play a role in management.

L13 STS - Surgery.

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Surgery is still the primary treatment for patients with soft tissue sarcomas (STS), and for tumors of the extremities, limb-sparing surgery have for the past 40-50 years been the preferred approach used in most cases. In bone sarcoma surgery a number of specific improvements have been introduced within the last 1-2 decades such as e.g. better and more durable tumor implant systems, custom-made implants, intraoperative CT and navigation, and patient-specific cutting guides. STS surgery (as bone sarcoma surgery) has benefitted from the significant improvement in the various newer scanning techniques allowing a more precise planning of surgery, but the STS surgery itself is basically still performed using the techniques and principles described by Enneking in the 1980ies. In most cases these principles are sufficient and together with postoperative radiotherapy, in especially deep-seated high-malignant tumors, it is a good treatment. However, on a regular basis we also see challenging cases

where we feel, we have to do something extra, to avoid severely mutilating surgery or amputation. The possibilities are many such as e.g. preoperative chemotherapy, preoperative radiation therapy, isolated limb perfusion, or brachytherapy, but the knowledge of these techniques are mainly located outside the orthopedic field. Challenging STS cases will be presented, and treatment strategy discussed.

L14 Fertility preserving efforts in oncology.

Catherine Rechnitzer
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L15 Early diagnosis of liposarcoma progression by a 3-gene-signature.

Anastassia Serguienko[1], Peder Braadland[1], Susanne Lorenz[1], Bodil Bjerkehagen[2], Ola Myklebost[1, 3]
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Background and purpose: Well differentiated liposarcoma (WDLPS) is a low-grade malignancy with a favorable prognosis which may dedifferentiate to DDLPS, an often high-grade aggressive counterpart. Currently the differential diagnosis to distinguish between these 2 forms is based on histological examination. Specific molecular alterations that can be used to distinguish between WDLPS and DDLPS subtypes have not been identified so far. Here we have tested a metabolic gene signature as a biomarker for the differential diagnosis between WD- and DDLPS, as well as for its ability to predict malignant evolution towards the DD form.

Patients and methods: We analyzed by RT-qPCR 8 atypical lipomatous tumors (ALT), 9 WDLPS and 21 DDLPS cases for the expression of several metabolic genes involved in general and adipose tissue-specific cellular metabolism. Tumor material was collected after surgery from patients that entered the clinic between 2014 and 2017.

Results: We identified a 3-gene signature based on *LIPE*, *PNPLA2* and *PLIN1*, which revealed the presence of DDLPS with 100% sensitivity and 90% specificity, even in specimens from the WD component of DDLPS.

Interpretation: The identified metabolic signature allows highly accurate differential diagnosis between WD- and DDLPS even in samples containing lipid droplets, a marker of differentiation, which makes it very suitable also for the use on biopsies. Importantly, our results support the recent observation that deletion of *PNPLA2* is a novel factor in liposarcoma pathogenesis.

L16 Molecular imaging with PET in sarcoma.

Mona E Revheim
Department of Radiology and Nuclear Medicin, Oslo University Hospital, Norway.

L17 STS metastasis - Thoracic surgeon´s perspectives.

Philip Ryom
Department of Thoracic Surgery, Rigshospital Copenhagen, Denmark.

L18 Radiotherapy and lung metastases.

Jacob Engellau
Department and Institution of Oncology, Lund University and Skane University Hospital, Lund, Sweden

L19 CT-guided percutaneous radiofrequency ablation of pulmonary malignancies.

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Radiofrequency ablation (RFA) is a minimally invasive CT –guided technique that has been used for the treatment of primary and secondary pulmonary malignancies since 2000. RFA, the most commonly used technique, has an 80-90% highest reported rate of complete ablation, with the best results obtained in tumors less than 2-3 cm. Tumor size, the stage of the disease, tumor localization and underlying comorbidities are the main predictors of survival. Microwave ablation is another option that may help overcome the limitations of RFA in the future.

We have performed more than 80 RFA treatments in our hospital and the results seem promising with no severe complications and low rate of tumor recurrence. RFA is a safe and effective method and could be beneficial for the improvement of treatment effect of inoperable pulmonary malignancies.
This lecture will address how to select suitable candidates for RFA, how to perform this CT –guided technique and how to manage possible complications.

L20 Mentor program to re-socialize sarcoma patients.

Lotta Våde
Oslo University Hospital, Oslo, Norway

Background and purpose: Research agrees that many cancer patients experience long term side effects after finished treatment. A mentorship program for sarcoma patients wants to find out if this program can help patients with their daily life after finished treatment.

Methods:16 patients have been included in 2 programs. Private companies participate by providing a personal mentor and 4000 Euro. The program lasts for 8 months.
Part 1: All the participants get a mentor from a private company. The mentors contribute with their ideas, experience and knowledge.
Part 2: All participants in the group should participate in 3 daily meetings. The program has a supervisor who will support both participants and mentors' during the program.
Part 3: All participants and mentors travel to Nepal. This travel will give the participants an increased reflection and understanding of themselves.
7 mentors and mentees are interviewed after they have finished the first pilot program.

Results and interpretation:7 patients have completed the program and 9 patients will finish the program in May 2019. Several diaries from patients and regular conversations with the mentors, indicate that the program is useful for both patients and mentors. This program is under evaluation and the results will give more information if this program is useful to help re-socialize after treatment.

L21 Mobile application for sarcoma patients and their relatives.

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Background and purpose: Patient information about sarcoma, sarcoma treatment and the time after treatment are often given as loose sheets. This information is not covering all patient needs. In a survey done with members of the patient-organization Sarkomer, 52 % of the members answered that they did not receive enough information. 82 % of the members said yes - they desire a mobile application with all information in one place. All sarcoma patients and their relatives will receive correct information at the right time point.

Methods: Complete a mobile application where you will receive information about sarcoma and sarcoma treatment according to the phase you are in - before treatment, during treatment and after treatment. Sarkomer is responsible for the mobile application, and health care professionals at Oslo University Hospital are responsible for the professional contents.

Results: A mobile application will be released during 2019.

Interpretation: Correct and enough information will make patients and their relatives feel safer and they will master their everyday life better.

L22 Oncology, advances in systemic therapy and inter-patient differences in treatment.

Robin Jones
The Royal Marsden NHS Foundation Trust, London, UK

L23 Novel design of clinical trials.

Matthew Sydes
MRC Clinical Trial Unit at UCL, London, UK

L24 Perioperative care in cancer patients with special emphasis on correction of Anaemia.

Ismail Gögenur
Department of Clinical Medicine, University of Copenhagen, Copenhagen, Denmark

L25 Surgery in abdominal sarcoma: transatlantic collaboration.

Carol Swallow
University of Toronto, Canada.

L26 Personalized medicine in soft tissue pathology.

Alexander Lazar
Department of Pathology, Division of Pathology and Laboratory Medicine, MD Anderson Cancer Center, Houston, Texas, USA

L27 Uterine sarcomas - Current apporaches and emerging therapueutic options.

