



Complete Summary

GUIDELINE TITLE

ACR Appropriateness Criteria™ for orbits, vision and visual loss.

BIBLIOGRAPHIC SOURCE(S)

Hasso AN, Drayer BP, Anderson RE, Braffman B, Davis PC, Deck MD, Johnson BA, Masaryk T, Pomeranz SJ, Seidenwurm D, Tanenbaum L, Masdeu JC. Orbits, vision, and visual loss. American College of Radiology. ACR Appropriateness Criteria. Radiology 2000 Jun; 215(Suppl):579-87. [23 references]

COMPLETE SUMMARY CONTENT

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SCOPE

DISEASE/CONDITION(S)

Disorders of the orbit and optic nerve

GUIDELINE CATEGORY

Diagnosis

CLINICAL SPECIALTY

Endocrinology
Family Practice
Internal Medicine
Neurology
Ophthalmology
Pediatrics
Radiology

INTENDED USERS

Health Plans
Hospitals
Managed Care Organizations
Physicians
Utilization Management

GUIDELINE OBJECTIVE(S)

To evaluate the appropriateness of initial radiologic examinations for disorders of the orbit and optic nerve

TARGET POPULATION

Patients with disorders of the orbit and optic nerve

INTERVENTIONS AND PRACTICES CONSIDERED

1. Plain films
2. Doppler sonography
3. Computed tomography:
 - Plain
 - With contrast
4. Magnetic resonance imaging:
 - Plain
 - With contrast
5. Magnetic resonance angiography

MAJOR OUTCOMES CONSIDERED

Utility of radiologic examinations in differential diagnosis

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

The guideline developer performed literature searches of recent peer-reviewed medical journals, primarily using the National Library of Medicine's MEDLINE database. The developer identified and collected the major applicable articles.

NUMBER OF SOURCE DOCUMENTS

The total number of source documents identified as the result of the literature search is not known.

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Expert Consensus (Delphi Method)
Weighting According to a Rating Scheme (Scheme Not Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Not applicable

METHODS USED TO ANALYZE THE EVIDENCE

Systematic Review with Evidence Tables

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

One or two topic leaders within a panel assume the responsibility of developing an evidence table for each clinical condition, based on analysis of the current literature. These tables serve as a basis for developing a narrative specific to each clinical condition.

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus (Delphi)

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

Since data available from existing scientific studies are usually insufficient for meta-analysis, broad-based consensus techniques are needed to reach agreement in the formulation of the Appropriateness Criteria. Serial surveys are conducted by distributing questionnaires to consolidate expert opinions within each panel. These questionnaires are distributed to the participants along with the evidence table and narrative as developed by the topic leader(s). Questionnaires are completed by the participants in their own professional setting without influence of the other members. Voting is conducted using a scoring system from 1-9, indicating the least to the most appropriate imaging examination or therapeutic procedure. The survey results are collected, tabulated in anonymous fashion, and redistributed after each round. A maximum of three rounds is conducted and opinions are unified to the highest degree possible. Eighty (80) percent agreement is considered a consensus. If consensus cannot be reached by this method, the panel is convened and group consensus techniques are utilized. The strengths and weaknesses of each test or procedure are discussed and consensus reached whenever possible.

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Not applicable

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Internal Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

Criteria developed by the Expert Panels are reviewed by the American College of Radiology (ACR) Committee on Appropriateness Criteria and the Chair of the ACR Board of Chancellors.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

ACR Appropriateness Criteria™

Clinical Condition: Orbits, Vision and Visual Loss

Variant 1: Infant or child with orbital asymmetry, proptosis and visual loss.

Radiologic Exam Procedure	Appropriateness Rating	Comments
Computed tomography	6	
Computed tomography with contrast	6	
Magnetic resonance imaging	6	
Magnetic resonance imaging with contrast	6	
Magnetic resonance angiography	4	
Plain films	4	
<p><u>Appropriateness Criteria Scale</u></p> <p>1 2 3 4 5 6 7 8 9</p> <p>1=Least appropriate 9=Most appropriate</p>		

Clinical Condition: Orbits, Vision and Visual Loss

Variant 2: Child with slowly progressive unilateral visual loss.

