Movement disorders in astrocytomas of the basal ganglia and the thalamus

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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.¹⁻⁴ Most of these neoplasms are benign or malignant astrocytomas.⁵⁻⁷ Since craniotomy and partial resection was associated with high mortality and morbidity, stereotactic diagnostic and therapeutic techniques have been favoured in recent years.^{7,8}

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,⁹ they range from 1-7% in recent^{5,7} and from 4-33% in earlier series.^{3,4}

Since 1965,¹⁰ no large series has focused on the subject. To characterise better 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)¹¹⁻¹⁶ at the time of biopsy.

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 stereo-

tactic biopsy. Fifteen patients were operated on before 1975 and 210 between 1975-86. The patients were referred from other neurosurgical or 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-Munding system as described elsewhere.^{17,19} The histological diagnosis was made using intraoperative smear preparations by methylene blue staining and was 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.

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 subthalamus 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

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Table 1 Clinical features, histological grading and neuroradiological findings of 20 patients with movement disorders in basal ganglia and thalamic astrocytomas

Case	Sex/Age at Biopsy/Age at Onset	Preop. KPS	Movement Disorder	Other Clinical Symptoms at Biopsy	Hemiparesis	Grade of Astrocytoma	Brain Imaging Study Results	Tumour Volume cm ³
1	M/25/13	60	Tremor, 3–4 Hz l arm: rest, post, int	R hypacusis, nystagmus	—	I	R thalamus, cyst extending to midbrain (circumscribed)	~25
2	M/3/3	80	Tremor, 3–5 Hz l arm > leg: post, int Athetosis, l hand: rest Head tilt	Papilloedema	—	I	R thalamus, internal capsule, pallidum (circumscribed)	~22
3	M/50/44	60	Tremor, 3–4 Hz l arm > leg: rest, post, int	R optic atrophy, r 3rd nerve palsy, dysarthria, gait ataxia	L	I	R postero-basal thalamus, nucleus ruber (circumscribed)	~1.5
4	F/49/43	60	Chorea face, bilateral l > r l arm > leg	Impaired memory, seizures	L	II	R frontomedial, basal, septum pellucidum, corpus callosum, caudate (diffuse)	>50
5	F/70/70	40	Parkinsonism hypokinesia, bilateral rigidity + tremor: rest, post, int	Impaired memory, obstructive hydrocephalus, Parinaud's syndrome, lethargy	—	II–III (8m) IV	R + L posteromedial thalamus, 3rd ventricle, corpus pineale, splenium corporis callosi (circumscribed)—upper brainstem compression	~20
6	M/37/31	60	Tremor, 4–5 Hz l arm: rest, post, int	Impaired memory, seizures, aphasia	—	II	R thalamus, internal capsule, pallidum, putamen, corpus callosum, septum pellucidum, r frontal + temporal, l frontal (diffuse)	>100
7	F/11/10	60	Dystonia l arm: rest, on action	Lethargy, Neurofibromatosis type I	L	I	R pallidum, subthalamus, internal capsule (circumscribed) + quadrigeminal plate + bilateral pons + cerebellum (diffuse)	~12
8	M/12/9	70	Dystonia l hand: on action Tremor, 4–5 Hz l arm: post, int	Impaired memory, obstructive hydrocephalus, l hemihyphaesthesia, l 6th nerve palsy, Parinaud's syndrome	L	I	R thalamus (circumscribed)	~15
9	M/42/41	70	Tremor, 4 Hz r arm: rest, post, int	Seizures, headaches	—	II	L basal ganglia + hemisphere, corpus callosum (diffuse, multifocal)	>100
10	M/25/25	60	Dystonia r arm: rest, on action	Impaired memory, aphasia, behavioural disorder, seizures, r hemihyphaesthesia	R	I	L thalamus, subthalamus, caudate, putamen, pallidum, internal capsule, corona radiata (circumscribed)	~40
11	M/15/13	70	Tremor, 4–5 Hz r arm: rest, post, int	Impaired memory	R	I	L pallidum, putamen, corona radiata (circumscribed)—compression of thalamus	~18
12	F/11/9	70	Chorea r arm > leg marked on action	Behavioural disorder, r hemihyphaesthesia	R	II	L subthalamus, internal capsule, medial caudate, posterobasal pallidum (circumscribed)	~18
13	F/10/9	60	Dystonia l arm > leg Chorea-Ballismus l arm > leg	Precocious puberty	L	I III (4y)	R subthalamus, thalamus, internal capsule, corona radiata, crus cerebri (circumscribed)	~25
14	F/12/3	60	Tremor, 3–5 Hz l arm: rest, post, int	Seizures, gait ataxia	L	II	R thalamus, quadrigeminal plate (circumscribed)	~25
15	M/16/15	40	Dystonia + Chorea l arm > leg > face: rest, on action	Obtundation, 3rd nerve palsy, cachexia	L	IV	R thalamus, upper brainstem (Ø CT)	>30
16	F/62/59	60	Tremor, high-frequency bilateral, l > r fingers + eyelids	Psychosis, lethargy, dysarthria	R	II	L anterior-ventrolateral thalamus subthalamus, internal capsule, crus cerebri (circumscribed)	~7
17	F/13/11	70	Dystonia, r arm: rest, on action Myoclonus, r shoulder	Dysarthria	R	I	L hypothalamus, thalamus extending over midline and to midbrain (Ø CT)	~25
18	F/3/2	70	Tremor, 3–4 Hz r arm: int	Behavioural disorder, anorexia, diminished vision l eye	—	I	L hypothalamus, thalamus (circumscribed)	~20
19	F/5/5	60	Dystonia, r arm: rest, on action	Behavioural disorder, diminished vision l eye	R	I	L subthalamus, thalamus, caudate, internal capsule, pallidum, putamen, insula, uncus (circumscribed)	~20
20	F/12/10	60	Tremor, 3–4 Hz r arm: int	Impaired memory, behavioural disorder, diminished vision l eye	R	I	L thalamus, subthalamus, crus cerebri (circumscribed)	~16

