Neuroradiology (1984) 26: 223–228

Neuroradiology

Radiological features of subependymoma with emphasis on computed tomography

J. M. Stevens[1], B. E. Kendall[1] and S. Love[2]

[1]Lysholm Radiological Department and
[2]Department of Neuropathology, National Hospital for Nervous Diseases, Queen Square, London, England

Summary. The features of 17 symptomatic subependymomas on X-ray computed tomography are described. Thirteen were reviewed from isolated case reports and 4 were original material. Over half were entirely intraventricular, 6 extended into brain substance and 2 into subarachnoidal cisterns. Twelve were isodense, 15 showed diffuse but irregular enhancement, and 5 contained nodular calcification. Large low density cysts, intratumoural haemorrhage and brain oedema were found almost exclusively in lesions extending into brain substance. It is concluded that subependymomas resemble ependymomas too closely on CT to be distinguished radiographically from them as a separate group. However subependymomas contain calcification slightly less frequently, and usually appear as mainly intraventicular lesions even when they occur above the tentorium.

Key words: Cerebral – cerebellar tumour – subependymoma – CT

Subependymomas are usually intraventricular tumours wich are occasionally found incidentally at autopsy [1, 2]. It has been estimated that only 66% of supratentorial and 36% of infratentorial lesions ever become symptomatic [5]. Their consistency is firm, rather than soft and friable like most ependymomas, and they tend to compress rather than infiltrate surrounding brain substance [1, 5, 6]. Total surgical excision may be possible and accurate preoperative diagnosis is therefore desirable. The appearances of subependymomas on computed tomography (CT) have been illustrated in a few isolated case reports, but a description of characteristic features based on a review of adequate material is lacking. A survey of the published cases with the addition of four new patients making a total of 17 lesions with CT data is presented.

Material and methods

The histology records of the National Hospitals for Nervous Diseases from 1974 to 1983 were reviewed. Four cases of subependymoma were found for which adequate CT scans were available. Thirteen further cases were gleaned from the literature.

Results

Case histories

Case 1: D. B., a 31-year-old Maltese woman had experienced fainting attacks for 2 years, difficulty in walking for 6 months and increasing headache and deterioration in mental state over 3 months. On arrival at hospital she was deeply comatose and had bilateral papilloedema.

A CT scan showed hydrocephalus with periventricular oedema associated with a rounded mass isodense with brain substance occluding the posterior part of the third ventricle (Fig. 1 a). The lesion increased slightly in density after intravenous contrast medium. Ventriculography demonstrated a slightly lobulated mass protruding from the posterior wall into the third ventricle (Fig. 1 b). Angiography revealed no pathological circulation and pinealoma was considered the most likely diagnosis.

At operation a firm, yellowish, avascular tumour was removed leaving a defect in the roof of the third ventricle. Histological examination revealed subependymoma with multiple foci of microcalcification (Fig. 2).

