Fractionated Stereotactic Radiation Therapy for Orbital Optic Nerve Sheath Meningioma – a Single Institution Experience and a Short Review of the Literature

Roberto PACELLI1,2*, Laura CELLA1,2, Manuel CONSON2, Fausto TRANFA3, Diego STRIANESE3, Raffaele LIUZZI1,2, Raffaele SOLL1,2, Antonio FARELLA2, Marco SALVATORE2 and Giulio BONAVOLONTÀ3

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Optic nerve sheath meningioma (ONSM) is a rare orbital tumor that generally induces a slow progressive visual loss in affected patients. Radiotherapy (RT) has currently become the first choice to treat ONSM. In this study our experience in ONSM treatment with fractionated stereotactic radiotherapy (FSRT) is reported. Five patients with diagnosis of orbital ONSM were treated between April 2007 and December 2009 at the Radiation Oncology department of our institution. All patients underwent history and physical, and ophthalmic examinations. Orbital MRI was performed before and 6 weeks after treatment; thereafter every 6 months for the first 2 years. By previous stereotactic localization of the target, RT was delivered with 28 daily fraction of 1.8 Gy by multiple non coplanar arcs dynamically conformed by a micro multileaf-collimator. At diagnosis, in all 5 patients, visual acuity limitations of different degrees were found, while exophthalmos was present in 2, diplopy in 2, orbital pain in 1, and proptosis in 1. In all patients pre-treatment MRI showed an orbital mass involving the optic nerve. After radiotherapy, previous symptoms improved in all patients. However, after RT the MRI consistently showed a stationary status compared to the MRI before RT. At a median follow up of 26 months (range 9–37) all patients had a subjective and/or objective better visual performance than before RT without any evidence of disease progression. No late side effects were recorded. Accordingly to the current literature, our experience confirms the efficacy and the safety of FSRT in patients with orbital ONSM.

INTRODUCTION

Optic nerve sheath meningiomas (ONSM) are uncommon benign tumors of the optic nerve, originating in the optic nerve sheath and represent 1–2% of all meningiomas. ONSM is the second most common optic nerve tumor after glioma primarily occurring in middle-aged females.1,2 Rarely, it may affect both eyes (5% of cases) at the same time.2 The tumor arises from cells that surround the optic nerve. It is typically a slow growing tumor, virtually never lethal,3 but its growth may cause compression of the optic nerve and/or its vasculature. This causes progressive loss of vision and often blindness if not treated. Furthermore, there is concern that if untreated the tumor can eventually extend intracranially into the brain and cause other types of neurological damage.4–6)

Improved imaging modalities such as computed tomography (CT), and particularly magnetic resonance imaging (MRI) with fat saturation after the administration of gadolinium, can precisely detect and demarcate ONSM thus allowing an earlier diagnosis. Characteristics signs at imaging are enlargement of the optic canal, optic nerve thickening and enhancement, and calcification within the tumor.1,7)

Even if untreated, the overall tumor-related mortality has been reported to be 0%,21 consequently management should be conservative in most cases. Historically, treatment strategies employed in ONSM have been observation, surgery, radiation therapy, and combinations of the above. However, in ONSM patients, surgery has rarely improved visual prognosis while in most patients the excision of the
tumor presented no benefit and has even led to blindness. As a consequence, surgery is now generally only indicated in rare cases with blindness or severe proptosis, and in patients with intracranial extension that may compromise even contralateral vision.\textsuperscript{2,3,8,9} Thus, at present, for patients with progressive visual deterioration, radiotherapy is employed as the primary treatment option aiming at preserving functional vision.\textsuperscript{11} Nonetheless, some concerns about radio-induced toxicity does exist.

In recent years, as radiotherapy techniques have extensively evolved, including the development of three-dimensional conformal radiotherapy with intensity-modulated and stereotactic target localization and even three-dimensional proton radiotherapy, the possibility to apply high doses to smaller volumes reducing visual and neurologic complications is now widely available.\textsuperscript{10–20} Accordingly, fractionated radiotherapy, with doses ranging from 50 to 55 Gy, has become the first choice to cure ONSM and to preserve, at the same time, visual function.\textsuperscript{3,21}

Here, together with a brief review of the literature, we report our experience with fractionated stereotactic radiotherapy (FSRT) in the treatment of orbital optic nerve sheath meningioma with a particular focus on dosimetric aspects related to the radiation tolerance of the optic apparatus.