Ane Gerda Zahl Eriksson
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Uterine sarcomas represent about 8% of uterine malignancies, with uterine leiomyosarcoma (uLMS) being the most common subtype. Uterine sarcomas carry a poor prognosis when compared to endometrial carcinoma. For women with uterine confined disease, hysterectomy is recommended. Routine lymphadenectomy is not indicated as the risk of nodal metastasis is low. However, bulky nodes should be removed. Removal of the adnexae is thought to be reasonable in peri- and post-menopausal women, and perhaps in premenopausal women since estrogen and progesterone receptors have been reported to be positive in 40-70% of cases with uLMS; however there are no data to show that oophorectomy improves survival. No adjuvant intervention has been shown to improve PFS or OS in uterine-confined LMS.
In the recurrent or metastatic setting, surgery should be considered if the disease is potentially completely resectable. There is no prospective data to guide post-resection management. Options are observation vs radiotherapy vs chemotherapy vs hormonal blockade.

For women with multisite metastatic disease not thought to be resectable there is no role for surgery. These women should receive systemic therapy to alleviate symptoms. Enrolment into clinical trials is encouraged.
Comprehensive genomic profiling can identify therapeutic targets and provide insight into the biology of uLMS. Targeted agents have shown some benefit in women with uLMS. PD-1 and PD-L1 expression is reported in uLMS and early data has shown some activity of immune checkpoints inhibitors.

L28 Radiology in abdominal sarcoma: the surgeon´s expectations.

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Soft tissue sarcoma in the abdomen and pelvis is a heterogenous group of tumors. Many of them are large masses at the time of diagnosis because they are usually clinically silent until they invade or compress vital organs. Soft tissue sarcomas located in the abdominal or trunk wall come to clinical attention earlier.
For some abdominal soft tissue sarcomas the radiological pattern is typical, notably tumors containing detectable fat and some abdominal wall tumors. For diagnosing GIST it is essential to show origin from the GI-tractus. For many other diagnoses the radiological pattern is indeterminate.
For the surgeon the tumor is always seen in a clinical setting with the patient´s age and history.
Is the tumor is found incidentally or due to symptoms? The surgeon wants to know the site / organ of origin of the tumor and if the radiology is typical for a sarcoma. It is important whether the lesion in question could be a tumor where oncological treatment is indicated (e.g. lymphoma) or where surgery should be avoided if possible for other reasons (e.g. fibromatosis).

Anatomical details are essential to evaluate if the tumor is resectable with an adequate margin and the extent of the surgery needed (e.g. multivisceral resection). Radiological signs of emergency must also be reported.

The emphasis of my lecture will be on showing radiological examples of abdominal sarcoma and differential diagnoses. Close cooperation between radiologist, surgeon, pathologist and oncologist is mandatory for a good outcome for these patients.

L29 Prognostic significance of peritoneal tumour penetration in gastrointestinal stroma tumour (GIST).

Toto Hølmebakk[1], Bodil Bjerkehagen[2], Ingvild Lobmaier[2], Ivar Hompland[3], Stephan Stoldt[1] and Kjetil Boye [3]
Departments of [1]Abdominal Surgery, [2]Pathology and [3]Oncology, Oslo University Hospital, The Norwegian Radium Hospital, Oslo, Norway

Background and purpose: Peritoneal penetration (PP) strongly affects prognosis in gastrointestinal carcinomas. In gastrointestinal stromal tumour (GIST), its significance in the absence of tumour rupture has not been analysed in detail.

Methods: Patients undergoing complete surgery for non-metastatic GIST from 2000 to 2017 were identified in the regional sarcoma database at Oslo University Hospital. Patients with extraperitoneal (oesophagus, rectum) or ruptured tumours were excluded. Rupture was defined according to the Oslo criteria, which also include transperitoneal infiltration into an adjacent organ. PP was assessed on routine histopathological examination by sarcoma pathologists.

Results: PP was recorded in 82 (24 %) of 341 patients. There were 32 recurrences, 23 in patients without PP, 9 in patients with PP. Despite statistically significant associations between PP and established risk factors (size, mitotic index, non-gastric location), 5-year recurrence-free survival rate was similar in patients with and without PP, 86 % and 90 %, respectively (hazard ratio 1.25, 95% CI 0.58 to 2.70; p= 0.6). Of 97 patients in the high-risk category, 53 received adjuvant imatinib. There was no statistically significant difference in recurrence rates between PP positive and PP negative patients in either group.

Interpretation: PP in the absence of tumour rupture does not influence prognosis in GIST. This lack of prognostic significance may reflect unexplored differences between epithelial and mesenchymal malignancies.

Table 1. Peritoneal tumour penetration related to clinical and pathological characteristics				
	Total n=341	No penetration n=259 (%)	Penetration n=82 (%)	*P* value
Age ≥ median	178	136 (76)	42 (24)	0.838
Age < median	163	123 (75)	40 (25)	
Males	168	129 (77)	39 (23)	0.723
Females	173	130 (75)	43 (25)	
Gastric tumour location	254	201 (79)	53 (21)	0.019
Non-gastric tumour location	87	58 (67)	29 (33)	
*Tumour size (cm)				0.008
≤5.0	193	155 (80)	38 (20)	
5.1-10.0	113	84 (74)	29 (26)	
>10.0	34	19 (56)	15 (44)	
Mitoses per 50 HPF†				0.030
0-5	269	213 (79)	56 (21)	
6-10	29	22 (76)	7 (24)	
>10	34	20 (59)	14 (41)	
‡Modified NIH classification				<0.001
Very low risk	26	21 (81)	5 (19)	
Low risk	143	117 (82)	26 (18)	
Intermediate risk	74	62 (84)	12 (16)	
High risk	97	59 (61)	38 (39)	
Adjuvant imatinib				0.004
No	288	227 (79)	61 (21)	
Yes	53	32 (60)	21 (40)	
Neoadjuvant imatinib				0.082
No	333	255 (77)	78 (23)	
Yes	8	4 (50)	4 (50)	

*One patient with indeterminate tumour size excluded; †nine patients with unspecified mitotic index excluded; ‡one patient at unspecified risk of recurrence excluded. HPF, high-power fields of the microscope; NIH, National Institutes of Health.

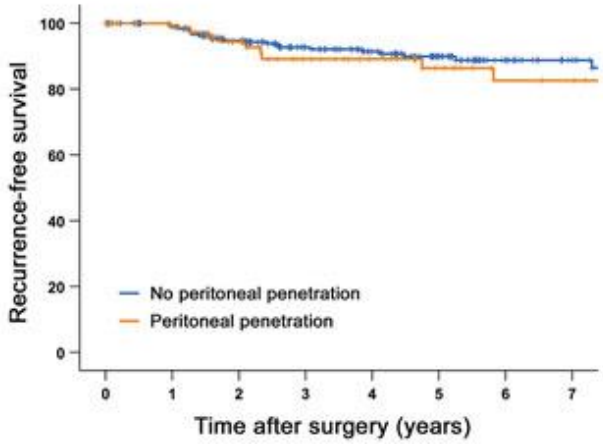

Figure 1. Estimated recurrence-free survival in patients with primary abdominal gastrointestinal stromal tumours without rupture.

L30 Surgical challenges in sarcomas of the torso.

Alessandro Gronchi
Department of Surgery, Fondazione IRCCS Istituto Nazionale dei Tumori, Milan, Italy.

L31 Oncological challenges in abdominal sarcoma.

Sebastian Bauer
West German Cancer Center, Medical Oncology, Essen, Germany

L32 Hyperbaric oxygen treatment (HBOT) for chronic wounds in irradiated skin.

