of MRI, PET and NA in terms of distinguishing stable from progressive disease in an interdisciplinary neurooncological tumor-board.

RESULTS: A statistically significant and high correlation was found for all 3 imaging modality (MRI, PET and NA) in terms of correlation with the TB (all p<0.01). Also over time, the TB maintained the statistically significant correlation with all testing procedures (all p<0.01). NA correlated with all testing procedures significantly (TB: r=0.69, CI 0.53–0.79; MRI: r=0.70, CI 0.55–0.81; PET10: r=0.48, CI 0.28–0.64, all p<0.01) in general. The correlations with MRI and PET increased over time, reaching a statistical significance at last FU. Consistency of test procedures increased over time. In case of inconsistent results in multimodal testing, NA and PET matched more frequently with the TB than MRI.

CONCLUSION: As a novel finding, a strong correlation of NA with the TB in general and with other testing modalities during consecutive FU could be demonstrated. These results may suggest that NA is not inferior as compared to MRI and PET in terms of correlation with the TB. Whenever multimodal testing revealed inconsistent results, NA and PET were more frequently in accordance with the TB than MRI. Our data encourages further research to improve NA in multimodal response assessment.

P08.50 RAMAN SPECTROSCOPY FOR THE IDENTIFICATION OF ISOCITRATE DEHYDROGENASE (IDH) MUTATED GLIOBLASTOMAS
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INTRODUCTION: Raman spectroscopy is an emerging biophotonic tool for the identification of disease. By probing the unique molecular vibrations that depend on the composition and structure of samples, it provides a wealth of information on a cellular and molecular level of both solid and liquid specimens without the use of external agents such as dyes, stains or radioactive labels. Glioblastomas can be stratified by the presence or absence of isocitrate dehydrogenase (IDH) 1 or 2. Determination of IDH status is critical for histological diagnosis and clinical decision-making, with evidence that the presence of IDH mutation confers better prognosis and a better response to chemotherapy. Mutation-positive tumours accumulate high concentrations of D-2-hydroxylutarate, resulting in metabolic and epigenetic changes which we hypothesise will be reflected in a change in Raman scattering. In this study, we investigate the feasibility of using Raman spectroscopy to differentiate between IDH1 positive (IDH1+) and negative (IDH1-) tumours through classification modelling.

METHOD: Forty biopsies of glioblastoma (WHO grade IV) in patients under the age of 40 years were classified into either IDH1+ (n=20) or IDH1- (n=20) using IDH-1(R132H) and BCAT-1 immunohistochemistry. 6µm thick sections were prepared from formalin fixed paraffin embedded specimens. Based on the hematoyxlin and eosin stained contiguous section, high tumour density in the unstained sections were identified and subjected to analysis. Raman maps of ca. 1mm² were acquired from up to two locations on each sample using an inVia Raman microscope (Renishaw, UK) configured with a 785nm excitation laser source. 30 spectral maps (60,325 spectra) from a total of 16 biopsy samples (8 IDH1+ and 8 IDH1-) have been collected to date. Principal component analysis (PCA) demonstrated good separation of the IDH1+ and IDH1- groups. A PCA-linear discriminant analysis classification model demonstrated 96.98% sensitivity and 90.1% specificity for predicting the presence of an IDH1 mutation.

CONCLUSIONS: The results demonstrate the feasibility of using Raman spectroscopy to accurately distinguish IDH mutated glioblastomas from their IDH1 counterparts. Raman spectroscopy could provide a powerful tool to aid neuropathological diagnosis and genetic classification of glial tumours in the future.

ACKNOWLEDGEMENTS: This work was supported by Cancer Research UK (CRUK) grant number C38302/A17319, and the NIHR Oxford Bio Medical Research Centre

P08.51 EXTRACRANIAL METASTASIS OF OLIGODENDROGLIOMA
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INTRODUCTION: Extracranial metastases from oligodendrogliomas are unusual and few cases have been described until now.

We describe a case of a patient who presents extracranial metastases of an anaplastic oligodendroglioma without evidence of local recurrence.