**PATIENTS AND METHODS**

Between April 2007 and December 2009 a total of five patients with diagnosis of orbital optic nerve sheath meningioma have been treated with exclusive radiotherapy at the Radiation Oncology department of our institution. The median age at the time of radiotherapy was 51 years (range 39–56). Patients were diagnosed with OSNM based on clinical presentation and imaging. Patients underwent history and physical, and ophthalmic evaluation before and after treatment. At diagnosis, in all 5 patients, visual acuity limitations of different degrees were found. Detailed patient characteristics and presenting symptoms are reported in Table 1.

For all patients a diagnostic contrast enhanced magnetic resonance imaging (MRI) was performed before and 6 weeks after treatment, thereafter every 6 months for the first 2 years. It must be noted that the follow-up protocol for ONSM is not established, however we used a schedule applied in our institution for brain neoplasms.

All patients underwent FSRT, and three-dimensional treatment planning was based on brain CT-scan performed in supine position using a stereotactic head frame. Scans were acquired using 2-mm slices of a multislices scanner. Also, an on purpose MRI-scan was performed using the same stereotactic head frame and with the patient in the treatment position. CT and MRI images were electronically transferred to the Focal Ease 4.2 CT Simulation software (Elekta, CMS Software) for stereotactic image fusion based on the localizer derived coordinate system\textsuperscript{22,23} and for target and critical organs (lenses, optic chiasm, retinæ, optic nerves, ocular globes) contouring. The retina was defined as the surface of the posterior two-thirds of the ocular globes.\textsuperscript{20}

For the optic nerves and optic chiasm delineation, we referred to Mayo et al.\textsuperscript{24} The gross tumor volume (GTV) included the tumor as visualized on CT- and MRI-scans; a 5-mm margin was added to the GTV for planning (planning target volume, PTV).

Stereotactic photon plans were designed on Ergo++ (Elekta) treatment planning system with 4 to 6 non coplanar arcs using 20 MV photon beams from a linear accelerator equipped with a dynamic micro-multileaf collimator. Sample beam arrangement is showed in Fig. 1.

Convolution 3D Pencil beam calculation algorithm and a 1 mm calculation grid were used. The prescription dose was specified at the centre of PTV. Arc weightings were adjusted so that the 95% isodose line encompassed at least the 95% of the PTV. A total 50.4 Gy in 28 daily fractions of 1.8 Gy was planned.

Time to event data was calculated from the first day of radiation treatment. Local progression was defined as tumor growth on repeated MRI after radiation treatment or increase of clinical symptoms.

### Table 1. Patients characteristics

| Patient | Age (y) | Sex | Side | Local. | Presenting symptoms | Visual Acuity |
|---------|---------|-----|------|-------|---------------------|--------------|
| 1       | 48      | M   | R    | Orbital, optic nerve canal and intracranial extension | Loss V. A. | 20/200 |
| 2       | 51      | F   | R    | Intraorbital | Loss V. A., diplopia, decreased color vision, campimetric alteration. | 20/60 |
| 3       | 54      | F   | L    | Intraorbital | Loss V. A. | 20/200 |
| 4       | 39      | F   | R    | Intraorbital | Loss V. A. Exophthalmos | 20/30 |
| 5       | 56      | M   | R    | Intraorbital | Loss V. A. Palpebral proptosis, diplopia, exophthalmos, orbital pain | 20/60 |

M: male; F: female; R: right, L: left, V.A.: Visual Acuity.
RESULTS

All patients were followed with clinical examination and MRI scans on a regular basis in our institution. Median follow-up was 26 months, ranging from 9 to 37 months.

Treatment characteristics

Median volume of GTV was 4.2 cm³ (range 1.9 to 6.8 cm³) and median volume of the PTV within the 95% isodose was 98% (range 97.6 to 99.8%). An example of transversal dose distributions and of dose volume histogram for one of the five patients is presented in Fig. 2. The median values of the maximum dose ($D_{\text{max}}$) and of the mean dose ($D_{\text{mean}}$) received by the critical structures are reported in Table 2. For all critical structures the $D_{\text{mean}}$ values were kept within tolerance values reported in the literature.²⁴,²⁵

