SHORT REPORT

Clinical and CT correlates in the diagnosis of intracranial tumours

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Abstract

The correlation between clinical and CT findings in cerebral tumours was prospectively studied in 1191 consecutive referrals for cerebral CT. CT revealed a mass lesion in 51 cases (4.3%): 32 neoplasms, five haematomas and one abscess. The diagnostic specificity of CT for neoplastic tumours was 86% (32 of 37). The clinical suspicion of a cerebral neoplasm was correct in 25 cases (78%) and the clinical localisatory hypothesis was correct in 20 cases (63%) of the neoplasms. A cerebral tumour was found in 5% (11 out of 226) of patients investigated for their first seizure and in 1% (two of 207) investigated for headache without clinical signs.

When MRI is not available CT is the method of choice for investigation of patients with symptoms or signs suggesting a space occupying intracranial (IC) lesion. The reported sensitivity of CT in brain tumours has been more than 90%, particularly when contrast enhancement has been used.¹⁻³ There is, however, disproportion between the supply of CT scanners and the demand for investigations.^{4,5} Early detection of a tumour does not necessarily lead to intervention and equivocal CT findings may cause unnecessary alarm and ethical problems which may lead to difficult decisions. CT may also fail to visualise a tumour and other diseases may simulate the appearance of a neoplasm.^{3,6-8}

Despite the importance of clinical assessment, little attention has been paid to the correlation between neurological symptoms and signs and CT findings in routine practice. We carried out a prospective survey of 1191 consecutive neurological patients referred for cranial CT during a one year period. The correlation between clinical evaluation and cranial CT was analysed, first, using information available at the time of referral for CT and, second, using all the clinical, CT and ancillary information accumulated during the patient's treatment.

Patients and methods

We studied all adult neurological patients who over a 12 month period were investigated by cranial CT at Oulu University Central Hospital which supplies neurological services

for an adult population of 280 000. There were 1191 cranial CT investigations (572 men and 619 women), and the patients' ages ranged from 15 to 87 years, mean 46 years. One fifth were investigated as emergencies the remaining were elective.

The decision to refer a patient for CT was always based on careful neurological examination. The clinician recorded the case history, symptoms and signs and suggested the anatomical site and nature of the CNS lesion. The main aetiological categories were: tumour, ischaemia, haemorrhage, degenerative CNS disease, malformation, hydrocephalus and trauma. The anatomical sites for suggested lesion were: cerebral hemisphere, posterior fossa or brain stem, other specified, generalised and undefined.

Two CT units were available (Somatom 2 and Toshiba TCT 80A). Axial scans were obtained, complemented by coronal sections when necessary. The slice thickness was 4-5 mm in the posterior fossa and 8-10 mm at the upper levels of the brain. The findings were analysed by a neuroradiologist; radiological diagnosis, localisation of the lesion and the use of contrast enhancement were recorded. The decision to use contrast medium was made by the neuroradiologist after reviewing the unenhanced images and the clinical history. Enhancement (a 50 ml intravenous bolus of 60-70% ionic iodine) was used in 537 cases (45%).

The cases of verified IC tumours were manually analysed in detail and BMDP Statistical Software (University of California, 1985) was used in the treatment of the data.

Results

CT revealed 51 mass lesions in the 1191 patients. These proved to be a neoplasm in 32 cases, a haematoma in four, and an abscess in one (table). The remaining 14 cases were follow up examinations of previously diagnosed IC tumours.

The 32 IC tumours consisted of astrocytomas (14), metastases (7), meningiomas (5), glioblastomas (3), tumours of unspecific pathology (1), hemangioblastoma (1) and meningeal melanoma (1). Histological confirmation was obtained in 26 cases (81%). Biopsy or operation was withheld in six cases, in which the presumed diagnosis was based on CT and angiography (case 18), or the presence of a

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Table Clinical and CT findings in 32 patients with intracranial tumours

