process such as VEGF, bFGF, COX 2, and a relatively high level of DNA topoisomerase II a therapeutic target of etoposide. All microrarray experiments were repeated twice. The high levels of growth factors and COX2 mRNAs were confirmed by immunoistochemistry analysis. On the basis of these results we preceded to antiangiogenetic approach. At this time after few months of follow up the instrumental examinations confirmed a disease stabilization in three patients, a light regression in a patient, while the rapid and extensive progression of the disease caused the death of a child after a month of antiangiogenetic therapy.
Thus, we showed that the gene expression monitoring could provide new insight into many aspects of posterior fossa tumors as revealing targets for antiangiogenetic therapy. New drug development and evaluation will likely be accelerated both through the identification of novel molecular targets and through the selection of patients for clinical trials with specific tumor gene expression profile.

1245 POSTER

Experience with treatment of lymphocyte predominance Hodgkin's disease in children

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Background: We retrospectively evaluate clinical characteristics and outcome in children with nodular lymphocyte predominant Hodgkin disease (LPHD), which is a rare entity characterized by neoplastic popcorn cells CD 20+, CD 30−, CD 15−, EMA+, Bc 16+ within nodular background composed of small B lymphocytes.
Material and methods: From January 1996 to December 2004, 155 children and adolescents with Hodgkin disease were treated in the Department of Paediatric Hematology and Oncology of the Faculty Hospital Motol in Prague. Nodular lymphocyte predominant Hodgkin disease was histologically confirmed in 7 children (4.5%) – 6 boys, 1 girl. The age range was 7–17 years (mean of 14.9 years). Initial staging included complete physical examination, blood studies and imaging studies as X rays, CT scans and 4 patients (57%) had PET scan. Disease presentation was localized in 5 patients (71%) and advanced in two patients (29%) – both Stage III. Only one patient presented with B symptoms and one patient had bulky disease. Neck was the common site of involvement (5 patients). All patients were treated with chemotherapy combined with involved field radiotherapy. Chemotherapy treatment was not uniform – 3 patients received 5 cycles DBVE-PC (doxorubicin, bleomycin, vinkristine, etoposid, prednisone, cyclofosfamide), two patients received 2 cycles DBVE, one patient 4 cycles DBVE due to partial response after the first two cycles and one was treated with 4 cycles ABVD/COPP. Involved field radiation therapy was administered to all patiens in dose 21–25.5 Gy.
Results: All patients achieved complete remission after combined modality treatment. At a median follow up of 3.2 years (range 2.2 to 9 years) 2 patients relapsed (29%). Both relapses were more than 1 year after primary diagnosis (20 and 28 months) and both patients achieved second complete remission.
Conclusion: Current strategy of treatment LPHD is aimed at high cure rates with less toxic regimens to limit risk of late complications and secondary malignancies. But careful long term follow-up is essential for risk of late relapses.
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1246 POSTER

Phase II study of Gemcitabine in children with solid tumors of mesenchymal and embryonal origin

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Background: Gemcitabine inhibits DNA synthesis and repair and shows efficacy in many types of adult malignancies, including previously untreatable pancreatic cancer. No data are available about its effectiveness in children. To determine the efficacy of gemcitabine the drug was administred by i.v. short term infusion over 30 min at a dose of 1200 mg/m^2 weekly for 3 weeks in children with first or subsequent recurrence of a solid tumor of embryonic or mesenchymal origin if standard therapy failed to offer any curative therapeutic option.
Results: From May 2003 to April 2005, 14 male and 6 female patients at the median age of 15.8 years (2–23) were recruited for the prospective open-label multicenter phase II study of gemcitabine in Germany and Austria. The patients suffered from soft tissue sarcoma (n = 8), Ewing's sarcoma (n = 4), Neuroblastoma (n = 3), Hepatoblastoma (n = 2), Osteosarcoma (n = 2) or Nephroblastoma (n = 1). Mean duration of therapy was 31.4 days (7–99), equalling 4.6 (2–11) courses of gemcitabine. 2 patients, whose "Best Overall Response" according to RECIST-criteria (i.e. minimal 6 courses) was evaluable, had stable disease documented for 69 and 70 days, respectively (neuroblastoma, Ewing's sarcoma), whereas no response to gemcitabine was documented. The other patients left the trial mainly due to early progress. The mean dosage per course was 1104 mg/m^2. In 33/88 evaluable courses dosage had to be reduced or omitted for grade 3–4 haematologic toxicity. No suspected unexpected serious adverse reactions (SUSAR's) were reported.
Conclusions: Gemcitabine at the dose and schedule of this trial was not effective for children with refractory solid tumors. Given the variety of other promising agents, further evaluation of gemcitabine as single treatment of childhood solid tumors does not appear to be warranted. Nevertheless, publishing of negative results is indispensable for diminishing the Publication only bias.

1247 POSTER

Alterations of brain metabolism after therapy of paediatric brain tumors. a serial proton magnetic resonance spectroscopy study

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Aim: The aim of the study was to evaluate metabolic changes in tumor bed occurring after therapy of pediatric brain tumors using serial proton magnetic resonance spectroscopy studies.
Material: The examined group consisted of 15 children with brain tumors treated with surgery, chemotherapy and radiotherapy. Eight children had medulloblastomas, four had astrocytomas, one had oligodendroglioma, one glioblastoma and one had a mixed tumor having features of both PNET and glioblastoma.
Methods: Short echo-time (TE 30 ms) point-resolved spectra were acquired using 2 Tesla clinical scanner (Elscint Prestige). The proportions of N-acetylaspartate (NAA), choline (Cho), myo-inositol (mI), lactate (Lac) and lipids (Lip) signal intensities were calculated using creatine (Cr) signal as an internal reference. The spectra were acquired from tumor bed and form unaffected brain tissue of contralateral hemisphere as a comparison. The first examination was made between third and sixth month after therapy, the second 8–12 months after therapy and the third examination was performed approximately 18 months after completion of therapy. The results were compared using t-test for dependent samples.
Results: In all cases there were significant disturbances in brain metabolism detected both in the spectra acquired from the tumor bed and from control area. The most important alterations were decrease of NAA/Cr and increase of Cho/Cr, Lac/Cr and Lip/Cr proportions. The observed changes did not differ significantly between subsequent examinations.
Conclusions: Alterations of brain metabolism after combined therapy of brain tumors in children affect both tumor bed and uninvolved area of brain tissue and are stable in subsequent examinations indicating long-lasting or permanent brain damage.

1248 POSTER

Expression profiles of 'minimal residual disease' (MRD) markers for neuroblastoma in peripheral blood and its cell fractions by real-time quantitative PCR (RQ-PCR)

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Background and Aim: In neuroblastoma (NBL) specific and sensitive markers are essential for MRD detection. NBL cells highly express tyrosine hydroxylase (TH), DOPA decarboxylase (DDC), chromogranin-B (CHGB), GD2 synthase (GD2), dopamin beta hydroxylase (DBH), paired-like homeobox 2B (PHOX2B), growth associated protein 43 (GAP43), synaptosomal associated protein (SNAP), stathmin-like 2 (STMN2), stathmin-like 4 (STMN4), and cholinergic receptor (CHRNA3). TH, DDC, GD2 and CHG are among common MRD markers for NBL. In previous experiments, PHOX2B had no expression in normal bone marrow (BM), DDC was expressed in 2/67, and other markers in $\geqslant$ 14/67 samples. In peripheral blood (PB), PHOX2B, DDC, CHRNA3 were negative; TH was positive in 2/22, DBH in 1/22 samples, and others in $\geqslant$ 10/22 of samples.