Table 2. Median values of doses to critical structures of all 5 patients

| Critical Organs | $D_{\text{max}}$ (Gy) | $D_{\text{mean}}$ (Gy) | Tolerance Doses (Gy)²⁴,²⁵ |
|-----------------|-----------------------|------------------------|---------------------------|
| Lens            | Homolateral           | 5.8                    | 2.5                       | 10*                       |
|                 | Contralateral         | 0.5                    | 0.3                       |                           |
| Optic nerve     | Homolateral           | 52.4                   | 50.1                      | 55§                       |
|                 | Contralateral         | 10.4                   | 2.2                       |                           |
| Retina          | Homolateral           | 51.8                   | 43.8                      | 45*                       |
|                 | Contralateral         | 4.2                    | 0.6                       |                           |
| Ocular globes   | Homolateral           | 52.1                   | 27.2                      | ND                        |
|                 | Contralateral         | 7.1                    | 0.6                       |                           |
| Optic Chiasm    |                       | 44.5                   | 8.4                       | 55§                       |

$D_{\text{max}}$: maximum dose; $D_{\text{mean}}$: mean dose; ND: not defined.

*Rate of toxicity < 3% with $D_{\text{max}} < 55$ Gy.²⁴

Table 3. Individual outcomes

| Patient | Follow-up (months) | MRI outcome | Acute Side effects | Visual Acuity | Clinical outcome |
|---------|--------------------|-------------|--------------------|---------------|------------------|
| 1       | 37                 | ↔           | No side effects    | 20/200        | ↔                |
| 2       | 32                 | ↔           | Mild conjunctivitis| 20/30         | ↑                |
| 3       | 26                 | ↔           | No side effects    | 20/200        | ↔                |
| 4       | 9                  | ↔           | No side effects    | 20/30         | ↔                |
| 5       | 14                 | ↔           | Orbital pain       | 20/40         | ↑                |

↑: improved, ↔: stable.
Imaging and visual response

The MRI following radiation therapy, compared to the pre-treatment MRI, showed no progression of the disease with a morphologic stability of the orbital lesion. All patients referred a subjective improvement in the visual function. However, at ophthalmologic evaluation only 2 showed an improved visual acuity and 3 remained stable at the most recent follow-up (Table 3). All symptoms the patients had before treatment disappeared after radiotherapy.

Regarding acute side effects, one patient suffered of orbital pain for a short period after radiotherapy. One patient had a mild conjunctivitis. No late side effects were recorded. Individual outcomes of treatment are reported in Table 3.

DISCUSSION

ONSM is a tumor that arises from meningotheial cells of arachnoid villi and may affect any tract of the optic nerve from ocular globe to chiasm.\(^{21}\) Despite its no lethal behavior,\(^2\) the growth of the neoplasm can cause important visual alterations and disturbing symptoms such as proptosis, motility disturbance, orbital pain, and lid edema.\(^{26}\) The surgical removal of the lesion is able to radically cure ONSM, but generally at a very high functional cost, rarely ameliorating and often worsening the visual function.\(^{3,8,27}\) Radiosurgery for treating ONSM has been only sporadically described,\(^{28,29}\) may be due to the low tolerance of the optic nerve to single session radiosurgery.\(^{24,30}\) Hypofractionation with multisession CyberKnife radiosurgery has been also proposed.\(^{31}\)

At present, external beam radiotherapy and in particular FSRT is the treatment of choice for ONSM due to its efficacy in stopping meningioma growth, to the possible improvement of visual function, and to its low toxicity profile. In accordance with the literature it is possible to obtain a visual control rate of 91.5% using highly conformal three-dimensional radiotherapy techniques\(^{13}\) with doses ranging from 50 to 55 Gy. A summary of findings of recent published studies on the issue is shown in Table 4.

The study published by Andrews et al.\(^{18}\) is of particular interest for the retrospective comparison of the functional outcome with patients previously observed or treated by surgery, and for the evaluation in some patients of tumor response by \(^{111}\)In-octreotide scintigraphy. The authors reported a retrospective series of 30 patients with ONSM treated by FSRT and underlined the advantage of radiation therapy relatively to the other treatment options. They also showed that the metabolic activity of tumors, apparently morphologically stable at MRI evaluation, had consistently decreased.

Best visual outcome with radiotherapy was also shown by Turbin et al.\(^{21}\) in a study on 64 ONSM bearing patients of whom 18 were treated exclusively with radiotherapy. The authors finally suggest fractionated radiation therapy at doses of 50–55 Gy as first choice therapy for ONSM. Similar conclusion was proposed by Baumert et al.\(^{14}\) who treated 23 patients with ONSM by FSRT with a median dose of 50.4 Gy describing a visual preservation rate of 95%.