G. Vaagbø
Head of Norwegian national centre for planned hyperbaric oxygen treatment,
Haukeland University Hospital

Chronic radiation injury persists/develops more than ten weeks post radiotherapy. It is characterized by obliterating endarteritis in the small nutrient vessels resulting in chronic hypoxia and malfunction of the skin. There are usually areas of hypo- and hyperpigmentation and hyperkeratosis. The skin is hairless and dry with epidermal scaling, atrophy, telangiectasia, subcutaneous fibrosis and in some occasions: necrosis. The condition is slowly progressive. Risk factors for developing wounds in irradiated tissues are: radiation dose, concomitant disease, tobacco smoking and if there are areas of sensitive skin involved. There might be development of carcinoma in irradiated skin; usually more than ten years post radiation. Skin with chronic radiation injury should be treated with skin conditioner and one should treat neuropathic pain according to guidelines.
If there is **ulceration of skin with chronic radiation injury** there is need of optimal wound bed preparation and extremely careful topical revision. Take a biopsy to exclude cancer and investigate whether the patient might be eligible for HBOT. If you are not familiar with HBOT, you should call the nearest HBOT centre to discuss the patient directly with the diving physician.
If you **plan to perform surgery in irradiated tissue**, you should consider HBOT pre- and post-surgery. This will require HBOT for at least 6 weeks, then waiting another 6-8 weeks for neoangiogenesis to occur before performing surgery. "Directly" (starting within a week) after surgery there should be 2 weeks of HBOT to further support the healing process. Timing of HBOT and the surgery should be planned in advance and in collaboration with the HBOT-team. The chronic wound must be followed by a specialist in treating this kind of wounds during HBOT.

HBOT means breathing 100 % oxygen while increasing the ambient pressure to at least two atmospheres. This will lead to a dramatic raise in the amount of oxygen dissolved in plasma and hyperoxygenation of the whole body. Mechanisms of action in wound healing are delivery of fuel and ammunition to the leucocytes, re-establishing oxygen-demanding healing processes and neoangiogenesis. These processes are time consuming and neoangiogenesis will take 12 – 14 weeks. The HBOT is performed once daily for 30 to 40 days in an

ambient pressure of 2.4 to 2.5 atmospheres (1.4 – 1.5 bar overpressure) which is the same ambient pressure you experience when diving to 14 - 15 meters of sea water. There are two kinds of pressure chambers: multiplace chambers where the patients are seated (or in bed) when pressurized with air and the patients breathe oxygen through a mask or a ventilator. Monoplace chambers are pressurized with oxygen and the patients are in a semi supine position breathing chamber atmosphere. There is extreme danger of fire which requires an abundancy of fire reducing precautions. It is for example prohibited to bring devices containing batteries into the chamber as they may explode. This means that in wounds treated with negative pressure, the tubes must be clamped, and the pump removed during treatment.

Side effects from elevated ambient pressure are frequent: barotraumas of ears, sinuses and lungs (rare). If the patient has air trapped between a tooth and a dental filling, the dental filling might fall out when the hyperbaric chamber is decompressed. Side effects from hyperoxygenation are visual impairments (transeint), reduction in small airways conductance (3% reduction in FEV1), fatigue (transient) and oxygen induced seizures (rare). Other side effects might be increased heart failure as HBOT will lead to increased preload and afterload of the heart. Claustrophobia might be triggered.

Contraindications to HBOT are: COPD Gould 3 or worse, major heart failure, bullae visible on thoracic x-ray, hypoventilated lung tissue (pneumonia), pneumothorax, pregnancy, ongoing treatment with Disulfiram, treatment history of Bleomycine, active cancer.

HBOT in Scandinavia: The Norwegian National Treatment Unit for Planned HBOT in Bergen is the largest HBOT-centre in Scandinavia. The other two hyperbaric units in Norway will provide emergency treatment only (decompression injuries, necrotizing soft tissue infections, gas embolisms, CO intoxication etc.). In Sweden and Denmark the HBOT centres treating the largest amount of planned patients are the ones in Gothenburg and Copenhagen.

Norway:
Haukeland universitetssykehus www.helse-bergen.no/trykkammer
Oslo universitetssykehus Ullevål https://oslo-universitetssykehus.no/avdelinger/akuttklinikken/avdeling-for-anestesiologi/hyperbarmedisinsk-enhet
Universitetssykehuset Nord-Norge Tromsø https://unn.no/steder/unn-tromso

Sweden: https://lakemedelsboken.se/kapitel/lakemedelsanvandning/behandling_med_hyperbar_oxygen_-hbo-.html#v1_2

Karolinska sjukhuset: https://www.karolinska.se/for-patienter/alla-behandlingar-och-undersokningar-a-o/anopiva-solna/HBOT/
Sahlgrenska sjukhuset https://www.sahlgrenska.se/omraden/omrade-2/anestesi-operation-iva/enheter/tryckkammare1/
Blekingesjukhuset https://regionblekinge.se/om-webb-platsen/sok.html?query=Tryckkammarenheten
Uddevalla sjukhus https://www.nusjukvarden.se/avdelningar-och-mottagningar/tryckkammarmottagning/
Helsingborgs lasarett https://vard.skane.se/helsingborgs-lasarett/undersokningar-och-behandlingar/tryckkammarbehandling/?highlight=tryckkammare

Denmark:
Rigshospitalet https://www.rigshospitalet.dk/trykkammer?rhKeywords=trykkammer
Aarhus Universitetshospital http://www.auh.dk/om-auh/afdelinger/bedoevelese-og-operation/pjecer-og-vejledninger/tryktanken/om-tryktanken/
Odense Universitetshospital
http://www.ouh.dk/system/search.asp#?cludoquery=trykkammer&cludopage=1&cludorefurl=http%3A%2F%2Fwww.ouh.dk%2Fwm252588&cludorefpt=Odense%20Universitetshospital

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L33 Regional hyperthermia in advanced solid tumors.

T. Nordberg
Department of Oncology, Haukeland University Hospital, Bergen, Norway.

L34 A patients wound narrative after removal of sarcoma.

L. Lernevall
Department of Plastic Surgery, Haukeland University Hospital, Bergen, Norway.

L35 Controlling pain with cannabis for medical use.

J. Thau
Rigshospitalet Copenhagen, Denmark.

L36 Mindfulness.

S. Bremerthur
Haukeland University Hospital, Bergen, Norway.

L37 Finding balance with Yoga and Ayurveda.

G. Foldnes, J. Winderl
Oslo University Hospital, Oslo, Norway.

L38 Rehabilitation at Haukeland University Hospital.

B. Dyngeland
Haukeland University Hospital, Bergen, Norway.

L39 Communicating with seriously ill patients; how can we improve informing sarcoma patients of the complex and dangerous treatment we offer?

Jacob Engellau
Department of Oncology, Skane University Hospital, Lund, Sweden.

L40 Doctors point of view.

Christoffer Johansen
CASTLE - Cancer Late Effect Research Unit, Oncology Clinic, Rigshospitalet, Copenhagen, Denmark..

The number of cancer survivors are exponentially increasing. In Scandinavia having a population of 22 million people this presents a problem as survivors of many cancers are facing late effects due to their treatment. The knowledge concerning these late effects is sparse and develop concurrently with the increasing survival expectations. However, there is also a constant introduction of new treatment principles and algorithms changing the landscape of late effects. This presentation will look into the pattern of late effects in cancer survivors across cancer types and discuss how it may be possible to prevent, early detect, limit the severity of or treat some common late effects in cancer survivors. The perspective touching on the increase in multimorbidity, polypharmacy and also socioeconomic inequality in both incidence and survival of cancer. The health professionals may consider to rethink the treatment models taking into account a more prehabilitating approach aiming at optimizing treatment outcome, quality of life and reducing the burden of late effects.

