Movement disorders in astrocytomas of the basal ganglia and the thalamus

Joachim K Krauss, Fritz Nobbe, Ajay K Wakhloo, Mohsen Mohadjer, Werner Vach, Fritz Mundinger

Abstract
In a series of 225 patients with astrocytomas (grades I–IV) of the basal ganglia and the thalamus, 20 had a movement disorder. In all patients the histological diagnosis was verified by stereotactic biopsy. Tremor was observed in twelve patients, dystonia in eight, chorea in three, and chorea/ballismus and myoclonus in one. The tumour involved the thalamus in 16 patients. Corticospinal tract dysfunction was evident in 70% of the patients with movement disorders and in 73% of those without. Demographic, clinical, histological and neuroradiological data of the patients with a movement disorder were compared with the data of patients without. CT data yielded no differences with respect to the involvement of anatomical structures. Movement disorders were significantly associated with low-grade astrocytomas.

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Tumours of the thalamus and the basal ganglia are relatively rare. They are estimated to make up 1–2.5% of intracranial tumours. Most of these neoplasms are benign or malignant astrocytomas. Since craniotomy and partial resection was associated with high mortality and morbidity, stereotactic diagnostic and therapeutic techniques have been favoured in recent years.

As movement disorders (MDs) are often related to dysfunction or structural lesions of the basal ganglia, a high incidence in intrinsic tumours might be expected, but they seem to be infrequent. While in some series they are not even mentioned, they range from 1–7% in recent and from 4–33% in earlier series.

Since 1965, no large series has focused on the subject. To characterise this relatively uncommon cause of MDs, we reviewed the literature and retrospectively analysed a series of 225 patients with histologically verified astrocytomas of the basal ganglia and the thalamus. Among those we identified 20 with MDs (classified according to criteria published elsewhere) at the time of biopsy.

Results
Twenty (9%) patients with astrocytomas of the basal ganglia and the thalamus displayed an MD at the time of biopsy. The MD was the initial symptom of the tumour in three patients. The mean latency between onset of any symptoms and biopsy was 3 years. Various other clinical symptoms were found (table 1). Signs of raised intracranial pressure at biopsy were evident in patients 2, 5, 6, 8, and 15. Obstructive hydrocephalus at biopsy was found in patients 5, 6, and 8. Five patients had received ventriculoatrial or ventriculoperitoneal shunts before stereotactic biopsy. Two had no MD before the shunt operation, in two others shunting did not alter the MD, and in patient 14 hemiparesis improved while tremor did not.

CT or additional MRI findings were obtained in 18 of the 20 patients (two had died before 1975). The tumour involved the thalamus in 16. Downward extension to the subthalamicum or the upper midbrain varied. This could not always be determined exactly due to the compressive effect of the tumour on adjacent structures or due to peritumoural oedema, or because some patients had had only axial CT scans. In 15 patients the tumour

tactic biopsy. Fifteen patients were operated on before 1975 and 210 between 1975–86. The patients were referred from other neurosurgical and neurological clinics to the department of stereotaxy. CT scans, (available in our institution since 1975) and also MRI were later obtained before biopsy. In patients operated upon before 1975, CT was performed as soon as it became available. The patients were followed up at various times, with latest follow-ups in 1989, 90 or 91. Patients’ clinical reports, the register of a tumour data bank, and film recordings were reviewed to identify 20 patients with MDs.

Stereotactic biopsy and interstitial radiotherapy were performed using the Riechert-Mundinger system as described elsewhere. The histological diagnosis was made using intraoperative smear preparations by methylene blue staining and confirmed with paraffin sections. The tumours were graded according to the classification of the World Health Organisation (WHO).

Demographic, clinical and histopathological data of patients with an MD were compared with those of patients without. For statistical analysis Fisher’s exact test, the Wilcoxon, and the Chi-square test were applied.