Radiologic Exam	Appropriateness	Comments
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Procedure	Rating	
Computed tomography	6	
Computed tomography with contrast	6	
Magnetic resonance imaging	6	
Magnetic resonance imaging with contrast	6	
Magnetic resonance angiography	4	
Plain films	4	
<u>Appropriateness Criteria Scale</u> 1 2 3 4 5 6 7 8 9 1=Least appropriate 9=Most appropriate		

Clinical Condition: Orbits, Vision and Visual Loss

Variant 3: Young adult with sudden onset of painless or painful visual loss.

Radiologic Exam Procedure	Appropriateness Rating	Comments
Magnetic resonance imaging	8	
Magnetic resonance imaging with contrast	8	
Computed tomography	6	
Computed tomography with contrast	6	
Magnetic resonance angiography	4	
Plain films	2	
<u>Appropriateness Criteria Scale</u> 1 2 3 4 5 6 7 8 9 1=Least appropriate 9=Most appropriate		

Clinical Condition: Orbits, Vision and Visual Loss

Variant 4: Adult patient with progressive proptosis and painful visual loss.

Radiologic Exam Procedure	Appropriateness Rating	Comments
Magnetic resonance imaging	8	
Magnetic resonance imaging with contrast	8	
Computed tomography	6	
Computed tomography with contrast	6	
Magnetic resonance angiography	4	Can be done if vascular abnormality suspected.
Plain films	3	
<p><u>Appropriateness Criteria Scale</u></p> <p>1 2 3 4 5 6 7 8 9</p> <p>1=Least appropriate 9=Most appropriate</p>		

Clinical Condition: Orbits, Vision and Visual Loss

Variant 5: Adult patient with uveitis, scleritis, and visual loss.

Radiologic Exam Procedure	Appropriateness Rating	Comments
Magnetic resonance imaging	8	
Magnetic resonance imaging with contrast	8	
Computed tomography	6	
Computed tomography with contrast	6	
Magnetic resonance angiography	4	
Doppler sonography	4	
Plain films	2	

Appropriateness Criteria Scale

1 2 3 4 5 6 7 8 9

1=Least appropriate 9=Most appropriate

Clinical Condition: Orbits, Vision and Visual Loss

Variant 6: Elderly patient with uni- or bilateral proptosis.

Radiologic Exam Procedure	Appropriateness Rating	Comments
Computed tomography	6	Usually only computed tomography or magnetic resonance imaging should be done depending on clinical preference.
Computed tomography with contrast	6	Usually only computed tomography or magnetic resonance imaging should be done depending on clinical preference.
Magnetic resonance imaging	6	Usually only computed tomography or magnetic resonance imaging should be done depending on clinical preference.
Magnetic resonance imaging with contrast	6	Usually only computed tomography or magnetic resonance imaging should be done depending on clinical preference.
Magnetic resonance angiography	4	
Doppler sonography	4	
Plain films	2	

Appropriateness Criteria Scale

1 2 3 4 5 6 7 8 9

1=Least appropriate 9=Most appropriate

Clinical Condition: Orbits, Vision and Visual Loss

Variant 7: Young adult patient with recent head injury and blindness.

Radiologic Exam Procedure	Appropriateness Rating	Comments
Computed tomography	8	
Magnetic resonance imaging	8	Usual ferromagnetic precautions.
Plain films	5	
Computed tomography with contrast	4	
Magnetic resonance imaging with contrast	4	
Magnetic resonance angiography	4	
Doppler sonography	2	
<u>Appropriateness Criteria Scale</u> 1 2 3 4 5 6 7 8 9 1=Least appropriate 9=Most appropriate		

Summary

Primary diseases of the orbit may present with proptosis with or without visual disturbances. Proptosis refers to abnormal protrusion of the globe and may be differentiated from exophthalmos, which refers to abnormal prominence of the globe. Clinically, it may not be possible to differentiate these two entities, but once imaging is performed, the differentiation is clear.