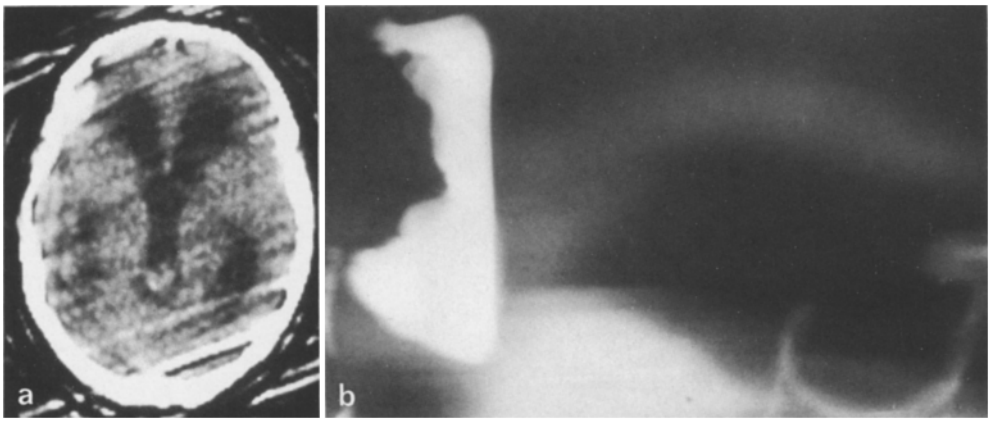

Fig. 1 a and b. Case 1. **a** Poor quality scans due to patient movement. There is hydrocephalus due to a lobulated mass in the posterior part of the third ventricle. Pre-contrast scan. **b** Midline tomogram of a myodil ventriculogram with the patient in brow-up position. Myodil outlines a large tumour mass with a lobulated surface protruding into the posterior part of the third ventricle

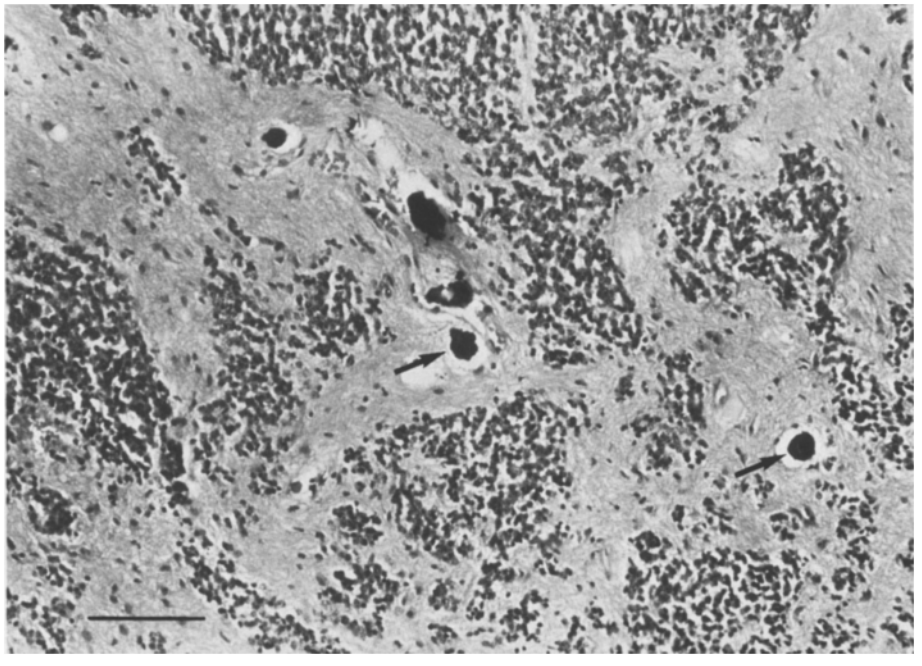

Fig. 2. Case 1. Pathology. Section of tumour showing histological features of a subependymoma: clusters of ependymal cells separated by broad bands of astrocytic processes. The tumour contains multiple small calcospherites *(arrows)*. Haematoxylin and eosin. Bar + 100 μm.

The patient was discharged without focal neurological deficit and has returned to Malta.

Case 2: W. B., aged 13 years presented with visual obscuration, worsening headache, nausea and vomiting, and progressive unsteadiness of gait of 3 months duration. Examination revealed ataxia of gait, nystagmus on extreme lateral gaze, and bilateral papilloedema.

A CT scan showed a lobulated mass in the fourth ventricle with extensions towards the lateral recesses and cerebral aqueduct, causing moderate hydrocephalus. The mass, of slightly greater density than brain, enhanced homogeneously but contained a small nonenhancing central area of low density. There was minimal surrounding low density in the cerebellar white matter, due to oedema (Fig. 3). The probable preoperative diagnosis was considered to be medulloblastoma.

At operation a firm, greyish tumour covered the foramen of Magendie and merged with the ependymal roof of the fourth ventricle. It extended into both lateral recesses and was somewhat adherent to the floor. Histology showed typical appearances of subependymoma. The patient made a slow post-operative recovery, but was discharged and has remained reasonably well over 18 months.