Methods
This study is based on a retrospective evaluation of 225 patients with low- or high-grade astrocytomas of the basal ganglia and the thalamus histologically confirmed by stereotactic biopsy.
Table 1 Clinical features, histological grading and neuroradiological findings of 20 patients with movement disorders in basal ganglia and thalamic astrocytomas

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex/Age at Biopsy</th>
<th>Diagnosis</th>
<th>Other Clinical Symptoms at Biopsy</th>
<th>Hemiparesis</th>
<th>Grade of Astrocytoma</th>
<th>Brain Imaging Study Results</th>
<th>Tumour Volume cm³</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M/25/13</td>
<td>Tremor, 3-4 Hz</td>
<td>R hypacusis, nystagmus</td>
<td>—</td>
<td>I</td>
<td>R thalamus, cyst extending to midbrain (circumscribed)</td>
<td>~25</td>
</tr>
<tr>
<td>2</td>
<td>F/3/3</td>
<td>Tremor, 3-5 Hz</td>
<td>R optic atrophy, 3rd nerve palsy, dysarthria, gait ataxia</td>
<td>—</td>
<td>II</td>
<td>R thalamus, internal capsule, pallidum (circumscribed)</td>
<td>~22</td>
</tr>
<tr>
<td>3</td>
<td>M/50/44</td>
<td>Tremor, 3-4 Hz</td>
<td>Impaired memory, seizures</td>
<td>L</td>
<td>II-III (5m)</td>
<td>R frontomedial, basal, septum pellucidum, corpus callosum, caudate (diffuse)</td>
<td>~50</td>
</tr>
<tr>
<td>4</td>
<td>F/4/43</td>
<td>Chorea face, bilateral</td>
<td>Impaired memory, obstructive hydrocephalus, Parinaud's syndrome, lethargy</td>
<td>—</td>
<td>IV</td>
<td>R + L posteroventral thalamus, 3rd ventricle, corpus pineale, splenium corporis callosi (circumscribed)—upper brainstem compression</td>
<td>~20</td>
</tr>
<tr>
<td>5</td>
<td>F/7/70</td>
<td>Tremor, 3-4 Hz</td>
<td>Impaired memory, seizures, aphasia</td>
<td>—</td>
<td>II</td>
<td>R thalamus, internal capsule, pallidum, putamen, corpus callosum, septum pellucidum, r frontal + temporal, f frontal (diffuse)</td>
<td>&gt;100</td>
</tr>
<tr>
<td>6</td>
<td>M/37/31</td>
<td>Dystonia</td>
<td>Lethargy, Neurofibromatosis type I</td>
<td>L</td>
<td>I</td>
<td>R pallidum, subthalamicum, internal capsule (circumscribed) + quadrigeminal plate + bilateral pons + cerebellum (diffuse)</td>
<td>~12</td>
</tr>
<tr>
<td>7</td>
<td>F/1/10</td>
<td>Dystonia</td>
<td>Impaired memory, motor apraxia</td>
<td>L</td>
<td>I</td>
<td>R thalamus (circumscribed)</td>
<td>~15</td>
</tr>
<tr>
<td>8</td>
<td>M/12/9</td>
<td>Dystonia</td>
<td>Seizures, headache</td>
<td>L</td>
<td>II</td>
<td>L basal ganglia + hemisphere, corpus callosum (diffuse, multifocal)</td>
<td>&gt;100</td>
</tr>
<tr>
<td>9</td>
<td>M/42/41</td>
<td>Tremor, 4 Hz</td>
<td>Impaired memory, aphasia, behavioural disorder, seizures, r hemihypaesthesia</td>