L41 An explanation of the variation in patient´s adaption to rehabilitation?

Tom Farmen Nerli
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The International Classification of Functioning, Disability and Health is the main framework we use in rehabilitation. Medical rehabilitation aims to close the gap towards “normalization” after an injury or disease. The focus has usually been on “body functions and structure,” activity and participation. Unfortunately, there has been less focus on contextual factors such as environmental factors and personal factors. I will illustrate the importance of contextual factors in rehabilitation by using patient examples with sarcoma. I will also talk briefly about Cognitive Behavioral Therapy (CBT) as an intervention for strengthening personal factors.

L42 Exercise oncology.

Tormod S. Nilsen,
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The field of exercise research in oncological settings has evolved tremendously over the past 30 years or so. From a general recommendation of bed rest up until the 90ies, to where we are now where systematic reviews generally report exercise as safe across the cancer continuum. Here, we will discuss the status of the current literature on exercise oncology, and identify some crucial limitations. Furthermore, we will point at some of the challenges in prescribing exercise to cancer survivors.

L43 Use of a simple form to facilitate communication on long-term consequences of treatment in sarcoma survivors.

Lena Fauske[1,2], Ivar Hompland[1], Geir Fagerjord Lorem,[3] Øyvind S. Bruland[1,4]
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Background and purpose: To report on our experience using a simple and optional form to facilitate communication between the patients and the oncologists during outpatient follow-up to detail the spectrum of challenges reported by sarcoma long-term survivors.

Methods: The form included topics related to late effects and unmet needs, such as fatigue, pain, mobility challenges, sexual problems and psychological issues. Of the 265 patients that received the form, 236 (89%) returned it. Semi-structured interviewed were conducted in 28 selected patients about their experiences with the form and analyzed by use of inductive thematic analysis.

Preliminary results: 207 (88%) patients had late-effect topics they wanted to discuss with their oncologist. Average time since diagnosis was 5.6 years. Fatigue was raised by 42% of the patients, pain by 27%, impaired mobility by 21%, sexual problems by 14% and psychological issues by 14%. Patients who had undergone multimodal treatment with chemotherapy raised fatigue more frequently than those who had only undergone surgery, radiotherapy or both.

The main findings of the interviews showed that the majority of the patients were satisfied with the oncological follow-up, but they requested more structured focus on the entire spectrum of psychosocial challenges. All 28 expressed that use of the form as an integrated tool during follow-up was justified and helpful.

Interpretation: A simple communication form regarding late effects could increase the attention to late effects during outpatient follow-up.

L44 Physiotherapists 'clinical practice facing the newly amputated patient.

Signe Ludvigsen
OUS Radiumhospitalet, Oslo

Patients go through amputation surgery throughout the country, and physiotherapists are involved in the treatment of these patients. However, we do not know enough about what the physiotherapist's work consists of, and what affects the choices and assessments the physiotherapists make facing the newly amputated patient. What are common features and differences in the physiotherapy treatment of newly amputated patients in Norway?

L45 Three cases with osteosarcoma in children.

Emelie Frisk
Astrid Lindgrens Barnsjukhus, Stockholm,

Different perspectives of physiotherapy; what can we expect?
What is the goal?
What do we as physiotherapists focus on?

L46 Psychosocial support to AYA with cancer.

Hilde Bøgseth
OUS Radiumhospitalet, Oslo

Adolescent and young adult (AYA) cancer patients face a unique set of challenges. They are often in a phase of life where there are a lot of changes and they deserve healthcare professionals who can address these issues.
It can be hard for teens and young adults with cancer to manage the emotional and social challenge that comes in to their lives. Therefore psychosocial support is very important. So, what do AYA`s want from us? What do they need? How can we help them in best possible way?

L47 Young and ill, but above all young.

Signe Ludvigsen
OUS Radiumhospitalet, Oslo

Sarcoma is more than just an illness. It can turn your life upside down and result in many concerns and questions, often concerning life and death. These questions may occur at any stage of the treatment and last for months, even years after the treatment has ended.
Patients going through treatment for sarcoma are in need of information, at every stage of the journey. However, it can be easy to get lost in the jungle of information out there. Thus the motivation for creating a personalized information app for sarcoma patients emerged.
Where are we now, what remains to be done and what have we learned from the process?

L48 Evaluation of muscle strength, balance and gait function with patients with tumor prosthetics in the knee joint – a pilot study (MAGUS).

Tormod S. Nilsen, Merethe Lia Johansen
Norges Idrettshøgskole and OUS Radiumhospitalet, Oslo, Norway

Due to the comprehensive nature of the surgical procedures, clinical experience shows that patients with knee tumor prostheses, experience impaired gait, and muscle strength. The scientific literature in this area is, however, limited, which obstructs evidence-based rehabilitation interventions. Therefore, we are

currently conducting a pilot study comparing gait function, one-legged sway (balance), as well as maximal voluntary force production in knee- and ankle joint movements, between patients with knee tumor prostheses and age- and sex-matched controls. We aim to identify differences and, more importantly, to identify possibilities for rehabilitation interventions. In this session, we will discuss the advantages of multidisciplinary collaborations in this field, present some preliminary observations, and briefly discuss future plans.

Posters (P)

P1 Targeting FRS2-amplified liposarcoma with FGFR inhibitors.

Ola Myklebost [1,2,7], Robert Hanes [1,2], Else Munthe [1], Iwona Grad [1], Jianhua Han [3], Ida Karlsen [3,4], Emmet McCormack [3,5], Eva Wessel Stratford [1], and Leonardo A. Meza-Zepeda [1,2,6]

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Background: FGFR inhibition has been proposed as treatment for dedifferentiated liposarcoma (DDLPS) with amplification of *FRS2*, coding for a signaling adaptor protein downstream of FGFR, but we previously could only demonstrate transient cytostatic effects when using the NVP-BGJ398 inhibitor.

Methods: The activity of the more potent FGFR inhibitor LY2874455 was investigated in 3 DDLPS cell lines and 1 xenograft by measuring effects on cell growth and apoptosis *in vitro* and also testing efficacy *in vivo*. Genome, transcriptome and protein analyses were performed to characterize the signaling components in the FGFR pathway.

Results: LY2874455 induced a stronger, longer-lasting growth inhibitory effect and moderate level of apoptosis for two cell lines. The third cell line did not respond to FGFR inhibition, suggesting that *FRS2* amplification alone is not sufficient to predict response. Importantly, efficacy of LY2874455 was confirmed *in vivo*, using an independent *FRS2*-amplified DDLPS xenograft model. Expression of *FRS2* was similar in the responding and non-responding cell lines and we could not find any major difference in downstream FGFR signaling. The only FGF expressed by unstimulated non-responding cells was the intracellular ligand FGF11, whereas the responding cell lines expressed extracellular ligand FGF2.

Interpretation: Our study supports LY2874455 as a better therapy than NVP-BGJ398 for *FRS2*-amplified liposarcoma, and a clinical trial is warranted.

P2 Intra-articular soft-tissue sarcoma of the knee – is extra articular resection and tumor endoprosthetic reconstruction the solution. A retrospective report on eight cases.