MATERIAL AND METHODS: clinical data and literature review

RESULTS: A 58-years-old man, diagnosed with anaplastic oligodendrogliaoma in May 2011, was submitted to surgery and radiotherapy (60Gy/30fr) and he started active surveillance. In Dec/Nov 2012, he presented with headache and emesis and a MRI was performed but there wasn’t evidence of recurrence. These symptoms were self-limited. In February 2013, suddenly the patient develop sciatic pain and the column CT showed spinal cord compression. Raman spectroscopy was performed and showed different tissue mass pressing the spinal cord in L3, L4 and luda equine but without cerebral recurrence. The patient was submitted to spinal cord decompression and excision of the mass. The histologic result revealed metastasis of the previous brain tumor and deletion of 1p/19q was positive. After surgery, radiotherapy was performed. Left pleuropulmonar lesion with minimal bilateral pleural effusion was detected in PET-CT. The patient was submitted to a toracocentesis that also revealed metastasis of CNS (Central Nervous System) tumor.

The patient started chemotherapy with temozolomide in June 2013 with improvement of clinical status. In April 2014 he presented with progressive clinical deterioration related with pleural progression and temozolomide was suspended. Best supportive care was offered until time of death, 3 months after.

The oligodendroglioma is a CNS tumour that rarely metastasize outside of CNS, despite the local aggressiveness. The first case was described in 1951, ever since were published about 30 cases. The low rate of extraneural expression of oligodendroglioma may be explained by the short survival of these patients, the presence of blood-brain-barrier and the absence of lymphatic drainage. The most frequent metastatic site is the lung, followed by lymph nodes, lung and pleura, scalp and other soft tissue. The presence of deletion 1p/19q is related with a higher chemosensitivity and better survival rates, however, it is also related with higher potential of extraneural disease.

CONCLUSION: This case describes an unusual behavior of the oligodendroglioma, although, extraneural expression of oligodendroglioma might be more frequent in long surviving pre-treated patients, especially in the presence of deletion 1p/19q.

P08.52 PROTON THERAPY RE-IRRADIATION IN LARGE-VOLUME RECURRENT GLIOBLASTOMA
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PURPOSE: To report preliminary results of re-irradiation with proton therapy (PT) in large-volume recurrent glioblastoma (rGBM).

MATERIAL/METHODS: Between January and December 2015 ten patients (pts) with rGBM were re-irradiated with PT. All pts were previously treated with photon radiotherapy (60 Gy) with concomitant and adjuvant TMZ for 1–20 cycles (median, 7). Seven pts were re-irradiated at first relapse/progression. Four patients were re-irradiated after partial tumor resection. Median age and Karnofsky performance status at re-irradiation were 57 years (range, 41–68) and 80%, (range, 70–100), respectively. Median time between prior radiotherapy and PT was 9 months (range, 5–24). Target definition was based on CT, MR, and 18F-DOPA PET imaging. GTV included any area of contrast enhancement after contrast medium administration plus any pathological PET uptake regions. CTV was generated by adding to GTV a 3-mm uniform margin manually corrected in 3D. GTV included any area of contrast enhancement after contrast medium administration plus any pathological PET uptake regions. CTV was generated by adding to GTV a 3-mm uniform margin manually corrected in 3D. CTV was expanded by 4 mm to create PTV. Median PTV volume was 90 cc (range, 46–231). All pts received 36 GyRBE in 18 fractions. Four pts also received concomitant temozolomide (73 mg/m2/die, 7 days/week). All pts were treated with active beam scanning PT using 2–3 fields with single field optimization technique.

RESULTS: All pts completed the treatment without breaks. Registered acute side effects (according to Common Terminology Criteria for Adverse Events version 4.0 - CTCAE) include grade 1–2 skin erythema, alopecia, fatigue, conjunctivitis, concentration impairment, dysphasia, and headache. There were no grade 3 or higher toxicities. One patient developed grade 1 neuropenia. Five pts started PT under steroids (2–7 mg/daily); two of them reduced the dose during PT, while three kept the same steroids dose. None of remaining pts needed steroids therapy. Registered late side effects (according to CTCAE version 4.0) include grade 1–2 alopecia, fatigue, concentration impairment, and dysphasia. During follow-up two pts (20%) developed radionecrosis (diagnosed at imaging) with mild symptoms controlled with steroids. There were no grade 3 or higher toxicities. The median progression-free survival (PFS) was 6.4 months, while the 3-, 6- and 9-month PFS rates were 80%, 67% and 22%, respectively. Median overall survival (OS) after PT was not achieved, while the 6- and 12-month survival after PT rates were 100% and 60% respectively.

CONCLUSION: PT re-irradiation of large-volume PTGMB showed to be feasible and safe even with concomitant chemotherapy administration. Despite the small number of patients and the retrospective nature of the study PFS and OS rates were promising and deserve further evaluation in a larger pts sample.