<td>R</td>
<td>I</td>
<td>L thalamus, subthalamicum, caudate, putamen, pallidum, internal capsule, corona radiata (circumscribed)—compression of thalamus</td>
<td>~40</td>
</tr>
<tr>
<td>10</td>
<td>F/25/25</td>
<td>Dystonia</td>
<td>Impaired memory, aphasia, hemiparesis</td>
<td>L</td>
<td>I</td>
<td>L pallidum, putamen, corona radiata (circumscribed)—compression of thalamus</td>
<td>~18</td>
</tr>
<tr>
<td>11</td>
<td>M/15/13</td>
<td>Tremor, 4-5 Hz</td>
<td>Impaired memory, aphasias,</td>
<td>R</td>
<td>I</td>
<td>R thalamus, subthalamicum, internal capsule, corona radiata, cr cerebri (circumscribed)</td>
<td>~25</td>
</tr>
<tr>
<td>12</td>
<td>F/1/9</td>
<td>Chorea</td>
<td>Behavioural disorder, r hemihypaesthesia</td>
<td>R</td>
<td>II</td>
<td>R thalamus, internal capsule, medial caudate, posteroventral pallidum (circumscribed)</td>
<td>~18</td>
</tr>
<tr>
<td>13</td>
<td>F/2/3</td>
<td>Dystonia</td>
<td>Precocious puberty</td>
<td>L</td>
<td>I</td>
<td>R thalamus, internal capsule, corona radiata, cr cerebri (circumscribed)</td>
<td>~25</td>
</tr>
<tr>
<td>14</td>
<td>F/12/3</td>
<td>Dystonia</td>
<td>Seizures, gait ataxia</td>
<td>L</td>
<td>II</td>
<td>R thalamus, quadrigeminal plate (circumscribed)</td>
<td>~25</td>
</tr>
<tr>
<td>15</td>
<td>M/16/15</td>
<td>Dystonia + Chorea</td>
<td>Obtundation, 3rd nerve palsy, cachexia</td>
<td>—</td>
<td>IV</td>
<td>R thalamus, upper brainstem (≥ CT)</td>
<td>&gt;30</td>
</tr>
<tr>
<td>16</td>
<td>F/6/59</td>
<td>Tremor, high-frequency bilateral, r &gt; l fingers + eyelids</td>
<td>Psychosis, lethargy, dysarthria</td>
<td>R</td>
<td>II</td>
<td>L anterior-ventrolateral thalamus subthalamicum, internal capsule, cr cerebri (circumscribed)</td>
<td>~7</td>
</tr>
<tr>
<td>17</td>
<td>F/3/13</td>
<td>Dystonia</td>
<td>Impaired memory, dystonia,</td>
<td>R</td>
<td>I</td>
<td>L hypothalamus, thalamus extending over midline and to midbrain (≥ CT)</td>
<td>~25</td>
</tr>
<tr>
<td>18</td>
<td>F/3/2</td>
<td>Myoclonus</td>
<td>Behavioural disorder, anorexia, diminished vision</td>
<td>R</td>
<td>—</td>
<td>L hypothalamus, thalamus (circumscribed)</td>
<td>~20</td>
</tr>
<tr>
<td>19</td>
<td>F/5/5</td>
<td>Dystonia</td>
<td>Impaired memory, hemiparesis, movement,</td>
<td>R</td>
<td>I</td>
<td>L subthalamicum, thalamus, caudate, internal capsule, pallidum, putamen, insula, uncus (circumscribed)</td>
<td>~20</td>
</tr>
<tr>
<td>20</td>
<td>F/12/10</td>
<td>Tremor, 3-4 Hz</td>
<td>Impaired memory, behavioural disorder, diminished vision</td>
<td>R</td>
<td>—</td>
<td>L thalamus, subthalamicum, cr cerebri (circumscribed)</td>
<td>~16</td>
</tr>
</tbody>
</table>