Markus Nottrott[1,2], Arne Streitbürger[1,2], Georg Gosheger[1], Wiebke Guder[1,2], Gregor Hauschild[1], Jendrik Hardes[1,2]
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Background: Intra-articular sarcoma of the knee joint is a very rare condition. Extra-articular resection and reconstruction with a tumor prosthesis is usually performed. We describe the results with this rare surgical procedure.

Methods: This retrospective study evaluated the clinical and functional results after extra-articular resection of the knee joint in eight patients with soft-tissue sarcomas of the knee that were reconstructed using a tumor endoprosthesis.

Results: 5 of the 8 patients ultimately had to undergo amputation, mainly due to periprosthetic infection. In addition, 2 patients experienced periprosthetic fractures. The mean Musculoskeletal Tumor Society score was 18 (range 10–22), as function was impaired due to a weak extensor mechanism.

Interpretation: These results suggest that in patients with intra-articular soft-tissue sarcomas, limb salvage procedures with tumor prostheses after extra-articular resection are associated with very high complication rates. In most cases, long-term limb salvage was not possible. When limb salvage is successful, function is also poor due to a weak extensor mechanism in the knee joint. The indication for this procedure should therefore be considered critically.

P3 Calcific myonecrosis. A rare benign mimicker of sarcoma.

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[1]Department of Radiology, Oslo University Hospital – Radiumhospital. [2] Division of Orthopaedic Surgery, Oslo University Hospital, Oslo, Norway

Background and purpose: Soft-tissue masses with calcification may be worrisome for sarcoma, such as synovial sarcoma, extraskeletal osteosarcoma or chondrosarcoma, especially when rapidly enlarging. Calcific myonecrosis is a very rare condition usually arising in the anterior compartment of the lower leg several years after trauma. Once recognised, the radiological characteristics are typical and can exclude sarcoma, and should obviate surgical biopsy or excision which has a high rate of complications.

Patients and methods: We describe the clinical history and imaging studies of 2 patients presenting with a calcified mass in the anterior compartment of the lower leg and who had a history of trauma 31 and 40 years earlier respectively.

Results: Patient 1 had few symptoms and was followed clinically without biopsy. Patient 2 had increasing pain and repeated radiographs and MRI just 3 weeks apart documented rapid enlargement. A needle biopsy with aspiration of thick paste-like material yielded fibrosis and calcification. Aspiration gave pain relief and he has gradual improvement in symptoms since.
Both patients had typical radiological findings as described in the literature, with shell-like calcification of a muscle compartment, central liquefaction and gravitational distribution of calcific milk/debris. Diagnoses were settled based on radiological findings.

Interpretation: The key to diagnosis of calcific myonecrosis is a history of trauma several years earlier and radiological findings of a peripherally calcified mass with central liquefaction. A correct diagnosis will exclude sarcoma and prevent futile and potentially harmful interventions.

P4 Combined low dose radiotherapy and denosumab as alternative to surgery in treatment of advanced metastatic bone lesions and impending fractures.

Mehdy Farhang, Richard Löfvenberg
Department of Orthopedics, University Hospital of Umeå, Sweden

Background and purpose: The incidence and prevalence of metastatic bone lesions is increasing probably due to an increase of the aging population. Surgery has generally been considered as the only treatment alternative in advanced metastatic lesions and impending fractures. Surgery however is associated with high rate of complications in this patient group. A non-surgical combination treatment with few side effects is presented; radiotherapy (single dose 8 Gy) combined with monthly denosumab injections (120 mg/sc/month).

Methods: This retrospective study includes 16 patients (8/8 F/M) with advanced skeletal metastatic lesions and impending fractures due to carcinoma. They were initially considered for surgical treatment but received the treatment described above between February 2015 and December 2018. Patients with primary bone tumors, sarcoma metastases and multiple myeloma are not included in this study.

Results: Mean follow up time was 16 months (median 9 months). Pain relief was reported by all patients. Increased local tumor growth was not observed in any of the patients during the treatment. Local recurrence occurred in one case one year after ceasing denosumab treatment. Bone healing was seen in 15 of the 16 patients. Trochanteric pathologic fracture occurred in 2 patients shortly after treatment initiation. These 2 fractures healed uneventfully after osteosynthesis. The treatment seemed to be well tolerated by the patients.

Interpretation: Single dose (8 Gy) radiotherapy in combination with denosumab results in pain relief and bone healing in advanced metastatic carcinoma bone lesions. The treatment is safe and could replace the need for extensive surgeries.

P5 Single-institution, multidisciplinary experience of soft tissue sarcomas in the chest wall.

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Background and purpose: We report our single-institution, nearly 20 years multidisciplinary experience of chest wall soft tissue sarcoma cases. This study report evaluates clinical outcome and survival.

Patients and methods: This is a retrospective review of 49 surgically treated patients with chest wall soft tissue sarcoma from 1997 to 2015.

Results: The median age of the patients was 57 years. There were 19 full-thickness and 30 partial-thickness resections. Reconstruction was warranted in 37 cases. Sarcomas were high grade in 31/49. Local recurrence developed in 8 and metastases in 9 patients. The median follow-up time was 7 years and 2 months. No 30-day mortality occurred. By the end of the study period, 35 patients were alive and 14 had died, 9 from sarcoma and 5 from other causes. The 1-, 5-, and 10-year survival rates were 94%, 76%, and 72%, while the overall recurrence-free rates were 84%, 71%, and 71% respectively. Favourable prognostic variables for survival included age <50 years and radical treatment (resection with wide margin or resection with marginal margin and adjuvant radiotherapy). Patients who had undergone non-radical treatment had a 3-fold lower chance of survival than those who had undergone radical treatment.

Interpretation: Our study suggests that surgical resection with wide margins should continue to be the mainstay for patients with chest wall sarcoma. If wide margins are not achieved, (neo)adjuvant radiotherapy should be considered to improve local control. Even extensive chest wall resections and reconstructions are safe.

P6 Insight into sarcoma biology from sarcoma cell line progression series.

Jiří Hatina [1], Michaela Kripnerová[1], Hamendra Singh Parmar[1], Zbynek Houdek[1], Pavel Dvořák[1], Katerina Houfková[1], Martin Pešta[1], Jitka Kuncová[2], Sieghart Sopper[3], Lenka Radová[4], Jiří Šána[4], Ondřej Slabý[4]*

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Background and purpose: Soft tissue sarcomas are known for their great variability in clinical behaviour. To shed light on the underlying genes and pathways, we analyzed transcriptomes of 2 single-background progression series of murine sarcoma cell lines: the JUN-fibrosarcoma progression series (Hatina et al., Tumour Biol. 2003;24:176-84) and the 3T3L1 – LM3D liposarcoma progression series (Mariani et al., Cancer Cell 2007;11:361-74). The latter has been also analyzed for expression profile of putative sarcoma stem cells.

Methods: The high-throughput gene expression analysis has been performed using the GeneChip Mouse Genome 430 2.0 Array (ThermoFisher Scientific). Subsequently, we identified a small group of genes coregulated in both highly transformed JUN-3 and LM3D sarcoma cells ("sarcoma progression" signature). Putative sarcoma stem cells were identified in both 3T3L1 and LM3D as side population (SP) cells, SP cell specific transcriptomes were identified in both cell line and their overlap yielded a "sarcoma stemness" signature.

