Giant-Cell Tumors of the Temporal Bone: Management Strategies

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ABSTRACT

Objective: To discuss the current management options for giant-cell tumors (GCTs) involving the temporal bone and present two case reports and a review of the literature. **Method:** In a tertiary-care academic medical center, two patients with GCTs of the temporal bone were evaluated and managed. The patients underwent gross total resection and curettage of GCTs involving the temporal bone. Afterward, both patients were evaluated for postoperative complications as well as for recurrence. **Results:** Two patients underwent operative excision using curettage. Clinical and radiographic studies demonstrated no evidence of recurrence with 3 years of follow-up in one patient and 10 years of follow-up in the second patient. **Conclusion:** Based on these results, we concluded that gross total removal and curettage of GCTs in the temporal bone is a viable treatment option. This finding is contrary to previous studies.

KEYWORDS: Temporal bone, giant-cell tumors, skull base

Giant-cell tumors (GCTs) are rare lesions of the temporal bone that are most commonly found at the epiphysis of long bones.¹ Two percent of these lesions present in the head and neck, with the most common sites being the sphenoid, ethmoid, and temporal bones.² The senior author has treated two patients with GCTs involving the temporal bone with long-term follow-up. This article describes their presentation, diagnostic workup, treatment, and follow-up, as well as provides a review of the literature.

CASE 1

A 42-year-old man presented to an outside physician with a long history of reduced hearing in the right ear. The patient reported progressive deterioration over the last 3 years, and, in the year before presentation, he noted complete loss of useful hearing in his right ear. The patient denied headache, otalgia, facial pain, trismus, facial weakness, and paresthesias. He denied vision changes, difficulty with swallowing and speech, and changes in voice.

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Skull Base 2009;19:291–302. Copyright © 2009 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New York, NY 10001, USA. Tel: +1(212) 584-4662.

Received: September 18, 2008. Accepted after revision: October 27, 2008. Published online: January 9, 2009.

DOI 10.1055/s-0028-1115324. ISSN 1531-5010.

On physical examination, he was healthy in appearance with no apparent abnormalities of the skull or scalp. The left external ear and tympanic membrane were unremarkable. The right external ear was unremarkable. The right osseous external auditory canal, however, was narrowed from the superior aspect. The right tympanic membrane was normal in appearance with normal ossicular landmarks and normal mobility. Oral cavity examination and temporomandibular joint examination were normal. The indirect examination of the nasopharynx and larynx was normal. The neck examination revealed no masses or adenopathy. Cranial nerve and neurological examinations were normal with the exception of right-sided hearing

loss. Audiometric evaluation revealed a right profound sensorineural hearing loss.

Gadolinium-enhanced magnetic resonance imaging (MRI; Fig. 1) revealed a lesion arising from the right anterior middle cranial fossa floor, measuring 4.0 cm (anteriorly to posteriorly) by 2.8 cm (medially to laterally) by 3.0 cm (cranially to caudally). The lesion was hypointense to brain on T1, T2, and fluid-attenuated inversion recovery (FLAIR) images and moderately enhanced in an irregular fashion. The lesion displaced the temporal lobe of the brain in the cranial direction, without apparent direct dural extension or involvement. There was apparent complete replacement of the cochlea and semicircular canals with preservation of

Figure 1 Preoperative T1 magnetic resonance imaging from case 1. (A) Axial image after administration of gadolinium demonstrates a large hypointense lesion (arrow) with peripheral rim enhancement and several small areas of enhancement within the lesion on the right. (B) Coronal image after administration of contrast demonstrates the same hypointense lesion (arrow) with small areas of enhancement.

Figure 2 Computed tomographic images with bone window algorithm from case 1. (A) Axial image demonstrates a large lytic lesion on the right with erosion into the otic capsule at the level of the vestibule (arrow). (B) Coronal image demonstrates the same right-sided lesion (arrow) with significant bone remodeling.

the internal auditory canal (IAC). The intrapetrous internal carotid artery was not displaced or attenuated. The lesion appeared to extend to, but not involve, the temporomandibular joint, and the mandibular condyle appeared intact.

