Incidental focal intracranial computed tomographic findings

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SUMMARY Of 3000 consecutive computed tomography (CT) head scans there were 28 adult patients who had a focal intracranial lesion, who presented with nonspecific symptoms, and who had normal initial neurological examination. These lesions included cerebral infarction (8), focal atrophy (5), intracerebral haematoma (3), chronic subdural haematoma (3), focal calcification (3) and intracranial neoplasm (6). Neurological consultation was obtained after the CT in all cases. Angiography was subsequently performed in 16, and eight patients underwent surgery. In no case was there evidence that clinical outcome was improved because of the early CT diagnosis.

Computed tomography (CT) has decreased the need for other neurodiagnostic studies. Because of its rapid acceptance as an imaging technique, utilisation and cost benefit analyses have been limited. Following its introduction, the waiting time for CT was prolonged. This encouraged more judicious use, and head CT scans were usually ordered only by physicians with training in neurodiagnosis and neurological disease. With increasing availability, scans are sometimes ordered by physicians who have no formal training in neurodiagnosis. In certain cases, the patients have no prior neurological consultation, and the scans are performed with poor indication. The purpose of this report is to analyse the clinical findings and outcome in 28 patients who had an incidental focal lesion detected by CT which required much time and expense to study and evaluate with little ultimate benefit.

Methods and material

Three thousand consecutive adult patients who had CT head scans were retrospectively analysed. Twenty-eight who had a focal abnormality were reported as having had a normal initial neurological examination, but none had had a prior neurological or neurosurgical consultation. Patients with systemic carcinoma, for example of lung or breast, in whom CT was performed as a staging procedure were excluded. The clinic and hospital records were analysed for the following information, (1) symptomatology necessitating hospitalisation, (2) neurodiagnostic studies performed prior to CT, (3) studies performed as a consequence of the abnormal CT, (4) the results of the neurological consultation, (5) subsequent hospital stay (prolonging hospital course) and treatment which resulted from the CT finding.

Results

The symptoms for which the scan was performed is listed in table 1. The lesions visualised by CT are listed in table 2. Eight patients showed CT findings consistent with a cerebral infarction. These patient's symptoms included cerebral infarction (8), focal atrophy (5), subdural haematoma-chronic (3), intracerebral haematoma (3), focal calcification (3) and intracranial neoplasm (6).

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ages ranged from 44 to 56 years. Initial history was reported as negative for cerebrovascular symptomatology; neurological examination was reported as normal and no carotid bruits were heard. These patients were initially hospitalised for symptoms including light-headedness, chest pain, palpitation, shortness of breath; however, none was found to have evidence of cardiac arrhythmias, myocardial infarction, cardiomyopathies or valvular heart disease. Four patients had systemic arterial hypertension and four others were normotensive. Prior to CT all patients had electroencephalograms (EEG), five of which were normal and three showed focal slow wave pattern. Only two patients had isotope brain scans; both had normal dynamic and static studies.

Following demonstration of CT abnormality, neurological consultation was obtained in all cases. Retrospective history indicated symptoms consistent with prior cerebral hemispheric (carotid territory) transient ischaemic attacks in all (8) patients; four of them also described episodes of focal neurological deficit lasting 24 to 72 hours. No patient had a transient ischaemic attack in the 3 months preceding CT. The consultant found mild focal neurological deficit in five patients. In three patients, CT showed post-contrast grey matter enhancement and angiography was performed to exclude a neoplasm or vascular malformation. One of three patients developed a hemiparesis-hemisensory deficit immediately following angiography and this persisted for 11 days. The performance of angiography in these patients prolonged hospitalisation by 6 to 14 days. Five other patients did not have angiography; hospitalisation was prolonged 4 to 8 days to obtain isotope brain scan, CSF analysis, noninvasive carotid flow studies, and cardiology evaluation (including echocardiogram and 24 hours Holter cardiac monitor to exclude potential embolic source).

On the basis of the CT diagnosis of cerebral infarction, all patients were treated with aspirin and dipyridamole. Follow-up CT was performed 2 to 6 weeks later in five patients; this showed no interval change in two patients with hypodense nonenhancing lesions and resolution of enhancement in three others. These eight patients were followed for 3 to 13 months by the neurologist and remained clinically stable and had no further transient ischaemic episodes.