M = male, F = female, KPS = Karnofsky Performance Scale, R = right, L = left. Tremor is qualified as: rest—tremor at rest, post—postural tremor and int—intention tremor.
thalamus. In the tenth (case 11) the tumour was predominantly located in the pallidum and putamen, sparing the thalamus, but nevertheless exerted considerable compression on the thalamus. In patient 3 the tumour was located mainly in the red nucleus and to a lesser degree in the postero-basal thalamus (fig 3). In patient 14 the astrocytoma extended from the thalamus to the quadrigeminal plate (fig 4).

Hemidystonia or hemiathetosis was seen in eight patients, in five of whom it was particularly marked on action. The arm was affected in all these patients, with additional involvement of the leg in two. Patients 2 and 8 with involvement only of hand and fingers had an astrocytoma confined to the thalamus. In patients 10 and 19 the tumour mainly involved the caudate and putamen, to a lesser degree the thalamus. In cases 13, 15, and 17 the main tumour mass was found in the thalamus, whereas in case 7 the thalamus only was compressed.

Choreatic MDs were seen in four patients. Two had hemichorea, one left hemichorea involving the face markedly on the left side, and one hemichorea/hemiballismus. Patients 13 and 15 presented with hemichorea and dystonia appearing as "choreoathetosis". Hemichorea was associated in two patients with involvement of the head of the caudate. In the patient with hemichorea/hemiballismus, the thalamic tumour involved the subthalamic nucleus. Choreatic hyperkinesias disappeared in all patients in the long term.

The following data were analysed comparatively for patients with and without MDs. The female/male ratio was 1:2:1 (11 females/9 males) for MD patients and 1:1 for those without (102/103). The 20 astrocytomas of the MD patients and the 205 astrocytomas of those without were distributed according to histological grading as follows: WHO I–12 (60%)/41 (20%); WHO II–6 (30%)/71 (35%); WHO III–0/56 (27%); and WHO IV–2 (10%)/37 (18%). In patient 16 the tumour was graded histologically as an astrocytoma WHO II, however, focal anaplastic transformation could not be excluded. The frequency of MDs in patients with low-grade (I + II) astrocytomas was 15%, in patients with high-grade (III + IV) astrocytomas 2-1%. This difference was statistically significant (p = 0.0017). The mean age of patients with MDs at the time of biopsy was lower (24 years) than that of the patients without (36 years), however, the proportion of low-grade astrocytomas was higher in MD patients and the mean age correlated with histological grading as follows: WHO I (mean age in years, MD patients/patients without)—15/20; WHO II–36/35; WHO III—/42; and WHO IV–43/47. There was also a trend for the mean age in patients with astrocytomas...

Figure 1 Axial enhanced CT scans of patient 9 with right resting, postural and intention 4 Hz tremor showing a diffusely spreading WHO grade II astrocytoma of the left basal ganglia and hemisphere extending contralaterally via the corpus callosum.

Figure 2 Enhanced CT scans of patient 16 with essential tremor showing a WHO grade II astrocytoma (focal anaplastic transformation could not be excluded) located predominantly in the left anterior/ventrolateral thalamus and the genu of the internal capsule.

Figure 3 Axial enhanced CT scans (lower row) with sagittal (upper row) and coronal (middle row) reconstructions of patient 3 with left resting, postural and intention 3–4 Hz tremor showing a relatively small circumscribed WHO grade I astrocytoma, predominantly located in the right red nucleus and posterobasal thalamus.
WHO I to be lower in MD patients (15 years) compared with those without (20 years).

Analysis of the CT data of patients with and without MD yielded no difference concerning the involvement of different anatomical structures. Comparing tumour volumes there was a different distribution, which reached statistical significance: MD patients more often had tumour volumes ranging from 16 to 25 cc (55%) than those without (17%). However, large tumours were found more often in astrocytomas WHO III and IV.

Seventy per cent of patients with MDs and 73% of patients without had a hemiparesis of variable degree contralateral to the tumour. In the astrocytoma WHO I group there was a tendency for MD patients to present more often with hemiparesis (75%) than those without (61%). The median Karnofsky Performance Score\(^\text{20}\) (KPS) in MD patients was 60. Considering the interrater variability the distribution of the KPS of patients with and without MD was similar.

**Discussion**

The frequency reported for MDs in patients with tumours of the basal ganglia and the thalamus varies considerably. It ranged from 1 to 33%\(^\text{1-7 9 10 21-24}\) (table 2) in previous studies

and our study was 9%. However, in some studies a histological diagnosis was not established in all patients and different histological diagnoses were grouped together without specifying separately those with astrocytomas, so that an exact comparison cannot be made. Only three series on tumours of the basal ganglia and the thalamus which mentioned patients with movement disorders dealt with histologically verified astrocytomas for the total study population or a defined subgroup.\(^\text{7 10 21}\) Since the involvement of the basal ganglia and thalamus in astrocytomas is different from that in other neoplasms, it may be better to consider the former tumours separately. The incidence of MDs seems to be generally lower in recent than in earlier studies. This might be explained by the availability of CT and MRI which also reveal smaller and oligosymptomatic tumours, and by the use of stereotactic techniques to establish an earlier histological diagnosis. We chose biopsy as the timepoint for calculating the incidence of MDs, since later MDs may be altered by, or even result from, therapeutic measures.