A dedicated thin section computed tomography (CT) scan of the temporal bone (Fig. 2) revealed a destructive lesion centered within the floor of the right middle fossa. There was significant bone destruction involving the otic capsule, superior aspect of the external auditory canal, and the squamous and petrous temporal bone. The lesion extended inferiorly into the glenoid fossa but did not appear to involve the mandibular condyle. The epitympanum, mesotympanum, hypotympanum, and mastoid were filled with abnormal soft tissue and/or fluid.

Via an endaural incision, a biopsy of the supra-auricular soft tissue mass was obtained for pathological examination and treatment planning. Superior to the zygomatic root, a skull defect filled with a brown-tan avascular lesion containing areas of ossification was biopsied. Pathological examination revealed GCT of bone (Fig. 3). Further treatment was delayed approximately 8 months due to patient preference. A middle cranial fossa approach was used to remove the entire tumor, including the middle ear and mastoid component

that was dissected from above. Extensive tumor involvement of the lateral skull, petrous apex, cochlea, semicircular canals, and IAC were identified at the time of surgery. The middle ear and external auditory canal skin and bone were involved with tumor. The lesion extended superiorly and extensively involved the dura of the temporal lobe. The temporal lobe over the area of dural invasion was infiltrated with tumor. The lesion extended anteriorly to the foramen ovale and the foramen rotundum and involved the dura overlying the second and third branches of the trigeminal nerve. There was extensive involvement of the temporomandibular joint, with tumor found within the joint capsule. The facial nerve was identified at the tympanic segment following exposure of the epitympanic space, and antrum from above. Tumor was gently removed to expose the ossicles. Once identified, the ossicles were removed. The facial nerve was then followed medially through the brown-tan, gelatinous, ossified tumor to the IAC. No identifiable bony landmarks were identified corresponding to the otic capsule. All visible areas of tumor were resected. To the extent possible, surrounding bone was curetted. A postauricular incision was then used to remove the tympanic membrane and external auditory canal skin. A modified Rambo technique was used to

Figure 3 Hematoxylin-eosin histopathology slide of specimen from case 1. Numerous multinucleated giant cells are seen in a background of spindle-shaped stromal cells and round monocyte-like cells.

close the external auditory canal.³ The eustachian tube was occluded, and the mastoid and middle ear were obliterated with abdominal fat. Bovine pericardium was used to repair the temporal dura in a water-tight fashion.

Prophylactic lumbar drainage was used for 3 days. The patient had an uncomplicated post-operative course and was discharged 5 days after surgery. Facial nerve function was normal at House-Brackmann grade I, and no cerebrospinal fluid (CSF) leak was encountered. The patient resumed all normal activities approximately 4 weeks following the procedure. He has had no complaints or temporomandibular joint dysfunction but has noted excessive lacrimation from the ipsilateral eye.

Follow-up at 3 years revealed no new clinical symptoms, and MRI revealed apparent complete resection of the tumor with stable reactive changes in the petrous apex and temporal lobe meninges (Fig. 4). Further follow-up and scanning will continue in the future.

CASE 2

A 47-year-old man with initial symptoms of prolonged left otalgia and aural fullness presented in 1995 after undergoing a subtotal resection via a middle cranial fossa approach at an outside facility in 1994. Pathology from this initial resection was consistent with a GCT of bone. The patient was subsequently found to have recurrence on radiographic imaging. Physical examination revealed mild left parotid fullness and decreased left auricular and external auditory canal sensation. Cranial nerve examination was unremarkable. Since the initial procedure, the patient reported decreased hearing. Audiometry revealed a mixed loss with speech reception threshold (SRT) 35dB and 96% discrimination on the left. Review of systems, past medical history, and family history were unremarkable.

A temporal bone CT scan revealed erosion of the tegmen tympani and encasement of the ossicular heads. There was extensive bony erosion superior

Figure 4 Postoperative magnetic resonance imaging (MRI) scans without fat saturation from case 1. (A) Axial T1 MRI without contrast demonstrates an area of hyperintensity (arrow) at the lateral aspect of the craniotomy. The hyperintense area represents the abdominal fat graft that was placed to reconstruct the defect as a result of tumor removal. (B) Postcontrast axial T1 MRI shows no change in the hyperintense area (arrow) and no new areas of enhancement. (C) Coronal T1 MRI with contrast shows an area of hyperintensity (arrow) that did not change from precontrast images.

and anterior to the cochlea with possible membranous labyrinth exposure. The mesotympanum and hypotympanum were aerated. Magnetic resonance imaging revealed an irregular enhancing lesion that eroded the squamous, petrous, and mastoid portions of the left temporal bone. The mass extended anteriorly and inferiorly to the internal carotid artery and cochlea and medially to the glenoid fossa and mandibular condyle.