Proptosis can be classified in one of three anatomic locations: ocular or bulbar; extraorbital; or, infectious and inflammatory lesions. Ocular or bulbar disorders typically cause exophthalmos, unless there is extraocular spread of the mass lesion. Many cases of proptosis are due to primary retrobulbar disorders. Extraorbital neoplasms of the face, paranasal sinuses, nasal cavities, or frontal cranial fossae can also cause proptosis. Similarly, a variety of infectious and inflammatory lesions can cause proptosis including cellulitis, abscess formation, mucoceles, inflammatory orbital syndrome or orbital pseudotumor, sarcoidosis, or Wegener's granulomatosis.

Several types of congenital or developmental lesions may be the cause of proptosis, such as macrophthalmia, colobomatous cysts, naso-orbital cephaloceles, or developmental fibro-osseous lesions such as fibrous dysplasia. Visual loss may be seen in infants and children with congenital absence of portions of the eye or visual system as well as septo-optic dysplasia. Computed tomography and magnetic resonance imaging are complementary diagnostic procedures and may be used together. Infants without proptosis, but with visual abnormalities, are best examined with magnetic resonance imaging.

Optic Nerve and Sheath Disorders

Primary disorders of the optic nerve and optic nerve sheath typically cause visual disturbances and occasionally proptosis in cases of large neoplasms. The primary neoplasms of the optic nerve include optic nerve tumors (gliomas, astrocytomas, hamartomas) or meningiomas. There may be a para-optic component of optic nerve tumors that is characteristically seen in patients with neurofibromatosis type I. The para-optic component is due to perineural arachnoidal gliomatosis consisting of a proteinaceous material that seeds and spreads within the optic nerve subarachnoid space and is a portion of the tumor. This process may result in optic nerve elongation and resultant kinking of the nerve just posterior to the posterior portion of the globe. There is no correlation between the size of the lesion and visual loss.

Extension of tumors into the optic chiasm, optic tracts and lateral geniculate bodies of the thalami is more accurately depicted on magnetic resonance imaging than on computed tomography. The size and shape of the optic canals are best assessed in the axial projection, while the size and shape of the optic nerves are best appreciated on coronal and oblique sagittal images. Many optic nerve tumors exhibit fusiform homogeneous enhancement, while the unenhanced portions of optic nerve tumors may represent the sites of gliomatosis, rather than true neoplasm.

Meningiomas are the most common optic nerve sheath tumors, which arise from the arachnoidal coverings of the optic nerve. Visual loss and optic atrophy are the usual presenting symptoms. Most cases are seen in middle-aged or elderly patients, more often in women. The tumor may be cuff-like surrounding the optic nerves or eccentrically located on only one side of the nerve. Computed tomography scans will often demonstrate calcifications and show typical postcontrast enhancement parallel to the length of the optic nerves. Magnetic resonance scans readily depict the irregular thickening along the optic nerves and spread into adjacent meninges on the postcontrast scans. Parallel optic cysts may be identified surrounding the optic nerve immediately distal to the meningioma. This process causes trapping of the cerebral spinal fluid in the subarachnoid space and can add to the mass effect and proptosis.