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.

appeared to be more or less circumscribed. In patients 6 and 9 (fig 1) the neoplasm involved the basal ganglia and large parts of the hemisphere diffusely. The tumour volume ranged from 1.5 to more than 100 cm³. It was located in the contralateral basal ganglia in patients with unilateral MDs.

Twelve patients had tremor. Only patient 5 had Parkinsonism with bilateral rigidity, resting and postural tremor, akinesia, masked face, and loss of postural reflexes, though tremor was also present on action. In this case the tumour was located in the midline and involved both thalami, but exerted marked compression on the midbrain. Patient 16 had a high-frequency tremor of fingers and eyelids,

presumably essential tremor, which was less severe on the right, slightly hemiparetic side contralateral to the tumour. The tumour predominantly involved the anterior and ventrolateral thalamus (fig 2). Ten patients had unilateral tremor, which in two was only present on action, in two others on action and on maintaining antigravity posture, and in six a combined resting-postural-intention tremor was found. These tremors had a frequency of 3 to 5 Hz. The slight increase in tone in some patients with an ipsilateral hemiparesis had features of spasticity rather than of rigidity. Tremor in these patients was not associated with akinesia or impaired postural reflexes. In nine of these patients the tumour involved the

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

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

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.

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 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 4 Sagittal SE 1040/40 and axial SE 1200/80 MR images of patient 14 with left resting, postural and intention 3–5 Hz tremor reveal a WHO grade II astrocytoma, predominantly located in the right thalamus and the quadrigeminal plate.

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 ccm (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 Score²⁰ (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%^{1–7 9 10 21–24} (table 2) in previous studies

Table 2 Large series of patients with tumours of the thalamus and the basal ganglia

Literature Review (by first author)	Number of patients with movement disorders/Total number of patients	
Ody, 1932	4/25	16%
Globus, 1942	3/21 *	14%
Arseni, 1958	5/50 *	10%
Roth, 1958	7/34 *	21%
McKissock, 1958	8/24 **	33%
Tovi, 1961	2/49 */**	4%
Tolosa, 1965	15/61	25%
Cheek, 1966	2/51 */**	"rare"
Paillas, 1970	13/55 *	23%
Hirose, 1975	4/18 **	22%
Bernstein, 1984	4/60 */**	7%
Beks, 1987	2/27 */**	?
Kelly, 1989	1/72	1%
Krauss, 1992	20/225	9%

*: Series with different types of histological diagnoses, but mainly astrocytomas I–IV.
**: Histological diagnosis was not obtained in all patients.

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.^{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"²³ or glioblastomas,¹⁰ others stress that most of the tumours producing "extrapyramidal" symptoms grow "very or rather" slowly.²⁵ 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^{21 26} sparing of the corticospinal tract was regarded as essential for MDs to occur.^{23 25} 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^{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. Clinicopathological correlations reported previously demonstrate primary lesions in caudate or putamen in the majority of cases with secondary dystonia.^{12 42 43} Choreatic hyperkinesias, which were transient in our patients, seem to be rare in basal ganglia tumours. We found only one case in the literature and six of "choreoathetosis". Hemiballismus was described in a cystic midbrain glioma,¹⁴ but as far as we know, not in thalamic or basal ganglia astrocytomas.