The patient underwent a revision left temporal craniotomy and infratemporal fossa approach for resection of infratemporal/middle fossa mass. Extensive erosion of the otic capsule and petrous temporal bone was identified at surgery. The tegmen tympani was eroded with exposure of the ossicles. The tumor extended inferiorly into the glenoid fossa and also invaded the cochlea. The floor of the middle fossa anterior to the glenoid fossa was dehiscent, through which, the residual tumor extended inferiorly. All visible tumor was removed, and the surrounding bone was curetted. The extensive middle fossa floor defect was reconstructed with a split-calvarial bone graft. Pathological examination was consistent with GCT of bone (Fig. 5).

Postoperatively, the patient made an uneventful recovery with normal facial nerve function. The procedure left the patient with

profound hearing loss and mild persistent instability, presumed to be due to the bony otic capsule erosion of the cochlea. The patient subsequently developed more severe instability and, in 1999, underwent a left transmastoid labyrinthectomy. The labyrinthectomy improved, but did not completely resolve, the patient's complaints of instability. At last follow-up (10 years postoperatively), MRI showed no evidence of recurrence (Fig. 6).

RESULTS

The two patients in this series presented with progressive hearing loss and otalgia with aural fullness. Each tumor was centered along the floor of the middle fossa and demonstrated extensive osseous erosion that directly involved the otic capsule and glenoid fossa in both cases. Tumor resection with curettage and drilling via a combined middle fossa/infratemporal fossa approach was used in both patients. The extensive erosion resulted in profound hearing loss in both cases. One patient developed persistent vestibular complaints requiring labyrinthectomy several years after the tumor resection. There

Figure 5 Hematoxylin-eosin histopathology slide of specimen from case 2. Several very large multinucleated giant cells are seen along with the spindle-shaped stromal cells and round monocyte-like cells.

was no disturbance of mastication or facial nerve function in either patient postoperatively. There has been no evidence of recurrence in patient one and patient 2 on follow-up clinical and radiographic examination 3 and 10 years, respectively, after curettage excision.

DISCUSSION

History

Sir Astley Cooper was the first to describe a GCT in 1818. Giant-cell tumors were previously known as

Figure 6 Postoperative magnetic resonance imaging (MRI) scans without fat saturation from case 2. (A) Axial T1 MRI without contrast demonstrates a very small isodense area (arrow) in the posterior petrous apex. (B) Postcontrast axial T1 MRI demonstrates an isodense area along the posterior petrous apex (arrow), which has remained unchanged for 9 years. (C) Coronal T1 MRI with contrast shows an area of hyperintensity (arrow) in the petrous apex that did not differ in signal from precontrast images and most likely represents bone marrow or fat.

myeloid sarcoma, tumor of myeloplexus, osteoblastoclastoma, or osteoclastoma.⁴ Since the initial description of this pathological entity, there have been few reports of head and neck involvement. The first GCT affecting the temporal bone was initially described by Doderlein in 1913.⁵

Epidemiology

Giant-cell tumors represent 4 to 9.5% of all primary bone tumors and 18 to 23% of benign bone tumors.⁶ These lesions are more common in young women, especially in those whose lesions involve the spine and craniofacial skeleton.⁶ The peak prevalence of this entity is in the third decade of life. Approximately 0.7% of patients will develop multifocal lesions with a high potential for those with two GCTs to develop more, usually within 2 years. Multiple lesions do not increase the rate of distant metastasis, but one must exclude multiple brown tumors of hyperparathyroidism.⁴ Approximately 2% of GCTs present in the head and neck, primarily in the sphenoid, ethmoid, or temporal bone as these sites develop from enchondral ossification.⁴ In a review 2404 cases of GCTs, only 1% occurred in the craniofacial region, with the most common sites being the temporal and sphenoid bones.⁷