Optic neuritis is seen on magnetic resonance imaging as focal or diffuse enlargement of the optic nerve associated with abnormal signal intensities and enhancement. This is caused by immunological inflammation of the optic nerve, which affects the myelin sheaths with relative preservation of the axons. As optic neuritis is the initial manifestation of multiple sclerosis in about 20% of cases and may occur at some point in the disease in approximately 50% of cases, the role of magnetic resonance imaging in the evaluation of acute optic neuritis is changing. A multicenter trial was developed to assess the efficacy of corticosteroid treatment for acute optic neuritis. This study showed that the utility of magnetic resonance imaging in establishing the diagnosis of optic neuritis was limited; however, with additional views of the cranial cavity the abnormal magnetic resonance images were able to differentiate those patients who would later develop multiple sclerosis from those who would not. Thus, magnetic resonance is a predictor of multiple sclerosis as it can help identify a subgroup of patients whose risk of developing multiple sclerosis appears to be low and therefore help to prognosticate the development of multiple sclerosis after optic neuritis.

The papilledema associated with pseudotumor cerebri may be detected on computed tomography or magnetic resonance imaging scans as enlargement of the optic nerve sheaths. If severe, there is a reversal of the optic nerve head with bulging forward into the posterior wall of the globe. This phenomenon is more readily detected on computed tomography than magnetic resonance imaging because of the chemical shift artifact inherent to the magnetic resonance studies. There is some correlation between the severity of the visual loss and the detection of enlarged nerves with reversed nerve heads. Patients with more severe visual loss demonstrate more frequent and more severe reversal of the optic nerve head.

Optic Nerve Neuropathies

Isolated visual disturbances may be caused by a variety of optic nerve neuropathies, caused by radiation, chemotherapy, compressive phenomena, or ischemia. Radiation-induced optic neuropathy is a rare catastrophic complication of radiation therapy regimens used to treat a variety of neoplasms of the skull base, sella, and parasellar regions. There is typically a latency period of six to 36 months following treatment. Clinically, the nerve head may appear normal, but gadolinium-enhanced magnetic resonance imaging will show patchy, linear, or confluent enhancement along the portions of the optic nerve, chiasm, or optic tract. This correlates precisely with the cause of delayed visual loss after radiation therapy. Treatment of radiation-induced optic neuropathy with corticosteroids may be helpful, although optic atrophy may develop with permanent visual loss.

Compressive optic nerve neuropathies can be caused by a variety of lesions in the orbital apex including inflammatory orbital syndrome (orbital pseudotumor), thyroid ophthalmopathy with muscular hypertrophy and edema causing compression of the intraocular portion of the optic nerve, or other systemic diseases with orbital manifestations such as sarcoidosis or Wegener's granulomatosis.

Compression of the optic nerve may occur as a result of cavernous carotid fistulae, arteriovenous malformations or orbital varices. Such vascular anomalies may produce retrograde flow through the ophthalmic vessels with subsequent dilatation of the orbital veins and passive congestion of the orbital tissues. This leads to progressive increase in the intraocular pressure and subsequent decrease in visual acuity, or even blindness. Spontaneous thrombosis of the ophthalmic veins occasionally occurs and may aggravate the mass effect within the apex of the orbit and the compressive neuropathy.

Computed tomography imaging will demonstrate the dilated ophthalmic veins, facial veins and other regional venous structures along with enlargement of the cavernous sinus. Large edematous extraocular muscles and other periorbital structures may be identified. These findings are optimally seen with magnetic resonance imaging, particularly as the addition of magnetic resonance angiography allows for flow assessments along with the static morphologic changes. In some cases, conventional angiography may be required to make the definitive diagnosis, although this is most commonly used in conjunction with therapeutic interventional procedures.

Traumatic optic neuropathy results in blindness following optic nerve injury. The injury is typically from head trauma without direct effects on the globe or retina. The demonstration of traumatic optic neuropathy may be delayed as the patients may present with depressed levels of consciousness. Post-traumatic visual loss can be evaluated by investigation of the soft tissue and osseous structures around the optic nerve and chiasm. Computed tomography scans, typically with thin-section studies, are the most useful. Such images offer secondary signs of injury to the optic nerve such as dehiscence of bony fragments into the optic nerve canal, narrowing of the optic canal, or significant bony separations which indicate likely optic nerve injury. Magnetic resonance images have been more sensitive indicators of optic nerve edema or avulsion, although the benefit of such information is unclear. Response to steroid therapy or orbital surgical decompressive procedures remains controversial and shows no clear-cut advantages.