Recently, the largest published studies on radiotherapy in ONSM are those by Arvod et al.\(^{15}\) and Saeed et al.\(^{34}\) that report on 25 and 34 ONSM patients, respectively. In the former, 17 patients were treated with radiotherapy alone, 8 had surgery before radiotherapy and all patients were treated with highly conformal radiotherapy; noteworthy half of patients were treated with proton irradiation. With a median follow up of 30 months a 95% control of visual function and 3 cases of asymptomatic retinopathy were reported. In the latter, data were reported on patients treated with fractionated conventional or stereotactic radiotherapy and followed for a median period of 58 months. The authors report an overall visual control of 91% and 2 cases of retinopathy were observed in those patients treated with conventional radiotherapy.

Of note however, in all the above mentioned studies, the significant occurrence of late radiation toxicity. Of special concern, in our opinion, is the reported incidence of retinopathy in a proportion of patients ranging between 5% and 20%. Indeed, the retina, even when sophisticated irradiation techniques are used, depending on the tumor location, can often be located within a moderate to high dose volume. Another aspect to consider is the timing of retinopathy onset that seems to be not less than 3–4 years. This means that for many patients with ONSM treated by radiation and reported in the reviewed literature (Table 4), the assessment of retina late toxicity is to be considered provisional. Even taking into account this toxicity, the functional outcome appears to be better preserved with radiation therapy compared to other treatment options.\(^{21}\) However, the reviewed data confirm the observation made in the study by Parsons et al.\(^{35}\) in which the authors found that a total dose greater than 45 Gy and daily fraction greater than 1.9 Gy significantly increased the risk of retinopathy. Interestingly, a preliminary experience using multisession radiosurgery in 3 ONSM patients was described by Romanelli et al.\(^{31}\) The treatment consisted of 20 Gy delivered by CyberKnife with 4 daily fraction of 5 Gy. At a mean follow up of 37 months, control of disease, preservation of visual function, and no toxicity were reported.

In our limited series, we achieved tumor growth control in all 5 patients as evaluated by periodic MRIs showing consistently stable disease. Visual acuity ameliorated in 2 patients and was stable in 3. MRI assessment after treatment did not correlate with clinical improvement. Nevertheless, stability of imaging evaluation is a common finding in post-radiotherapy follow up of benign neoplasms,\(^{36,37}\) and in particular of meningioma,\(^{38}\) not representing a treatment failure.

Although no late side effects were reported and the mean dose values for all critical organs are kept within tolerance limits, the follow-up is too short to draw final conclusions.
about late toxicity, especially considering that in our series, may be due to the close proximity of the lesions to the globe, the homolateral retina or part of it is often in a relatively high dose volume, as shown in Fig. 1 and Table 2. In particular for one of the patients the overlap of the PTV with the retina resulted in a mean dose to the retina of 47 Gy. However the dose limit of 45 Gy is reported on a basis of a total dose delivered with a dose fraction of 1.9–2.0 Gy\textsuperscript{25,35} instead of 1.8 Gy as in our series.

Of notice, our study, unlike earlier studies, addresses the issues of contouring and dosimetric assessment for critical optic structures involved in ONSM irradiation. We believe that a more uniform approach in the literature regarding ONSM is needed to define risk of toxicity.

In conclusion, the results of our study are in agreement with the literature data about the effectiveness of FSRT compared to other treatment options in ONSM. However, a longer follow-up of our and other published series is necessary to better assess the late toxicity of this kind of treatment, so as to define a reliable cost/benefit ratio of the radiation treatment for this benign yet potentially vision-threatening tumor.

**REFERENCES**

1. Saeed P, et al (2003) Optic nerve sheath meningiomas. Ophthalmology 110: 2019–2030.
2. Dutton JJ (1992) Optic nerve sheath meningiomas. Surv Ophthalmol 37: 167–183.
3. Jeremc B and Pitz S (2007) Primary optic nerve sheath meningioma: stereotactic fractionated radiation therapy as an emerging treatment of choice. Cancer 110: 714–722.