Individuals with Paget's disease have an increased risk of developing a GCT that often involves the craniofacial bones. Patients who develop GCTs in the setting of Paget's disease are typically older and male with polyostotic involvement and have had the disease for some time before these lesions are found. Giant-cell tumors in patients with Paget's disease typically involve the skull and facial bones.⁶ Multiple GCTs of Paget's disease usually behave less aggressively clinically and show a good response to systemic steroid therapy alone. Bone scintigraphy is useful to follow patients with multifocal GCTs.⁴

Clinical Presentation

Otalgia, headache, hearing loss, and cranial nerve palsies are the most common presenting symptoms

of temporal bone GCTs. Headache is often the initial symptom followed by cranial nerve palsies.⁸ Hearing loss is typically conductive as a result of infratemporal fossa invasion with resultant occlusion of the eustachian tube. Sensorineural, or mixed, hearing loss can also occur if there is compression of the contents within the IAC or frank involvement of the otic capsule as seen in the case studies.⁹ Tumors that involve the mastoid, middle ear, or jugular bulb often present with conductive hearing loss and a mass in the middle ear. Lesions involving the petrous apex often present with vertigo and/or sensorineural hearing loss. If lesions extend to involve the sphenoid bone, headache and diplopia are not uncommon.¹ Headache and pain were the initial presentation in 39% of 23 previously reported cases of temporal bone GCTs.¹⁰ A conductive hearing loss was found in the majority of the patients presenting with hearing loss—22% presented with a supra-auricular, postauricular, or external auditory canal mass; 17% presented with facial nerve weakness or paralysis. Cranial nerve impairment was present in 56% of patients, the most common of which were facial, trigeminal, and abducens.¹⁰

Differential Diagnosis

The differential diagnosis of GCTs involving the temporal bone is listed in Table 1. The principal diagnostic dilemma consists of distinguishing GCT from giant-cell reparative granuloma (GCRG) because the presentation can be similar; however, the prognosis differs.^{11–15} Giant-cell reparative granuloma, unlike a GCT, is a granulomatous, not neoplastic, process.^{11–13} It most commonly arises in the mandible or maxilla of females.^{14,16} These lesions are thought to arise in the periosteal connective tissue from intraosseous hemorrhage as a result of trauma, as opposed to GCTs that arise from the connective tissue in bone marrow.¹³ On histopathological examination, GCRG demonstrates fewer giant cells, which are arranged in groups around hemorrhagic foci, and numerous spindle-shaped

Table 1 Differential Diagnosis

Chondrosarcoma
Osteoblastoma
Osteolytic metastasis
Brown tumor of hyperparathyroidism
Aneurysmal bone cyst
Fibrous dysplasia
Chondroblastoma
Ossifying fibroma
Giant-cell reparative granuloma

Data from Rock JP, Mahmood A, Cramer HB. Giant cell tumor of the skull base. *Am J Otol* 1994;15:268–272; Larsson SE, Lorentzon R, Boquist L. Giant-cell tumor of bone: a demographic, clinical, and histopathological study of all cases recorded in the Swedish Cancer Registry for the years 1958 through 1968. *J Bone Joint Surg Am* 1975;57:167–173; and O'Donnell RJ, Springfield DS, Motwani HK, Ready JE, Gebhardt MC, Mankin HJ. Recurrence of giant-cell tumors of the long bones after curettage and packing with cement. *J Bone Joint Surg Am* 1994;76:1827–1833.

fibroblastic cells.⁴ Giant-cell reparative granulomas tend to have lower recurrence rates (10 to 15%) when compared with GCTs (0 to 47%).^{14,16}

Pathology

Giant-cell tumors are thought to arise from non-osteogenic stromal cells of bone marrow at the epiphysis in cartilaginous bone.⁷ Given the lack of pathognomic radiographic features, a biopsy of a solid (not cystic) component of the lesion is the only definitive way to confirm the diagnosis of GCTs.^{4,8} On gross examination, these lesions appear gray to yellow brown with small cystic areas and gray-white necrotic regions.¹⁷