Inflammatory Orbital Syndrome

Inflammatory orbital syndrome (orbital pseudotumor, inflammatory fibromyotendinitis) may appear as an acute or chronic cause of proptosis and visual loss that develops as a diffuse or focal entity. In the diffuse form, there is inflammatory infiltrate of the orbital fat, extraocular muscles and adnexal structures, particularly in the orbital apex. The more focal forms of inflammatory orbital syndrome commonly involve the tendinous portions of the extraocular muscles (myositic form), the uveal structures (anterior form), the scleral region (posterior form), or the lacrimal gland (lacrimal form). All forms of the disease show extensive infiltration that is histologically composed of polyclonal lymphocytes, plasma cells, neutrophils, and macrophages with various amounts of fibrosis. Commonly, the retrobulbar fat has a "dirty" appearance.

The acute form of inflammatory orbital syndrome is typically unilateral, developing over days to weeks and is characterized by pain, swelling and tenderness. The chronic form of the disease may occur bilaterally in approximately 10-15% of cases, develops insidiously over weeks to months, and lacks inflammatory symptoms.

Both computed tomography or magnetic resonance imaging may show intra- or extraconal soft tissue lesions that are diffuse or localized and commonly involve the orbital apices. Occasionally, there may be a well-defined mass lesion. In virtually all cases, there is prominent enhancement on postcontrast computed tomography or magnetic resonance scans. In the chronic form of the disease there is increased fibrosis in the lesions resulting in decreased signal on T2-weighted images.

Many cases of inflammatory orbital syndrome resolve following steroid therapy, but the process may progress to a lymphoproliferative disorder or lymphoma. The differentiation of inflammatory orbital syndrome from lymphoma is based on histopathologic examination with a predominant polyclonal lymphocytic population in the earlier disorder and a monoclonal lymphocytic process in the latter disorder. Computed tomography or magnetic resonance scans may be used to follow the course of the illness until it resolves or recurs in the chronic form of the disease.

As previously mentioned, there may be involvement of the optic nerve resulting in alterations of visual acuity and/or involvement of the ipsilateral cavernous sinus. When there is secondary thrombosis of the sphenoidal veins or cavernous sinus, a painful ophthalmoplegia results with the presumptive diagnosis of Tolosa-Hunt syndrome. Although usually confined to the orbital soft tissues, inflammatory orbital syndrome can produce bone destruction or extraorbital extension.

A small subset of patients with isolated ocular manifestations of inflammatory orbital syndrome have posterior scleritis. These patients are seen for ocular pain and proptosis including limitation of gaze, scleral effusions, disc edema, and intraocular hemorrhages. Posterior scleritis shows inflammatory signs in the sclera with thickening of the posterior coat of the eye (sclera) that may be identified as areas of enhancement on computed tomography or magnetic resonance imaging images. The thickened sclera enhanced by contrast, presents as a so-called "ring sign." This phenomenon may be more clearly seen on computed tomography, if magnetic resonance imaging images do not include fat saturation with the postcontrast images. The inherent chemical shift artifact with conventional magnetic resonance images may preclude the identification of posterior scleritis.

Sarcoidosis (neurosarcoidosis) and Wegener's granulomatosis both simulate inflammatory orbital syndrome, lymphoproliferative disorders, or metastatic neoplasms. There are no key clinical differentiating features nor is the response to corticosteroid therapy an indication of the etiology. The definitive diagnosis is made by biopsy and is often essential in order to direct primary treatment.

Endocrine Disorders

Thyroid ophthalmopathy (Graves' disease) may be detected in patients who are hyper-, hypo-, or euthyroid. Most (25-50%) of these cases occur in patients with Graves' hyperthyroidism and occasionally in patients with Hashimoto's thyroiditis. The female-to-male ratio is 4 to 1 and approximately 15% of patients are under age 15. In all age groups, approximately 15% of unilateral orbital proptosis and the majority of bilateral proptosis are secondary to thyroid ophthalmopathy.