Tremor was the most frequent MD in our

Table 3 Movement disorders in patients with histologically verified astrocytomas of the thalamus and the basal ganglia: literature review and present series

Movement Disorder	Present Study Number of Patients	Literature Review Number of Patients	By first Author (Number of Patients)
Tremor/Parkinsonism	12	47	Alexander (1), Arseni (5), Bailey (1), Brzezicki (1), Cheek (3), Coers (1), Garcin (1), Globus (2), Kelly (1), Kulali (1), McGirr (1), Mettler (1), Ody (3), Polyzoidis (1), Roth (5), Sciarra (3), Smyth (1), Tolosa (15) ^{2 7 8 10 21 23 25 35}
Myoclonus	1	—	—
Chorea/Ballismus	1	—	—
Chorea	3	1	Alexander (1) ²⁷
"Choreoathetosis"	(2)	6	Arseni (2), Millichap (1), Ody (1), Parker (1), Roth (1) ^{21 23 26 36 37}
Dystonia/Athetosis	8	7	Chorobski (1), Marsden (2), Masten (1), Narbona (1), Sciarra (1), Urechia (1) ^{2 34 38 41}

series as well as in the literature. Interpretation of the literature is limited, since in the 47 cases of basal ganglia and thalamic astrocytomas we collected from 1927–91 (table 3) data concerning frequency and characteristics of the tremor were not always reported and the interpretations have differed considerably. While McKissock pointed out that the tremor was not "parkinsonian" in any of the eight cases he presented,³ it was stated to be "parkinsonian" in all patients with tremor in other series.^{6 10} As was pointed out by Ody, "rigor" or "rigidity" in some patients with space occupying cerebral tumours may have been more a sign of partial decerebration than a Parkinsonian feature.²¹ Although the tremor observed in some patients of this study had a frequency corresponding to Parkinsonian tremor, it differed in other characteristics. It was present on, or even only on, intended movements and was not accompanied by rigidity. Only one patient had tumour-induced Parkinsonism. Categorisation of these tremors raises certain difficulties.^{16 44 45} Tremors have been denominated according to proven or assumed distinct lesions generating different types of tremor. In our opinion, however, denotation following clinical phenomena should not have topological or anatomical implications: "midbrain tremor", "rubral tremor" or "cerebellar tremor at rest". Thus we would not label the tremor in our patients as "thalamic", although there is evidence that tremor-driving impulses might originate in the thalamus.⁴⁶ Furthermore, as stated above, axial CT scans may be inadequate to determine exactly the direct or indirect extension of involvement of the upper midbrain. Involvement of thalamic projections from cerebellar nuclei might be the common denominator for postural and intention tremors, with an additional structural or functional lesion of the red nucleus and/or the substantia nigra and their projections being necessary to produce rest tremor. Tumour-induced Parkinsonism seems to be more common in extra-axial than in intrinsic tumours.³³

We could not find previous reports on the effect of thalamic or basal ganglia tumours on pre-existing movement disorders. In patient 16, who presumably had essential tremor, the

rightsided tremor may have been less marked because of slight hemiparesis. However, it is also conceivable that it was reduced due to the tumour being located mainly in the left ventrolateral thalamus exerting a functional suppression.

We think that clinicopathological 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 structures involved primarily by the tumour might result from "internal" compression of neurons or neural pathways or from altered metabolism by reduced vascular supply. But tumours or a peritumoural oedema may also induce symptoms by "external" compression of adjacent structures or their vascular supply.⁴⁷ The question has been raised as to why tumours of the basal ganglia are not associated with MDs more often. The situation becomes more complex, since these tumours may also involve structures of the basal ganglia which are known to abolish or to reduce MDs when lesioned (predominantly the ventrolateral thalamus, the zona incerta, the pulvinar thalami and the internal pallidum and their fibre connections). Again these structures may be functionally damaged by "external" compression.

Neither necropsy findings nor the present comparative analysis of in vivo CT data showed topographical differences in thalamic and basal ganglia astrocytomas that do or do not cause MDs. However, tumours which are apparently identical on imaging studies may nevertheless differ in functional terms. These aspects might be at least partly explained using models such as that of parallel distributed processing⁴⁸ provided more detailed information were available. However, the functional state of the basal ganglia in brain tumours causing MDs has only rarely been studied. In a case of a left frontal meningioma with right hemi-Parkinsonism PET indicated impaired oxygen metabolism and tissue perfusion in the striatopallidal region.⁴⁹ In one case of Parkinsonism secondary to a craniopharyngioma biochemical findings on necropsy revealed considerably reduced striatal catecholamines and severely decreased dopamine receptor binding sites in the caudate.⁵⁰ Further functional studies are needed and should provide deeper insights into the pathophysiological processes underlying the generation of movement disorders.

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