Three different cell types are found in GCTs of bone: (1) multinucleated giant cell, (2) round monocyte-resembling cell, and (3) spindle-shaped fibroblast-like stromal cell.^{4,18} The latter of these is the proliferating component of the tumor. This stromal cell, which may be of osteoblastic origin, secretes monocyte chemoattractants that are essential for osteoclast differentiation. The multinucleated giant cell resembles an osteoclast and is able to resorb bone. Thus the giant cell and monocyte are the reactive components of the tumor, and the stromal spindle cell is responsible for tumor

proliferation.^{1,18} Mitotic figures are almost always present in these lesions.⁶ The multinucleated giant cells are evenly distributed throughout the tumor and can display up to 100 nuclei, as opposed to osteoclasts, which have 3 to 15 nuclei.^{4,18} A polyclonal proliferation pattern has been demonstrated in GCTs, suggesting a mesenchymal as opposed to a hematopoietic origin.¹⁹

Several new methodologies to distinguish GCTs from other giant cell-rich neoplasms have been reported.²⁰ One study, which examined 17 GCTs, identified an isoform of p63 in the mononuclear cell population in all tumor specimens. Expression of p63 was also identified in a high proportion of aneurysmal bone cyst and chondroblastoma tumor specimens. Detection of p63 was not seen in central giant-cell granuloma, GCT of tendon sheath, and pigmented villonodular synovitis.²⁰ These results suggest that immunostaining tumor specimens for p63 may allow a more objective means of differentiating GCTs from GCRGs.

Recurrence rates for GCTs is 10 to 40% in the orthopedic literature.²¹ Antal et al, using DNA-cytophotometry, demonstrated a link between DNA ploidy and risk of recurrence in 69 cases of GCT in the extremities.²¹ Thirty-one percent of primary, untreated GCTs with diploidy had eventual recurrence as opposed to 64% of aneuploid tumors. In 23 recurrent GCTs, only five cases (22%) had diploidy, whereas 18 cases (78%) had aneuploidy. The overall recurrence rate in diploid tumors was 38% as opposed to 75% in the aneuploid group ($p = 0.0106$).²¹ Increasing levels of the tumor proliferation marker Ki-67 were identified in tumor recurrences.²¹

Giant-cell tumors of bone appear to be capable of malignant degeneration.²² Malignant GCTs are classified as primary or secondary, with the latter being more common. Secondary malignant GCTs occur 3 or more years after surgery and/or radiation. These lesions are high-grade sarcomas originating in a previous area of biopsy-confirmed GCT.²² Primary malignant GCTs are composed of high-grade sarcoma adjacent to benign GCT with no history of previous treatment. The incidence of

primary or secondary malignant GCTs was 1.8% in one series. The histological subtypes found in malignant GCTs include: osteosarcoma, malignant fibrous histiocytoma, and fibrosarcoma.²² The average latency period for the development of secondary malignant GCTs appears to be longer for patients who had surgery (18 years) versus those who had radiation (9 years).²² The rate of malignant transformation in previously irradiated lesions is between 5 and 10%.⁴ There appears to be a benign metastatic variant of GCT, which occurs in 2 to 5% of cases. This particular variant has a benign histological appearance and most commonly metastasizes to the lung.⁴ The prognosis for patients with primary or secondary malignant GCTs is poor, with a slightly better outcome seen in primary malignancies.²²

Radiology

Magnetic resonance imaging is the ideal study to determine soft tissue extension, whereas CT can determine the extent of bony erosion.⁴ Giant-cell tumors usually appear hypointense on T1 and T2 imaging and enhance with the administration of contrast.⁴ Expansion and/or extension through bone with prominent bony trabeculation and multiple areas of loculation are often evident on CT.^{2,4} Calcifications are unusual and tend to lead more toward the diagnosis of chordoma, chondrosarcoma, craniopharyngioma, and meningioma.⁹ Giant-cell reparative granuloma has a similar radiographic appearance but typically is located in the mandible, maxilla, hands, or feet. A characteristic donut sign (increased uptake with central photopenia) is present in 57% of cases on bone scintigraphy.⁴

Management

Complete surgical excision is the treatment of choice for GCTs outside of the head and neck.^{1,8} In the orthopedic literature, recurrence rates as high

as 60% have been reported with curettage alone, whereas total excision is associated with only a 7% recurrence rate.^{11,23-26} In a series of 137 patients with GCTs of the axial skeleton, the local recurrence rate after curettage alone was 17%.¹⁶ When these patients were analyzed further, intraosseous tumors had a significantly lower recurrence rate (7%) with curettage than did tumors with extraosseous extension (29%).¹⁶ The curettage technique, described in the orthopedic literature, involves removal of the tumor from the intraosseous cavity followed by removal of the surrounding bone within the tumor cavity with a high-speed drill. The resulting defect is then filled with various materials: bone cement, autologous bone, or allogenic bone.^{25,26} A similar technique was used in our two cases. In each case, after the tumor was removed in a piece-meal fashion, the underlying bone was carefully removed with a high-speed drill to ensure a gross total resection.