Patients			Tumour type	Tumour localisation		Tumour size* (cm)	Main symptom/sign	Duration of symptoms
Case No	Age	Sex		Clin	CT			
1	35	m	Astrocytoma	RH	RT	2.5	Seizures	2 years
2	59	f	Astrocytoma	RH	RF	3.0	Focal seizures	1 month
3	71	m	Metastasis	UN	RT	9.0	Confusion	2 months
4	62	m	Metastasis	RH	RT	4.0	Headache/hemiparesis	1 month
5	65	m	Astrocytoma	RH	RF	5.0	Seizures/hemiparesis	6 months
6	64	f	Meningeoma	UN	RP	3.0	Psychosis/headache	1 year
7	56	m	Metastasis	RH	RP	4.5	Seizures/hemiparesis	6 months
8	33	f	Astrocytoma	RH	RF	2.3	Seizures	2 months
9	68	f	Astrocytoma	RH	RP	6.0	Confusion/hemiparesis	2 months
10	49	m	Tumour UN	RH	RT	4.5	Seizures/headache	1 year
11	44	f	Astrocytoma	RH	RF	5.5	Progress frontal	1 month
12	71	m	Astrocytoma	LH	LT	2.0	Focal seizures	1 year
13	54	m	Meningeoma	LH	LP	3.5	Focal seizures	4 months
14	62	f	Glioblastoma	UN	LT	5.9	ICP increased	5 months
15	68	f	Astrocytoma	LH	LT	4.0	Seizures	2 months
16	57	m	Astrocytoma	LH	LF	4.0	Seizures	1 year
17	59	m	Glioblastoma	LH	LT	4.5	Headache/dysphasia	1 week
18	31	m	Hemio-astroblastoma	PoFo	PoFo	4.5	Progress cerebellum	1 year
19	36	m	Metastasis	PoFo	PoFo	4.0	Progress cerebellum	1 month
20	57	m	Metastasis	PoFo	PoFo	4.0	Progress cerebellum	1 month
21	47	f	Melanoma	PoFo	PoFo	2.8	Progress cerebellum	6 months
22	43	m	Meningeoma	PoFo	PoFo	4.0	Unconsciousness	4 days
23	34	m	Astrocytoma	PoFo	PoFo	2.0	Progress cerebellum	10 months
24	33	f	Astrocytoma	UN	PoFo	3.0	ICP increased	1 week
25	66	m	Metastasis	LH	Se	3.5	ICP increased	1 month
26	58	f	Astrocytoma	UN	Se	3.4	Confusion/headache	1 month
27	62	f	Metastasis	PoFo	Se	3.0	Headache	1 year
28	39	f	Meningeoma	UN	Se	2.0	Seizures	1 year
29	38	m	Astrocytoma	LH	Sa	2.5	Hemiparesis	5 years
30	63	f	Meningeoma	LH	Sa	3.2	Focal seizures	3 years
31	55	m	Glioblastoma	UN	biF	7.0	ICP increased	1 month
32	49	f	Astrocytoma	UN	biF	3.5	Headache	9 months

m = male; f = female; RH = right hemisphere; LH = left hemisphere; UN = undefined; R/LT = Right/Left temporal; R/LF = Right/Left frontal; R/LP = Right/Left parietal; biF = bifrontal; PoFo = posterior fossa; Se = sellar; Sa = sagittal; ICP = intracranial pressure; * = largest diameter.

known malignant primary tumour (cases 20 and 27); two cases (12 and 3) were inoperable, and one headache patient with CT findings compatible with a small meningioma (case 6) was followed up without operation.

In the 32 new cases of IC tumour the clinical diagnosis was correct in 25 (78%). The seven cases in which the clinical diagnosis was wrong included four mimicking stroke (9, 14, 15, 22). In a further case (22) chronic alcoholism and sudden progression of CNS symptoms suggested trauma rather than neoplasm. In another case (27) the clinical diagnosis of neuro-sarcoidosis had been based on known pulmonary sarcoidosis, a raised ESR and multiple neurological symptoms. In two further cases (24 and 29) the primary diagnosis had been a generalised CNS disorder (atrophy, hydrocephalus) possibly associated with a tumour. The most common symptoms in the 32 patients with IC tumour were progressive headache combined with focal neurological signs (14 cases) such as hemiparesis, diplopia, dysphasia or signs of cerebellar dysfunction. Two hundred and twenty six patients had epileptic seizures; a tumour was found in 11 (4.9%) especially when seizures were focal (five cases). In six cases seizures were the only symptom. The average duration of the main symptoms was eight months in the patients with tumour (table). Of the 1191 patients, 226 were examined due to seizure disorder and 207 due to headache. Two of the latter were found to have a tumour (1%), other findings were: infarct (3), malformations (2), atrophy (20), and miscellaneous minor abnormalities (20).