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**Table 4. Literature overview**

| Series            | Year | N° of patients | RT characteristics | Dose (Gy) | Follow-up (months) | Visual Outcome | Toxicity |
|-------------------|------|----------------|---------------------|-----------|--------------------|----------------|---------|
| Becker et al.\textsuperscript{19} | 2002 | 15 (16 O.N.)   | FSRT                | 54        | 35                 | 1 O.N. improved 15 O.N. stable | no      |
| Liu et al.\textsuperscript{27}    | 2002 | 5              | FSRT                | 45–54     | 24                 | 4 improved 1 stable | no      |
| Andrews et al.\textsuperscript{18} | 2002 | 30 (33 O.N.)   | FSRT                | 50–54     | 22                 | 9 O.N. improved 11 O.N. stable | 13% (2 visual loss, 1 optic neuritis, 1 transient orbital pain) |
| Turbin et al.\textsuperscript{31} | 2002 | 18             | 3DCRT               | 40–55     | 100                | 8 improved | 33% (4 retinopathy, 1 iritis, 1 temporal lobe atrophy) |
| Narayan et al.\textsuperscript{10} | 2003 | 14             | 3DCRT               | 50.5–56   | 51                 | 5 improved 7 stable 2 worsened 16 improved | 35.7% (1 retinopathy, 2 iritis, 1 orbital pain, 1 dry eye) |
| Baumert et al.\textsuperscript{14} | 2004 | 23             | FSRT                | 45–54     | 20                 | 5 stable 1 worsened 6 improved | 5% (1 retinopathy) |
| Landert et al.\textsuperscript{15} | 2005 | 7              | FSRT                | 54        | 23                 | 2 improved 1 stable 1 worsened | 14% (1 acute eye lid edema) |
| Richards et al.\textsuperscript{16} | 2005 | 4              | FSRT                | 43.4–45   | 24                 | 6 improved | no      |
| Sitathane et al.\textsuperscript{32} | 2006 | 6              | FSRT                | 51.6–59.1 | 34                 | 4 improved 2 stable | 17% (1 vitreous hemorrhage) |
| Smee et al.\textsuperscript{33} | 2009 | 15 (17 O.N.)   | 3DCRT, FSRT, SRS    | 50–56 (20 SRS) | 86         | Not assessed (100% local control) | no      |
| Arvold et al.\textsuperscript{12} | 2009 | 22             | FSRT, PRT           | 45–59.4   | 30                 | 14 improved 7 stable 1 worsened | no      |
| Saeed et al.\textsuperscript{34} | 2010 | 34             | 3DCRT, FSRT         | 45–54     | 58                 | 14 improved 17 stable 3 worsened | 29% (5 dry eye, 2 retinopathy, 3 cataract) |