The use of radiation therapy remains controversial. A single modality extremity radiation therapy protocol using 40 to 60 Gy over 3 to 6 weeks yielded 85% local control rate, as determined by examination, resolution of pretreatment symptoms, and radiographic studies.²⁷ Two out of 20 patients in this series had recurrences. Fourteen patients were treated after limited surgery or as primary modality, and six patients were treated for recurrences. Complete or partial recovery was noted in the four patients with pretreatment neurological deficits. Tumor location, soft tissue extension, and treatment regimen did not seem to alter outcomes. Two patients developed lung metastasis in disease that was locally controlled. There was no evidence of malignant transformation in any irradiated tumor at last follow-up.²⁷ In a study of 21 patients, Malone et al demonstrated a 90% local control rate in primary and recurrent GCTs. Fourteen of these 21 patients received surgical treatment in addition to radiation therapy. The two failures were ultimately salvaged, creating a final cure rate of 100%. No patients have been found to have malignant transformation on follow-up that ranged from 2.2 to 34.1 years. Doses less than 30 Gy with

prolonged fractionation result in increased local recurrence and are not recommended.²⁸ Recurrence in lesions of the extremities is common, especially within the first 2 years.¹ Overall, the recurrence rate of lesions involving the axial skeleton is 2 to 25%, and 80 to 90% of these recur within the first 2 years.⁴

The morbidity of sacrificing crucial, adjacent neurovascular structures within the head and neck often makes an en bloc or complete excision an unrealistic option in lesions involving the temporal bone. Curettage with or without radiation was an accepted procedure in Glasscock and Hunt's original description of two cases involving the temporal bone.¹ In a series of 15 patients with GCT involving the skull or skull base treated at the Mayo clinic, all but one patient were treated with debulking, curettage, or subtotal removal followed by postoperative external beam radiation therapy (XRT). Of these 15 patients, two expired as a direct result of tumor recurrence. Six patients were alive without disease, and four were alive with persistent tumor.⁶ Malignant degeneration as a result of XRT has been reported in the range of 7 to 29% from earlier reports issued before the use of megavoltage treatment.⁹ Radiotherapy may be useful as an adjunct, but when used as a single modality recurrence rates as high as 63% have been reported.^{1,8} Recurrence rates as high 50% in a 4-year period have been found for skull base lesions that have not been completely excised.² A more recent report describes a patient who has had no recurrence 6 years following surgical excision and postoperative radiotherapy of an extensive temporal bone GCT.²⁹

The two patients treated with curettage only in the current series were free of disease 2 years after surgery, as determined by clinical examination and radiographic follow-up. Wide local excision has yielded recurrence rates < 10%; however, this often proves difficult in the temporal bone given the complex anatomy and adjacent vital structures.⁹ The use of curettage in the two cases presented in this series has resulted in minimal morbidity and no evidence of recurrence to date. The lack of recur-

rence in the two cases presented is difficult to explain. A possible explanation may be the different embryological development of the skull base compared with the extremities. Another, more likely explanation for our success with the curettage technique is our very limited sample size of two patients. Giant-cell tumors rarely involve the temporal bone, thus making meaningful analysis of treatment modalities in this location challenging.

CONCLUSION

Giant-cell tumors represent a rare lesion involving the temporal bone. Since these lesions were first reported, a majority of authors have recommended subtotal temporal bone resections for complete surgical excision. This is in concordance with treatment paradigms for GCTs when they arise in the axial skeleton. The current study presents a less radical, but effective approach in the treatment of these lesions by means of curettage. In the two patients where this treatment approach was used, there has been no evidence of recurrence despite a prior attempt at complete excision in one of the patients.

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