Three patients had CT evidence of intracerebral haematoma, but no mention was made in the initial history of a stroke syndrome. They were men (age 52, 57, 59 years); all had systemic arterial hypertension and were taking multiple antihypertensive medications. The purpose of the hospitalisation was to improve the blood pressure control as their physicians believed they were noncompliant. EEG and isotope scan were normal in all cases. The history subsequently obtained by the neurologist indicated a prior episode which was characterised by sudden onset of severe but evanescent headache followed by focal weakness or numbness lasting 3 to 4 days. This had occurred 2 to 4 weeks prior to hospitalisation. The neurologist reported that two patients had focal neurological deficit. The CSF was clear. A haematoma in the basal ganglia was believed to be of hypertensive aetiology; angiogram was not performed but an evaluation for a coagulation disorder was undertaken. Two other patients with lobar haematomas had angiography to exclude an underlying aetiology (angioma, aneurysm or neoplasm). In these three cases, hospitalisation was prolonged 6 to 12 days following CT; however medical treatment was not altered by this finding. In one case, follow-up CT showed resolution of the haematoma three months later.

Five patients had CT evidence of focal cerebral hemispheric atrophy. The purpose of CT was to define an aetiology for diffuse paraesthesiae and light-headedness in two patients; three others had no neurological symptoms and indication for CT was to “rule out intracranial pathology”. These patients were being evaluated for diabetes, abdominal pain, urological symptoms and neck pain. EEG had been performed prior to CT in one case; it showed slight focal slowing. Following CT, EEG was performed in four others, it showed focal slowing in two and was normal in two. Isotope scan was negative in all five cases. Neurological consultation showed unilateral pronator drift in one case, homonymous hemianopia in one and was normal in three. Two patients had a prior history of head trauma accompanied by loss of consciousness. No patient underwent angiography. Hospitalisation was prolonged for 3 days after the CT to obtain neurological consultation, EEG and isotope scan. None of the patients had seizures and none had developed seizures within follow-up extending for 12 to 24 months.

Three patients had CT scan evidence of a unilateral chronic subdural haematoma. In two cases, there was evidence of mass effect but this was absent in one other. These patients had been hospitalised because of systemic medical disease; but CT was performed for headache only. The headache was located in the occipital region, was aching and band-like in nature, did not awaken the patient and was not associated with vomiting. Prior to CT, isotope scan was negative in all, and EEG was normal in two and diffusely slow in one. Following CT demonstration of subdural haematoma, neurological consultant found evidence of pronator drift and Babinski sign in two and no abnormality in one. Angiography confirmed the presence of the haematoma in all
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cases. One patient who had no abnormal neurologi-
ical finding and CT evidence of subdural haematoma
without mass effect did not have surgery; he remains
asymptomatic 14 months later. The hospital course
was prolonged 6 days for angiography, neurological,
and neurosurgical consultation. He has had two
further CT scans which show no change in the
lesion. Two patients had surgery with drainage of 50
to 75 ml of liquified proteinaceous fluid with disap-
ppearance of the subdural mass on CT. One patient
was neurologically normal, but one patient
developed non-haemorrhage infarction with
residual hemiparesis and hemisensory deficit. Hos-
pitalisation was prolonged 16 to 20 days in these two
cases.

In three patients, CT showed evidence of
calcification; this was in the frontal-parietal, medial
temporal and basal ganglia regions. In these three
patients, the indication for the CT was to "rule out
intracranial pathology" as hospitalisation was neces-
sitated for an acute medical problem. EEG showed
a focal spike pattern in two and was normal in one;
isotope scan was normal in three. No calcium or
phosphate abnormality was discovered. In two
patients with an EEG spike focus, angiography was
performed to exclude an underlying mass or vascula-
lar malformation. For this, hospitalisation was pro-
longed 6 to 11 days. Follow-up in these three
patients has extended for 9, 12 and 26 months;
neither seizures nor interval CT change has oc-
curred.

Three patients had CT findings consistent with a
primary intracranial malignant neoplasm. One
patient was evaluated for syncope; two others had
dizziness and head pain with weight loss and
anorexia. Prior to CT, EEG showed mild degree
focal slowing in two and was normal in one; isotope
scan was negative in all. The neurologist's examina-
tion showed focal deficit in two patients; the other
patient had obvious episodes of partial complex
seizures. Angiography was performed in all and it
showed an avascular mass. All patients had surgery
and had histological evidence of an intracranial
glioma. Following surgery neurological deficit
worsened in two; major motor seizures developed in
all within 2 weeks of operation. All received brain
irradiation. Hospitalisation was prolonged for 5 to 8
weeks by subsequent diagnostic and therapeutic
intervention. These patients later required reopera-
tion for tumour recurrence within 5 months of initial
treatment. Two patients had CT findings consistent
with meningioma. The parietal convexity lesion was
2-8 cm in diameter and there was slight evidence of
mass effect without hydrocephalus; the tentorial
meningioma was 1-2 cm in diameter without associ-
ated hydrocephalus. Symptoms included headache
and dizziness. Prior to CT, EEG was normal in both
cases; isotope scan was positive in the parietal and
negative in the tentorial tumour. Neurological
examination performed by both neurology and
neurosurgical consultants were negative. Angio-
graphic findings were consistent of meningioma; this
diagnosis was confirmed surgically. Hospitalisation
was prolonged by 16 to 26 days by surgery. Nine
months post-surgery the patient with the parietal
meningioma developed persistent focal seizures with
no CT evidence of tumour recurrence; and the
patient with the tentorial meningioma had a severe
gait ataxia.