Clinical localisation of the tumour had been correct in 20 cases (out of 32, 63%). Fourteen of the 17 cerebral hemisphere tumours and six

of seven tumours in the posterior fossa had been localised correctly, the others being undefined. Localisation had been incorrect in the four para- or suprasellar tumours and two parasagittal and two bifrontal neoplasms.

Contrast enhancement was used in 28 of the 32 tumour cases (88%). In the remaining four cases the findings on the unenhanced CT were considered adequate. In most cases enhanced CT was more informative for the size or type of tumour; contrast enhancement also showed the boundaries of the tumours more clearly.

In the 1191 referrals for CT an IC tumour was suggested clinically in 433. In the majority of these (273, 63%) the clinician wished to exclude a tumour as the cause of epileptic seizures (28%), headache (23%), balance disturbance (11%), or other symptoms (38%). The neurologist had found some evidence to support a space occupying lesion in 160 patients, in 20 of whom (12.5%) a brain tumour was confirmed by CT.

Discussion

The correlation between clinical and CT findings in IC tumours has been discussed in a number of previous studies,^{3,8-12} but generally in selected groups and little information is available concerning routine neurological practice. Our study concerns all neurological referrals for cranial CT, both emergency and elective. The proportion of new patients in whom an IC tumour was shown by CT was 2.7% (32 of 1191) of all referrals for cranial CT. A cerebral tumour was found by CT in 13% of cases with CNS symptoms and signs potentially attributable to a tumour but was also found in 4% of patients in whom clinical

findings were normal but the history suggested a tumour. This emphasises the importance of a careful history and examination.

Overall, the clinician's localisation of the tumour was correct in 63% of cases. Sellar, parasagittal and frontal tumours posed problems and failure of localisation was most common in patients with symptoms and signs of increased IC pressure as the only manifestations of the disease.

The correlation between the size of the tumour and clinical localisation proved weak. A large tumour was often found to cause nonfocal disturbances, as might be expected in slowly growing tumours. This indeed became evident when the correlation between histology and clinical localisation was analysed. Meningiomas were localised less successively than invasive parenchymal tumours but were associated with a considerably longer symptomatic period. Seven tumours were not diagnosed clinically. The criteria for cranial CT should not be too strict. The previously reported prevalence of IC tumours as the cause of epilepsy in adult patients has varied from 4% to 38%.^{11 13 14} One CT study¹¹ of 148 patients after their first seizure revealed nine (6%) with a tumour, which agrees with our results.

Investigation of patients with headache but without neurological signs was not fruitful. Two patients (1%) were found to have an IC tumour: a relapsing frontal astrocytoma, and a parasellar metastasis associated with a one year history of headache, in none of the others did the CT findings change treatment. Our findings support previous conclusions^{9 10 12} concerning the absence of correlation between headache as an isolated symptom and abnormal CT findings. In one study of 505 patients with headache as their sole symptom and normal findings in a complete neurological examination, cerebral CT revealed a tumour in 13 (3%) and some other abnormality in a further 25 (5%).¹⁰ Thus the cost effectiveness of CT in investigating patients with headache and normal neurological findings is low. On the other hand, exclusion of a mass lesion is often necessary, perhaps by means of an abbreviated CT examination, as has been suggested previously.¹⁰

Where there is a continuing disproportion between the supply of and demand for cranial CT, a careful clinical examination remains necessary for adequate patient selection. CT is, however, often required for exclusion of a space occupying lesion, to make therapeutic decisions possible. When the symptoms and signs remain unexplained after CT and ancillary investigations, clinical follow up is needed. CT has not completely solved the differential diagnostic problems, for example, of distinguishing between infarcts and low grade astrocytomas and therefore clinical follow up is needed when symptoms and signs remain unexplained after CT and ancillary investigations. The ever growing demand for imaging investigations of the CNS can only be met by careful clinical selection of patients.

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