3DCRT: 3 dimensional conformal radiotherapy, FSRT: fractionated stereotactic radiotherapy, SRS: stereotactic radiosurgery, PRT: proton radiotherapy, O.N.: optic nerve.
4. Kennerdell JS, et al (1988) The management of optic nerve sheath meningiomas. Am J Ophthalmol 106: 450–457.
5. Ito M, et al (1988) Intraorbital meningiomas. Surgical management and role of radiation therapy. Surg Neurol 29: 448–453.
6. Clark WC, Theofilos CS and Fleming JC (1989) Primary optic nerve sheath meningiomas. Report of nine cases. J Neurosurg 70: 37–40.
7. Lindblom B, Truwit CL and Hoyt WF (1992) Optic nerve sheath meningioma. Definition of intraorbital, intracranial, and intracranial components with magnetic resonance imaging. Ophthalmology 99: 560–566.
8. Roser F, et al (2006) The role of surgery in meningiomas involving the optic nerve sheath. Clin Neurol Neurosurg 108: 470–476.
9. Eddleman CS and Liu JK (2007) Optic nerve sheath meningioma: current diagnosis and treatment. Neurosurg Focus 23: E4.
10. Narayan S, et al (2003) Preliminary visual outcomes after three-dimensional conformal radiation therapy for optic nerve sheath meningioma. Int J Radiat Oncol Biol Phys 56: 537–543.
11. Kurt G, et al (2010) Fractionated gamma knife radiosurgery for optic nerve tumors: a technical report. Turk Neurosurg 20: 241–246.
12. Arvold ND, et al (2009) Visual outcome and tumor control after conformal radiotherapy for patients with optic nerve sheath meningioma. Int J Radiat Oncol Biol Phys 75: 1166–1172.
13. Grant W 3rd and Cain RB (1998) Intensity modulated conformal therapy for intracranial lesions. Med Dosim 23: 237–241.
14. Baumert BG, et al (2004) Early improvements in vision after fractionated stereotactic radiotherapy for primary optic nerve sheath meningioma. Radiother Oncol 72: 169–174.
15. Landert M, et al (2005) The visual impact of fractionated stereotactic conformal radiotherapy on seven eyes with optic nerve sheath meningiomas. J Neuroophthalmol 25: 86–91.
16. Richards JC, Roden D and Harper CS (2005) Management of sight-threatening optic nerve sheath meningioma with fractionated stereotactic radiotherapy. Clin Experiment Ophthalmol 33: 137–141.
17. Liu JK, et al (2002) Optic nerve sheath meningiomas: visual improvement after stereotactic radiotherapy. Neurosurgery 50: 950–955; discussion 955–957.
18. Andrews DW, et al (2002) Fractionated stereotactic radiotherapy for the treatment of optic nerve sheath meningiomas: preliminary observations of 33 optic nerves in 30 patients with historical comparison to observation with or without prior surgery. Neurosurgery 51: 890–902; discussion 903–894.
19. Becker G, et al (2002) Stereotactic fractionated radiotherapy in patients with optic nerve sheath meningioma. Int J Radiat Oncol Biol Phys 54: 1422–1429.
20. Miralbell R, et al (2000) Optimizing radiotherapy of orbital and paraorbital tumors: intensity-modulated X-ray beams vs. intensity-modulated proton beams. Int J Radiat Oncol Biol Phys 47: 1111–1119.
21. Turbin RE, et al (2002) A long-term visual outcome comparison in patients with optic nerve sheath meningioma managed with observation, surgery, radiotherapy, or surgery and radiotherapy. Ophthalmology 109: 890–899; discussion 899–900.
22. Schad LR, et al (1992) Radiotherapy treatment planning of basal meningiomas: improved tumor localization by correlation of CT and MR imaging data. Radiother Oncol 25: 56–62.
23. Schlegel W, et al (1992) Computer systems and mechanical tools for stereotactically guided conformation therapy with linear accelerators. Int J Radiat Oncol Biol Phys 24: 781–787.
24. Mayo C, et al (2010) Radiation dose-volume effects of optic nerves and chiasm. Int J Radiat Oncol Biol Phys 76: S28–35.
25. Emami B, et al (1991) Tolerance of normal tissue to therapeutic irradiation. Int J Radiat Oncol Biol Phys 21:109–122.
26. Cantore WA (2000) Neuronal orbital tumors. Curr Opin Ophthalmol 11: 367–371.
27. Moyer PD, Golnik KC and Breneman J (2000) Treatment of optic nerve sheath meningioma with three-dimensional conformal radiotherapy. Am J Ophthalmol 129: 694–696.
28. Kwon Y, et al (2005) Visual changes after gamma knife surgery for optic nerve tumors. Report of three cases. J Neurosurg 102 Suppl: 143–146.
29. Klink DF, Miller NR and Williams J (1998) Preservation of residual vision 2 years after stereotactic radiosurgery for a presumed optic nerve sheath meningioma. J Neuroophthalmol 18: 117–120.
30. Stafford SL, et al (2003) A study on the radiation tolerance of the optic nerves and chiasm after stereotactic radiosurgery. Int J Radiat Oncol Biol Phys 55: 1177–1181.
31. Romanelli P, Wovra B and Muacevic A (2007) Multisession CyberKnife radiosurgery for optic nerve sheath meningiomas. Neurosurg Focus 23: E11.
32. Sitathance C, et al (2006) Stereotactic radiation therapy for optic nerve sheath meningioma; an experience at Ramathibodi Hospital. J Med Assoc Thai 89: 1665–1669.
33. Smee RI, Schneider M and Williams JR (2009) Optic nerve sheath meningiomas — non-surgical treatment. Clin Oncol (R Coll Radiol) 21: 8–13.
34. Saeed P, et al (2010) Primary radiotherapy in progressive optic nerve sheath meningiomas: a long-term follow-up study. Br J Ophthalmol 94: 564–568.
35. Parsons JT, et al (1994) Radiation retinopathy after external-beam irradiation: analysis of time-dose factors. Int J Radiat Oncol Biol Phys 30: 765–773.
36. Roeder F, et al (2010) Intensity modulated radiotherapy (IMRT) in benign giant cell tumors — a single institution case series and a short review of the literature. Radiat Oncol 5: 18.
37. Rudiger HA, et al (2010) Radiation therapy in the treatment of desmoid tumours reduces surgical indications. Eur J Surg Oncol 36: 84–88.
38. Milker-Zabel S, et al (2005) Fractionated stereotactic radiotherapy in patients with benign or atypical intracranial meningioma: long-term experience and prognostic factors. Int J Radiat Oncol Biol Phys 61: 809–816.

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