One patient had CT evidence of an intrasellar
non-calcified enhancing lesion with suprasellar exten-
sion; however he had no endocrine or visual symp-
toms. This patient was hospitalised because of
headache, lightheadedness and inability to concen-
trate which had developed after mild head trauma.
Following CT, tangle screen visual field examina-
tion was normal. Angiography confirmed the CT
findings and showed no evidence of aneurysm. Bi-
frontal craniotomy was performed with removal of a
chromophobe adenoma. Hospitalisation was for 6
weeks. Following completion of radiotherapy, visual
fields showed superior bitemporal quadrantanopsia to
5 mm red test object.

Discussion

In the initial period following the introduction of
CT, there was a waiting period of up to 2 months for
CT. Because of this limited access, physicians with
training in neurological disorders acted as a triage
to determine the need for CT. With the dramatic
increase in the number of scanners installed and a
decrease in the waiting time for CT, this triage has
been abandoned; physicians without specialised
training in neurological disorders or neurodiagnostic
techniques routinely order CT, and in patient's with
questionable indications. In this study, 28 patients
who had a focal CT lesion did not have a prior
neurological consultation and in no case is there evi-
dence that outcome was improved by early CT diag-
nosis. Many more CT scans are ordered than should
be. From this study it was not possible to assess the
incidence because many scans were ordered from
several different patient sources. In certain institu-
tions very careful screening for CT was performed
but in others no screening was possible.

This trend toward CT without initial neurological
consultation was not related to lack of availability of
neurologists or neurosurgeons, but it appears that
CT was being used as a screening neurological pro-
cedure. This approach generates several types of
problems; (1) a negative CT study does not exclude
the presence of neurological disease, for example meningitis and subarachnoid haemorrhage are diagnosed by lumbar puncture and CSF analysis; (2) overreliance on CT diagnostic accuracy especially if the clinical findings suggest an alternative diagnosis; for example, when the CT findings show cerebral atrophy but clinical findings suggest a functional affective disorder; (3) aggressive management of incidental lesions.

Necropsy studies have reported that 30% of normal patients have incidental pituitary adenomas. These lesions are usually small; however certain larger lesions which would have been expected to cause endocrine or visual deficit have also been reported. Incidental meningiomas have also been reported. With the increased diagnostic sensitivity of CT, certain of these incidental tumours are now detected; and this presents a therapeutic dilemma. The anaesthetic and surgical risks combined with the unknown benefit associated with early removal in an asymptomatic patient makes this an uncertain decision. Prior studies have reported the increased incidence of low-grade (Grade I) gliomas detected in the post-CT era; however, early diagnosis has not had a positive effect on outcome. In three patients who had CT and operative findings of glioma (Grade I, 2 cases; Grade II, 1 case), the outcome was poor with early recurrence. In the three other patients with intracranial neoplasms, outcome was not improved by early diagnosis.

In patients with symptoms of cerebrovascular disease, CT is completely accurate in excluding clinically symptomatic intracerebral haematoma and is quite reliable in detecting cerebral infarction. In two prior studies, the CT diagnosis of an intracranial haematoma was clinically unsuspected, similar to the patients in this series. Abnormal CT findings in cerebral infarction consisting of grey matter enhancement has been reported in rare instances of patients with transient ischaemic attacks. Careful neurological history in the eight patients with CT evidence of cerebral infarction or ischaemia documented prior vascular episodes which were previously undetected by the non-neurologist. The abnormal CT findings may be important in assessing the need for anticoagulation, angiography or carotid endarterectomy.

The natural history of subdural haematoma is not definitely known. It is assumed that it causes neurological disturbances when mass effect develops; however two patients were asymptomatic even though mass effect was present and focal findings were present. In one patient, there has been no change in CT appearance or clinical findings 14 months later with no surgery or corticosteroid. In this case, since the hypodense lesion effaced the cortical sulcal spaces with normal sized and positioned ventricles, it is possible that this represents a subdural haematoma and not a subdural hygroma or focal atrophy.

It has been reported that almost one-quarter of patients who are admitted to medical services in community hospitals have major neurological problems. If these patients are not carefully assessed clinically with critical evaluation of the need for CT, two potentially dangerous trends may develop. Firstly, unnecessary diagnostic and therapeutic intervention may be carried out in asymptomatic patients with incidental unrelated lesions. Secondly, a negative CT scan result may then be accepted as evidence that neurological disease does not exist; and consultation from a physician with training in neurodiagnosis is not subsequently obtained.

References

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