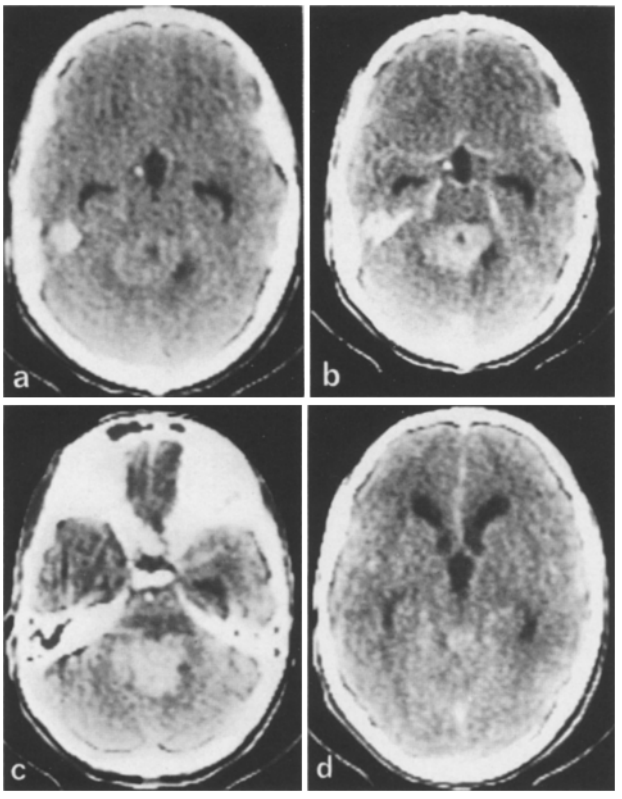

Fig. 3 a–d. Case 2. Plain (**a**) and enhanced (**b**) scans showing an enhancing mass in the fourth ventricle with associated hydrocephalus. Extensions from the mass towards the lateral recesses (**c**) and cerebral aqueduct (**d**) are visible

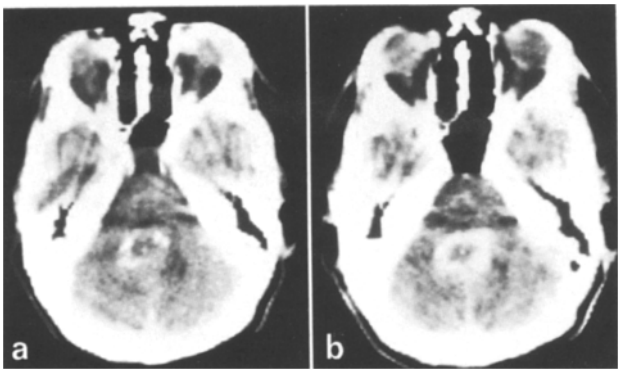

Fig. 4 a and b. Case 3. Plain (**a**) and enhanced (**b**) scans showing an enhancing mass in the fourth ventricle with a non-enhancing central zone. It contains nodular calcification

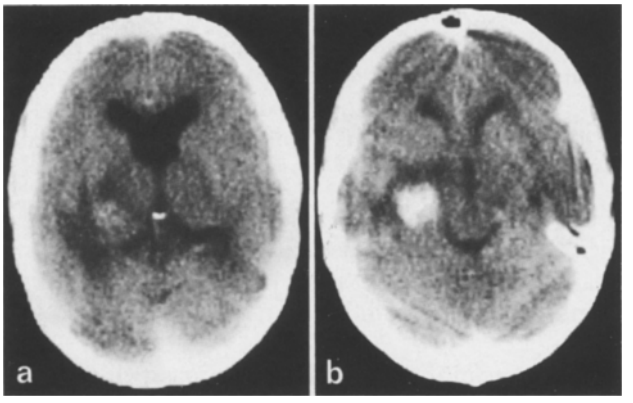

Fig. 5 a and b. Case 4. **a** Plain scan showing an approximately isodense mass in the left thalamus with oedema extending beyond the thalamus into the white matter. **b** Enhanced scan showing diffuse homogeneous enhancement of the mass which extends into the medial part of the temporal lobe beneath the thalamus