Previous series on basal ganglia and thalamic tumours differ considerably with respect to the proportion of low- or high-grade astrocytomas in MD patients. While some series report that the majority of patients had "malignant gliomas"\(^\text{22}\) or glioblastomas,\(^\text{23}\) others stress that most of the tumours producing "extrapyramidal" symptoms grow "very or rather" slowly.\(^\text{24}\) In our study MDs were found significantly more often in low-grade astrocytomas.

Although well documented cases of basal ganglia and thalamic tumours presenting with MDs and ipsilateral hemiparesis had been published earlier,\(^\text{21 26}\) sparing of the corticospinal tract was regarded as essential for MDs to occur.\(^\text{25 26}\) In contrast, our data show that tremors as well as dystonia and chorea may be associated with a mild or moderate degree of hemiparesis. Comparing the patients with and without MDs we did not find a significant difference in the frequency of corticospinal tract dysfunction.

Unlike previous series and case reports on histologically verified astrocytomas of the basal ganglia and the thalamus the distribution of MDs differs in our study\(^\text{2 7 8 10 12 21 23 25-41}\) (table 3). We observed comparatively more cases with dystonia and chorea. In patients with dystonia the thalamus was involved more frequently than the caudate or putamen. However, there may be secondary putaminal involvement due to the mass effect of the tumour. Clinico-pathological correlations reported previously demonstrate primary lesions in caudate or putamen in the majority of cases with secondary dystonia.\(^\text{12 42 43}\) Chorea is more common in the basal ganglia as a whole, and it is more frequent in our patients with dystonia of the thalamus. This may be due to the mass effect of the tumour. Clinico-pathological correlations reported previously demonstrate primary lesions in caudate or putamen in the majority of cases with secondary dystonia.\(^\text{12 42 43}\) Chorea is more common in the basal ganglia as a whole, and it is more frequent in our patients with dystonia of the thalamus. This may be due to the mass effect of the tumour.
Table 3 Movement disorders in patients with histologically verified astrocytomas of the thalamus and the basal ganglia: literature review and present series

<table>
<thead>
<tr>
<th>Movement Disorder</th>
<th>Present Study</th>
<th>Literature Review</th>
<th>By first Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tremor/Parkinsonism</td>
<td>12</td>
<td>47</td>
<td>Alexander (1), Arseni (5), Bailey (1), Brzezicki (1), Cheek (3), Coers (1), Garcia (1), Globus (2), Kelly (1), Kuhali (1), McGirt (1), Meffert (1), Ody (3), Polyzoides (1), Roth (5), Sciarra (5), Smith (1), Tolosa (15)²,²⁴,²⁵,²⁹,³¹,³²,³³,³⁴,³⁶,³⁸,⁴⁰,⁴¹</td>
</tr>
<tr>
<td>Myoclonus</td>
<td>1</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Chorea/Ballismus</td>
<td>1</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Chorea</td>
<td>3</td>
<td>1</td>
<td>Alexander (1)⁹, Arseni (2), Millichap (1), Ody (1), Parker (1), Roth (1)²,²⁴,²⁵,²⁹,³¹,³²,³³,³⁴,³⁶,³⁸,⁴⁰,⁴¹</td>
</tr>
<tr>
<td>Dystonia/Athetosis</td>
<td>8</td>
<td>7</td>
<td>Chorobiski (1), Marsden (2), Masten (1), Narbona (1), Sciarra (1), Urcelgia (1)²⁴,²⁵,²⁴,²⁵,²⁹,³¹,³²,³³,³⁴,³⁶,³⁸,⁴⁰,⁴¹</td>
</tr>
</tbody>
</table>

We think that the clinical and pathological correlations in thalamic and basal ganglia astrocytomas should be made only with certain reservations. Interaction of different mechanisms may or may not lead to clinical symptoms. Functional lesions of the thalamus may be primarily induced by tumours or by other pathological conditions.

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