Results: The "sarcoma progression" signature features the complex downregulation of the canonical Wnt/β-catenin signalling (upregulated genes include Dickkopf-2 and -3, Apcdd1, Meg3, Fibulin-5, and Ints6), simultaneously suggesting a switch to the Wnt5-Ror2 noncanonical pathway. Stemness regulation seems to be dominated by an extensive array of Hippo-pathway genes (Yap, FoxM1, Pttg, Tacc3, Btf3, as well as Lats2).

Interpretation: We believe that our models and differentially expressed genes identified provide important new information on biology of soft tissue sarcoma, and may be useful towards identification of new prognostic markers and potential therapeutic targets.

Supported by the Czech Science Foundation project No 17-17636S.

P7 Functional precision medicine for sarcoma at the Karolinska University Hospital.

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Background and purpose. The biggest challenge in sarcoma diagnosis and treatment is heterogeneity and low incidence. Chemotherapy, although relatively efficient for some sarcoma subtypes, generally results in poor clinical responses and is mostly recommended for advanced disease.
New generation sequencing techniques have been applied to elucidate the genome landscapes of several sarcoma subtypes, such as that of leiomyosarcoma, osteosarcoma, Ewing sarcoma and liposarcoma. The analysis has led to the identification of specific genetic alterations. However, to date, approved drugs can target only few of these alterations, and the information generated is seldom used to guide patient treatment.

Methods. We have evaluated a functional approach for precision medicine in sarcoma. In this system, we have cultured and characterized patient-derived sarcoma cells and evaluated their sensitivity to 525 approved and investigational anti-cancer agents. The sarcoma biopsies and derived cells were characterized by pre-defined gene panel sequencing, cancer-driver gene expression and, by detecting and quantifying the expression of fusion oncoproteins in the cultured cells *in situ*.

Results. Bone and soft tissue sarcomas as well as healthy muscle cell cultures were established from surgical biopsies or fine needle aspirations from 40 sarcoma patients. In our first pilot study we showed that the patient-derived sarcoma cells preserve the genetic characteristics of the tumor of origin. Drug sensitivity testing on patient derived sarcoma cells has led to the identification

of specific inhibitors active on sarcoma subtypes. The sarcoma associated non-receptor protein kinase (cSrc) inhibitor Dasatinib was identified as an active drug in sarcomas carrying chromosomal translocations. Interestingly, we found an association between the drug response of the patient sarcoma cells *ex-vivo* and the response to the actual treatment of the patient.

Interpretation. Our results show that functional screens on patient derived sarcoma cells is a feasible approach for the identification of potential treatments or new therapeutic indications for approved drugs for sarcoma patients with poor treatment options. It is however necessary to evaluate the clinical impact of this approach in predicting patient response to new treatments.

P8 Solitary fibrous tumor, association between *NAB2-STAT6* fusions and clinical outcome.

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Background: Solitary fibrous tumor (SFT) is a rare mesenchymal neoplasm with fibroblast-like differentiation whose clinical behavior is difficult to predict based solely on histopathological examination. Recently, a pathognomonic *NAB2-STAT6* fusion has been discovered in most SFTs. In the current study, we have investigated the association between different *NAB2-STAT6* fusion breakpoints, clinicopathological parameters and clinical outcome.

Methods: Tumors from 44 SFT patients, diagnosed by strong STAT6 immunostaining, were analyzed. 22 patients developed metastasis after the median of 5,3 years (range 0-19 years) and 22 patients had only localized disease. Histopathological criteria like tumor size, cellularity, necrosis, atypia, mitotic counts, and infiltrative pattern as well as clinical data have been evaluated. RNA was successfully isolated from FFPE and fresh frozen tumor tissue from 39 tumors and sequenced using TruSight RNA Pan-Cancer Panel to identify *NAB2-STAT6* fusions.

Results: We identified a *NAB2-STAT6* fusion in 90% of the samples, where 11 different breakpoints were detected. The most frequent fusion types were *NAB2ex6-STAT6ex16/17* (37%) and *NAB2ex4-STAT6ex2* (23%). We divided all

the fusion types into 2 groups by the length of STAT6 gene. Kaplan-Meier analysis showed that the 2 groups had statistically significant difference in metastasis-free survival (log-rank test, p=0,003).

Interpretation: In the current study we show that different breakpoints of *NAB2-STAT6* fusion are associated with clinical outcome. The discovered association can be a potential tool for better stratification of SFT patients into high and low-risk groups.

P9 Reconstruction of the humerus using the Comprehensive Segmental Revision System in patients suffering from metastatic bone disease.

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Background and purpose: Surgical treatment of malignant bone tumors of the humerus is often followed by reconstruction with a tumor prosthesis. The Comprehensive Segmental Revision System® (Zimmer-Biomet, Warsaw, IN, USA) (SRS) offers a new option for such reconstructions and we evaluated implant failure incidence, surgical complications, and clinical results.

Patients and methods: A study of 22 consecutive patients (F/M= 9/13, mean age = 67 (51-83) years) suffering from metastatic bone disease having surgery with bone resection of the humerus (18 proximal humerus replacements (hemiarthroplasty/total-reverse joint = 4/14) and 4 distal humerus replacements) and reconstruction using the SRS prosthesis from May 2014 to January 2017.
Statistics: Kaplan-Meier survival analysis (patient survival) and Aalen-Johansson estimate (incidence of implant failure) presented with 95%-confidence intervals (CI). Results are given as mean (range).

Results: 2-year overall patient survival was 41% (CI: 20-62%). 5 patients suffered from surgical complications: radial nerve palsy (n=2) and superficial postoperative infection (n=3). 2 patients experienced revision surgery: soft tissue revision (n=1) and a hemiarthroplasty revised to a reverse total shoulder replacement (n=1). 2-year implant failure incidence was 5% (CI:0-13%). The mean MSTS score (n=6) was 16 (11-25), 221 (95-360) days postoperatively.

Interpretation: The use of the SRS prosthesis in orthopedic oncology patients resulted in low incidence of implant failure. Since the introduction of the SRS prosthesis in our department represented at shift from using hemiarthroplasty to prefer total-reverse joint implants, the fact that we observed no shoulder dislocations was a positive short-term result.

P10 Surgical treatment of Solitary Fibrous Tumors in the thoracic cavity.

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Background and purpose: Solitary Fibrous Tumor (SFT) is a rare tumor of mesenchymal origin. 30 % originate in the thoracic cavity. Most SFTs are macroscopically demarcated, have a stalked attachment, and a fibroelastic consistency. They can display an invasive nature and metastasize. A diameter exceeding 10 cm, invasiveness and necrosis are radiological indicators of malignancy. Histopathologic indicators of malignant transformation of importance for prognosis are a high number of spindle cells, high mitotic activity and atypia. SFTs are often asymptomatic and therefore large at the time of diagnosis. There are no known risk factors or gender predisposition.

Patients and methods: Retrospective review of patients operated for SFT at Haukeland University Hospital from January 2009 through February 2019.

Results: 13 patients were operated for thoracic SFT, median age was 66 years (range 53 – 69) and 7 were female. 8 patients had a history of smoking, while 2 were exposed to asbestos. Diagnosis was established based on symptoms in 4 patients, while the others were accidentally discovered. Patients with benign histology had no signs of tumor recurrence. Of the 4 patients with malign histology, 3 died from recurrence and metastatic disease after 9 months, 3 and 8 years, respectively.