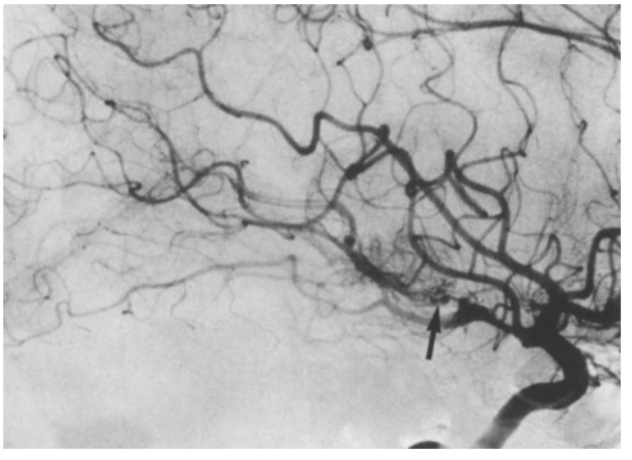

Fig. 6. Case 4. Left internal carotid arteriogram. There is a group of abnormal vessels *(arrow)* supplied mainly by the anterior choroidal artery, and the basal cerebral vein and straight sinus have filled early. Although a diagnosis of angioma was made at the time of angiography, an area of diffusely increased capillary circulation above the abnormal vessels suggested the probable neoplastic nature of the lesion

Case 3: A. M., aged 30 years, complained of increasing headache, nausea and vomiting over the past 2 years, and visual obscurations for 9 months. Examination revealed horizontal nystagmus on lateral gaze and bilateral papilloedema.

Plain CT showed a mass mainly isodense with brain but containing nodules of calcification lying within the lower half of the fourth ventricle. Intravenous contrast confirmed a lobulated tumour which enhanced irregularly and extended towards the lateral recesses (Fig. 4). There was moderate hydrocephalus and a cyst-like structure, thought to be a dilated fourth ventricle, was visible above the lesion. Angiography showed a slight increase in capillary circulation behind the upper medulla. The lesion was considered most likely to be an ependymoma.

At operation an irregular mass was found in the fourth ventricle and adjacent part of the left cerebellar hemisphere. There was a large cyst containing 5 ml of brown fluid in the upper part of the lesion.

Histological examination showed a mixed tumour, the bulk of the lesion being composed of subependymoma but with small area of ependymoma. Post-operatively the nystagmus persists but the patient is otherwise well without evidence of tumour recurrence after 4 years.

Case 4: D.W., aged 21 years suddenly developed right homonymous hemianopia which then remained static or 1 year prior to admission. Examination revealed, in addition, a mild right facial weakness.

A CT scan showed an isodense mass with moderate, uniform enhancement in the posterior part of the left thalamus and the medial wall of the trigone (Fig. 5). There was surrounding oedema and mild hydrocephalus. Angiography demonstrated a small collection of abnormal vessels in the medial part of the temporal lobe, supplied by an enlarged anterior choroidal artery, and early filling of the basal vein. The lesion was diagnosed as an angioma (Fig. 6). An air encephalogram confirmed that it was extra-ventricular.

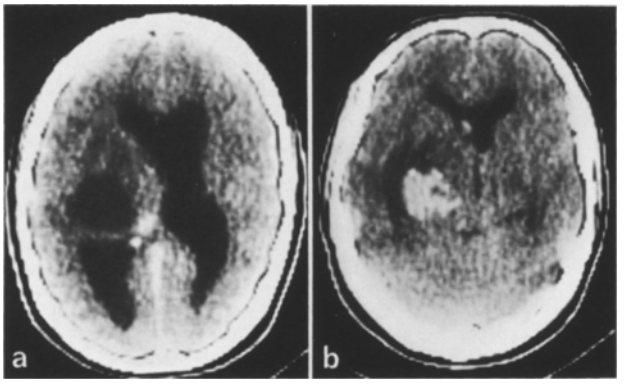

Fig. 7 a and b. Case 4. A large cyst has appeared in the left cerebral hemisphere above the tumour mass. **a** Enhanced scan in which the upper part of the tumour is visible posteromedial to the cyst. **b** Enhanced scan showing the subthalamic part of the tumour which has increased only slightly in size