Interpretation: Thoracic SFTs have potential for malignant transformation, which is associated with poor prognosis. Early resection should therefore be

considered in all patients regardless of histology in order to accomplish long-term survival.

P11 Ewing´s sarcoma of the calcaneus treated by limb sparing surgery with calcanectomy and reconstruction with a composite of an allograft and a vascularized osteocutaneous fibula graft.

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Background and purpose: Primary malignant bone tumurs of the calcaneus are very seldom, and due to poor possibilities to do surgery with wide margins in this region and limited options for reconstruction after calcanectomy many orthopedic oncologists use amputation as the preferred surgical treatment in such cases. We present two cases of Ewing´s sarcoma of the calcaneus treated with calcanectomy and reconstruction with a composite of an allograft and a vascularized osteocutaneous fibula graft.

Patients and methods: Two girls almost 6 years old (case 1) and 16 years old (case 2) at the time of calcanectomy in respectively August 2012 and October 2013. Both patients received pre- and post-operative chemotherapy.
In both cases removal of the calcaneus was performed using a combined medial and lateral incision. In case 1 a femoral head allograft was fitted to replace the removed calcaneus, and in case 2 a calcaneus allograft was used. In both cases, with the aim of obtaining arthrodesis, the allograft was fixed to the talus and cuboid bone with screws. A distally pedicled osteocutaneous flap was use for reconstruction of soft tissue, and a 5-6 cm piece of vascularized fibula bone was fitted into the allograft and fixed using staples.

Results: Arthrodesis between talus and the graft healed and full weight-bearing was allowed in both cases 8-9 months postoperatively. At follow-up 6½ and 4½ years after surgery both patients were without local recurrence or metastases, and they were both pain-free and able to walk using normal footwear without any walking-aids.

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Hb (g/l)	Hb (mmol/l)	Kroppvekt 50 – < 70 kg	Kroppsvekt ≥ 70 kg
≥ 10	≥ 6,2	1 000 mg	1 500 mg
< 10	< 6,2	1 500 mg	2 000 mg

Behandlingseffekt bør følges opp med blodprøver. Kumulativ jerdose må noen ganger justeres for å oppnå ønsket Hbkonsentrasjon. ***Jernsubstitusjon for blodtap:*** *Voksne inkl. eldre:* Behandling skal rettes mot substitusjon av samme mengde jern som i mengde tapt blod. Hvis Hb-nivået er redusert benyttes Ganzoni-formelen over, forutsatt at jerndepotene ikke behøver å fylles. Hvis mengden mistet blod er kjent, motsvarer tilførsel av 200 mg jern den jernmengde som finnes i 1 enhet blod. Jerndose som skal erstattes (mg Fe) = antall tapte enheter blod × 200 (mg Fe). ***Spesielle pasientgrupper:*** *Barn og ungdom <18år:* Preparatet skal ikke benyttes, da det ikke foreligger tilstrekkelig dokumentasjon for effekt og sikkerhet. ***Tilberedning/Håndtering:*** Kun til engangsbruk, se for øvrig pakningsvedlegg. *Ved i.v. bolusinjeksjon:* Dosen kan gis ufortynnet eller fortynnes med maks. 20 ml steril 9 mg/ml (0,9 %) natriumkloridoppløsning. *Ved i.v. dråpeinfusjon:* Skal blandes med maks. 500 ml steril 9 mg/ml (0,9%) natriumkloridoppløsning. ***Administrering:*** Preparatet kan gis som i.v. bolusinjeksjon, som i.v. dråpeinfusjon eller som en injeksjon direkte i venesløngen ved hemodialyse. Monofer bør ikke gis samtidig med oral jernbehandling, da opptaket av oralt jern kan bli redusert. *I.v. bolusinjeksjon:* Doser opptil 500 mg pr. injeksjon opptil 3 ganger ukentlig og med injeksjonshastighet på opptil 250 mg jern/minutt kan tilføres. Dosen kan gis ufortynnet eller fortynnes, se Tilberedning/Håndtering. *I.v. dråpeinfusjon:* Hele jerndosen, opptil 20 mg jern/kg kroppsvekt, gis i 1 engangsinfusjon eller som ukentlige infusjoner til den ønskelige kumulative jerndosen er gitt. Hvis den kumulative jerndosen overstiger 20 mg jern/kg kroppsvekt, deles dosen i 2 infusjoner som gis med minst 1 ukes intervall. Det anbefales, når det er mulig, å gi 20 mg jern/kg i 1 administrering. Avhengig av klinisk skjønn kan 2. administrering avventes oppfølgende laboratorieprøver. Doser opptil 1000 mg må infunderes i løpet av mer enn 15 minutter, doser over 1000 mg må infunderes i løpet av 30 minutter eller mer. Skal fortynnes før infusjon, se Tilberedning/Håndtering. *Injeksjon under hemodialyse:* Preparatet kan tilføres under hemodialyse ved injeksjon direkte inn i venesløngen i dialysatoren under hemodialysen. Samme prosedyre som beskrevet for i.v. bolusinjeksjon benyttes. **Kontraindikasjoner:** Overfølsomhet for innholdsstoffene eller for andre parenterale jernpreparater. Anemi uten at det foreligger jernmangel (f.eks. hemolytisk anemi). For høyt jernnivå eller dårlig utnyttelse av jern (f.eks. hemokromatose, hemosiderose). Dekompensert leversykdom. **Forsiktighetsregler:** Parenteralt administrerte jernpreparater kan gi overfølsomhetsreaksjoner, inkl. alvorlige og potensielt dødelige anafylaktiske/anafylaktoide reaksjoner. Overfølsomhetsreaksjoner er også sett etter tidligere bivirkningsfrie doser av parenterale jernkomplekser. Risikoen er økt ved kjente allergier, inkl. legemiddelallergier, herunder pasienter som tidligere har hatt alvorlig astma, eksem eller annen atopisk allergi, og ved immunitets- eller inflammatoriske tilstander (f.eks. systemisk lupus erythematosus, revmatoid artritt). Behandlingen må stoppes umiddelbart ved overfølsomhetsreaksjoner eller tegn på intoleranse under administrering. Plutselig rødhet (flushing) i ansiktet, akutte brystsmerter og/eller ryggsmerter og følelse av trang het i brystet forekommer under behandling med i.v jern (sjeldent). Dette kan ligne på tidlige symptomer på en anafylaktoid/anafylaktisk reaksjon. Infusjonen bør stoppes og pasientens vitale parametre måles. Symptomene forsvinner kort tid etter at administreringen av jern er stanset. De vender vanligvis ikke tilbake dersom administreringen startes på nytt med en lavere infusjonshastighet. Utstyr for hjerte-/åndedrettsgjenopplivning og utstyr for håndtering av akutte anafylaktiske/anafylaktoide reaksjoner skal være tilgjengelig, inkl. en injiserbar 1:1000-adrenalinoppløsning. Ytterligere behandling med antihistaminer og/eller kortikosteroider skal gis ved behov. Hos pasienter med nedsatt leverfunksjon, skal parenteralt jern kun administreres etter en grundig nytte-/risikovurdering. Preparatet skal unngås hos pasienter med hepatisk dysfunksjon (ALAT og/eller ASAT >3 ganger øvre normalgrense) der jernoverskudd er en utløsende faktor, spesielt ved porphyria cutanea tarda (PCT). Grundig overvåkning av jernstatus anbefales for å unngå jernoverskudd. Parenteral jernbehandling bør brukes med forsiktighet ved akutt eller kronisk infeksjon. Preparatet bør ikke gis ved aktiv bakteriemi. Overfølsomhetsreaksjoner kan oppstå hvis i.v. injeksjon skjer for raskt. Det skal utvises forsiktighet for å unngå paravenøs lekkasje ved administrering. Paravenøs lekkasje kan gi hudirritasjon og potensielt langvarig brun misfarging på injeksjonsstedet. Ved paravenøs lekkasje må administreringen avbrytes øyeblikkelig. **Interaksjoner:** For utfyllende informasjon fra Legemiddelverket om relevante interaksjoner, se B03AC. Bør ikke gis samtidig med orale jernpreparater, da absorpsjon av oralt jern vil reduseres. Oral jernbehandling bør tidligst påbegynnes 5 dager etter siste injeksjon **Graviditet, amming og fertilitet:** *Graviditet:* Begrensede data fra bruk hos gravide. Grundig nytte-/risikovurdering er derfor nødvendig før bruk under graviditet. Skal ikke brukes under graviditet hvis ikke absolutt nødvendig. Hvis fordelen med behandling er vurdert å oppveie mulig risiko for både mor og foster, bør behandling begrenses til 2. og 3. trimester. Bradykardi hos fosteret er sett hos gravide med hypersensitivitetsreaksjoner. *Amming:* I terapeutiske doser forventes ingen negativ effekt hos diende nyfødte/småbarn. *Fertilitet:* Ingen tilgjengelige data. Fertilitet er ikke påvirket i dyrestudier. **Bivirkninger:** Akutte, alvorlige overfølsomhetsreaksjoner kan forekomme ved tilførsel av parenterale jernpreparater. De opptrer vanligvis i løpet av de første minuttene etter infusjonsoppstart og er karakterisert ved plutselig innsettende respirasjonsbesvær og/eller sirkulasjonssvikt; dødsfall er rapportert. *Vanlige* (≥1/100 til <1/10): Gastrointestinale: Kvalme. *Øvrige:* Reaksjoner på injeksjonsstedet (erytem, hevelse, sviende følelse, smerter, blåmerker, misfarging, ekstravasasjon, irritasjon). *Mindre vanlige* (≥1/1000 til <1/100): Gastrointestinale: Magesmerter, brekninger, dyspepsi, forstoppelse, diaré. Hjerte/kar: Takykardi, hypertensjon, hypotensjon. Hud: Pruritus, urticaria, utslett, rødhet, svetting, dermatitt. Immunologiske: Overfølsomhet, inkl. alvorlige reaksjoner. Luftveier: Brystsmerter, dyspné, bronkospasme. Muskelskjelettsystemet: Ryggsmerter, myalgi, artralgi, muskelkramper. Nevrologiske: Hodepine, parestesi, dysgeusi, uklart syn, bevisstløshetstap, svimmelhet, utmattelse. Stoffskifte/ernæring: Hypofosfatemi. Undersøkelser: Forhøyede leverenzymer. Øvrige: Reaksjoner på injeksjonsstedet; pyreksi, frysninger/skjelvinger, infeksjon, lokal flebitt. *Sjeldne* (≥1/10 000 til <1/1000): Hjerte/kar: Arytmi. Hud: Angioødem. Immunologiske: Anafylaktoide/anafylaktiske reaksjoner. Nevrologiske: Dysfoni, kramper, tremor, endret mental status. Øvrige: Malaise, influensalignende symptomer. Beskrivelse av utvalgte bivirkninger: Forsinkede hypersensitivitetsreaksjoner kan forekomme etter i v jern og kan være alvorlige. De er karakterisert ved leddsmerter, muskelsmerter og av og til feber. Debut varierer fra noen timer inntil fire dager etter infusjon. Symptomene varer vanligvis 2–4 dager og kan gå tilbake spontant eller etter bruk av vanlige smertestillende midler. **Overdosering/ Forgiftning:** Lav toksisitet. Preparatet tolereres godt, og har en minimal risiko for utilsiktet overdosering. *Se Giftinformasjonens anbefalinger:* For jern III-verdig, injeksjonspreparater B03AC. **Egenskaper:** *Virkningsmekanisme:* Kontrollert og langsom frigivelse av biotilgjengelig jern til jernbindende proteiner, med liten risiko for fritt jern. Etter i.v. infusjon opptas jernkomplekset hurtig av cellene i RES, spesielt i lever og milt, som splitter komplekset i komponentene jern og isomaltosid 1000. Jernet bindes umiddelbart til tilgjengelige proteinstrukturer og danner hemosiderin eller ferritin, de fysiologiske lagringsformene for jern. En mindre del bindes til transportmolekylet transferrin. Det fysiologiske jernet brukes til dannelse av Hb og til oppbygging av jerndepotene. *Halveringstid:* Enkeltdose på 100–1000 mg gir plasma $t_{1/2}$ 1–4 dager. *Metabolisme:* Isomaltosid 1000 blir enten metabolisert eller utskilt uomdannet. *Utskillelse:* Jern utskilles ikke lett fra kroppen og opphopning kan være giftig. Komplekset utskilles ikke via nyrene pga. dets størrelse. Små mengder jern utskilles i urin og feces. **Oppbevaring og holdbarhet:** Preparatet bør av mikrobiologiske hensyn brukes umiddelbart etter åpning, hvis ikke åpningen er utført slik at fare for mikrobiologisk forurensning er eliminert. Etter fortynning opptil 1:250 med steril 0,9 % natriumkloridoppløsning er preparatet påvist kjemisk og fysisk stabilt i 48 timer ved 30°C. Hvis preparatet ikke brukes umiddelbart, er det brukeren selv som er ansvarlig for oppbevaringstiden og -forholdene før bruk. **Sist endret:** 21.08.2017 (priser og ev. refusjon oppdateres hver 14. dag) **Basert på SPC godkjent av SLV/EMA:** 19.05.2017.

Monofer, INFUSJONS-/INJEKSJONSVÆSKE, oppløsning:

Styrke	Pakning og varenummer	Refusjon[1] Byttegruppe	Pris (kr)[2]	R.gr.[3]
100 mg/ml	5 × 1 ml (hettegl.) 089801	– –	1542,40	C
	5 × 5 ml (hettegl.) 089913	– –	7417,90	C
	2 × 10 ml (hettegl.) 089936	– –	6061,00	C

[1]Gjelder forhåndsgodkjent refusjon. For informasjon om individuell stønad, se HELFO. [2]Pakninger som selges uten resept er angitt med stjerne * i kolonnen Pris. Det er fri prisfastsettelse for pakninger som selges uten resept, og maksimal utsalgspris kan derfor ikke angis. [3]Reseptgruppe: Utleveringsgruppe.[3]Reseptgruppe: Utleveringsgruppe.

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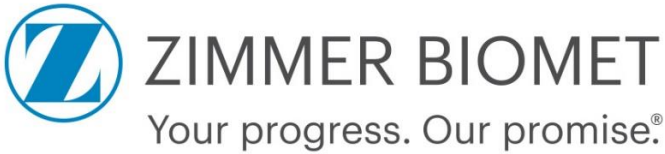
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Wednesday 8 May, 2019

18.30 Deaparture from conference hotel
19.00 Dinner at Fløyen restaurant Bergen

Thursday 9 May, 2019

18.45 Departure from conference hotel
19.00 Dinner at Grieghallen foyer

Notes

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