Four months later a second CT scan indicated the development of a large low density cyst which extended superolaterally from the thalamus across the posterior limb of the internal capsule and lentiform nucleus and into the upper part of the temporal lobe (Fig. 7). The isodense component had increased slightly in size and projected into the floor of the left lateral ventricle. At surgery a large cyst containing yellow fluid was evacuated and a firm nodule projecting into it from the brain substance was excised and shown histologically to be a subependymoma.

Table 1. Previously reported cases of subependymoma included CT scans

Source	Site	Age (years)	Hydrocephalus	Histology
Swartz et al. [6]	4th ventricle	5	yes	pure
	cerebellopontine angle and lateral recess of 4th ventricle.	26	?	mixed
	cerebellopontine angle and lateral recess of 4th ventricle	?	?	pure
Lobato et al. [7]	frontal horn, frontal lobe eroding into orbit, very large	14	no	pure
Clarenbach et al. [8]	4th ventricle and cerebellar hemisphere	22	yes	pure
	4th ventricle and cerebellar hemisphere	22	yes	pure
Changaris et al. [9]	trigone and temporal lobe	16	no	pure
Gandolfi et al. [10]	4th ventricle	63	yes	pure
Vaquero et al. [11]	frontal horn	64	no	pure
	septum pellucidum	50	yes	pure
Kazner et al. [12]	trigone	6	no	pure
	body of lateral ventricle	49	yes	pure
	body of lateral ventricle	?	yes	pure

Table 2. Radiological features of 18 subependymomas in different situations

Site	Number	Average age (years)	Isodense	Enhancement after I/V contrast	Calcification	Low density cyst formation	Haemorrhage	Brain oedema
Intraventricular	9	30.3 range 5 to 64	7	7	2	2	0	1
Ventriculocisternal	2	26	2	2	2	0	0	0
Ventricular-parenchymal	6	19	3	6	1	2	2	2
Total	17							

Radiotherapy was given post-operatively, and during a 4 year follow-up period there has been no significant deterioration.

CT findings

Cases obtained from the review of the literature included in this analysis are summarised in Table 1. The CT features of the total 17 cases are shown in Table 2 in which the lesions are arranged into three groups based on their location with respect to ventricular cavities, subarachnoid cisterns and brain substance.

Discussion

Subependymomas are composed of clusters of isomorphic cells separated by densely fibrillary bundles of astrocyte processes. The name is derived from the similarity of their structure to that of the periventricular matrix layer or subependymal plate [1]. Although the tumour cells show some morphological features of astrocytes [1, 2], the presence of basal ciliary bodies, and appearances in tissue culture and on electron microscopy indicate that they are ependymal in origin [3, 4, 13]. The apparently inert nature of many subependymomas prompted Russell and Rubinstein to speculate that some of the lesions may be hamartomatous or a reaction to chronic ependymitis [2].

The appearances of the subependymomas on CT were more like ependymomas than astrocytomas. Just over half occurred in the fourth ventricle and most extended into lateral recesses, cerebral aqueduct or subarachnoid cisterns. Approximately three quarters were isodense and nearly all increased in density after the administration of intravenous contrast medium. Low density within some lesions corresponded histologically to microcyst formation or occasionally to larger cystic cavities within the tumour, and slightly increased density was associated with microcalcification or haemorrhage. In a review of 26 cases of ependymoma Swartz and Zimmerman [6] found most to be approximately isodense with grey matter, usually enhancing diffusely after intravenous contrast medium; in 292 astrocytomas Trumme and Steindoff [14] showed that 97% of well differentiated tumours (grade I) were of lower attentuation than normal brain and hardly ever enhanced after intravenous contrast medium, but as malignancy increased higher density areas became more frequent and the incidence of contrast enhancement rose to 89% in grade II and 100% in grades III and IV.

Many of the CT features of glial tumours are the result of secondary processes such as dystrophic calcification, cyst formation, haemorrhage, necrosis or brain oedema. Nodular calcification was seen on CT in 30% of subependymomas, slightly lower than the incidence reported in ependymomas [6] but higher than in most types of glioma [14]. Large cysts occurred in 23% haemorrhage in 12% and brain oedema in 17%, these features being more frequent in supratentorial lesions, which again conforms with observations in ependymomas [6, 15]. Furthermore oedema, large cysts and haemorrhage occurred almost exclusively with subependymomas extending from the ependymal surface into brain substance (Table 2).

The CT appearances of subependymomas are not sufficiently specific to distinguish them from ependymomas or mixed lesions. However supratentorial subependymomas more frequently than ependymomas are entirely or largely intraventricular, and calcification visible on CT is slightly less frequent. The angiographic findings available in three of our cases were too varied to be of value in preoperative diagnosis. It should be added however that subependymoma is one of the rarer types of intraventricular tumour; the radiological features of lesions in this situation have been recently reviewed [16].

References

1. Scheinker LM (1945) Subependymoma: a newly recognized tumour of subependymal derivation. J Neurosurg 2: 232–240
2. Russell DS, Rubinstein LJ (1977) Pathology of tumours of the nervous system, 4th ed. Arnold, London, pp 212–213
3. Azzarelli B, Rekale HL, Roessman U (1977) Subependymoma: a case report with ultrastructural study. Acta Neuropathol 40: 279–282
4. Friede RL, Pollak A (1978) The cytogenetic basis for classifying ependymomas. J Neuropathol Exp Neurol 37: 103–118
5. Scheitauer BW (1978) Symptomatic subependymoma. Report of 21 cases with review of the literature. J Neurosurg 49: 689–696
6. Swartz JD, Zimmerman RA, Bilanik LT (1982) Computed tomography of intracranial ependymomas. Radiology 143: 97–101
7. Lobato RD, Cabello A, Carmena JJ, De la Fuente M, Munoz MJ (1981) Subependymoma of the lateral ventricle. Surg Neurol 15: 144–147
8. Clarenbach P, Klethues P, Matzel E, Dichgans J (1979) Simultaneous clinical manifestation of subependymoma of the fourth ventricle in identical twins. Case report. J Neurosurg 50: 655–659
9. Changaris DG, Powers JM, Perot JR, Perot PL, Hungerford GD, Neal GB (1981) Subependymoma presenting as subarachnoid hemorrhage. Case report. J Neurosurg 55: 643–645

10. Gandolfi A, Brizzi RE, Tedeschi F, Paini P, Bassi P (1981) Symptomatic subependymoma of the fourth ventricle. Case report. J Neurosurg 55: 841–844
11. Vaquero J, Cabezudo JM, Mombella L (1983) CT scan in subependymomas. Report of two cases. Br J Radiol 56: 425–427
12. Kazner E, Wende S, Grumme TH, Lankscha W, Stochdorph O (1982) Computer tomography in intracranial tumours. Springer, Heidelberg Berlin New York, pp 119, 121, 126
13. Fu YS, Chen ATL, Kay S (1974) Is subendendymoma (subependymal glomerale astrocytoma) an astrocytoma? A tissue culture study. Cancer 34: 1992–2008
14. Grumme TH, Steinhoff H, Wende S (1976) Diagnosis of subtentorial tumours with CT. In: Lankscha W, Kazner E (eds) Cranial computed tomography. Springer, Heidelberg Berlin New York, pp 81–85
15. Coulon RA, Till K (1977) Intracranial ependymoma in children. A review of 45 cases. Childs Brain 3: 154–168
16. Kendall B, Reider-Grosswasser I, Valentine A (1983) Diagnosis of masses presenting within the ventricles on computed tomography. Neuroradiology 25: 11–22

Received: 14 July 1983

Dr. B. E. Kendall
Lysholm Radiology Department
National Hospital for Nervous Diseases
Queen Square
London WCI 3BG
England