The disease presents with eyelid lag, upper lid retraction, diffuse conjunctival edema, and vascular injection at the insertion of the rectus muscles. The disease is characterized as an immune disorder with both cell-mediated and humoral components. The clinical features are secondary to overproduction of glycosaminoglycans, which are produced by stimulated fibroblasts. The overstimulation of fibroblasts is thought to be centered around the T-cell lymphocytes which recognize fibroblasts' cross-reactive antigens and release cytokines. The overly-produced glycosaminoglycans deposit into muscle bellies of the extraocular muscles in a perivascular location. This results in endomesial fibrosis secondary to the mucopolysaccharide deposits.

On computed tomography and magnetic resonance studies, there is enlargement of one or more of the extraocular rectus muscles. Recent studies show multiple muscle involvement as being more common than one or two isolated muscle involvement. Although the posterior and middle third of the muscle bellies are affected, the tendons near their insertions are usually not thickened. The inherent soft tissue contrast of magnetic resonance scans provides elegant morphologic information regarding the involvement of the extraocular muscles in patients with

thyroid ophthalmopathy. The main role of imaging is in demonstrating the relationship of the extraocular muscles to the optic nerve at the orbital apex, particularly if surgery is contemplated. The ability to measure the T2 signal intensity on magnetic resonance imaging has useful information both in determining which patients may benefit from corticosteroid therapy (those with high T2 values), and/or which patients require combined therapies including cyclosporin (based on a measurable response on serial magnetic resonance images).

Disorders of Size or Shape of the Globe

A staphyloma represents a diffusely enlarged globe with thin scleral margins resulting from degeneration of the bulbar coverings. The lesion is thought to be secondary to the normal aging process and is only seen in the elderly population, either uni- or bilaterally. Imaging studies will demonstrate the enlarged globe with thin walls and no other lesions. Most often, enlargement is an incidental finding on images that were obtained for unrelated conditions. A diffusely enlarged globe is seen in patients with severe axial myopia, which, unlike a staphyloma, is a heritable condition treated by corrective lenses or keratotomy.

Retinal/Choroidal/Subhyloid Detachments

Serous choroidal detachments result from inflammatory diseases (uveitis, scleritis) or by accidental perforation of the eyeball. As the edema of the choroid increases, fluid accumulates in the subchoroidal potential space. Hemorrhagic choroidal detachments often occur after a contusion, a penetrating injury, or as a complication of intraocular surgery. The differentiation of choroidal effusion from choroidal hemorrhage may be obtained on varying magnetic resonance sequences. There is a crescentic ring-shaped area of increased signal on both T1- and T2-weighted images. With choroidal hemorrhages, the signal intensity varies according to the age of the hemorrhage. In acute hemorrhages, computed tomography may be more specific, showing the increased density of subchoroidal hemorrhage.

Retinal detachments as a complication of systemic diseases such as hypertension or diabetes are fairly common and rarely require imaging. Retinal detachments may, however, occur with primary ocular neoplasms such as retinoblastomas in childhood-aged patients and uveal malignant melanomas in adults and elderly patients. Ocular sonography may be more accurate in detecting small tumors, however, enhanced magnetic resonance images can be useful in determining the true extent of lesions beyond the ocular structures and also demonstrating associated retinal detachments. Some improvement in the differential diagnosis is based on postcontrast T1-weighted images which are most helpful in detecting uveal melanomas and in differentiating melanomas from subretinal fluid collections. There is enhancement in the case of neoplasms, but not from fluid collections. The differentiation of an amelanotic melanoma from a hemorrhagic subretinal hemorrhage is based on both the pre- and post-contrast T1-weighted images. Of note are metastatic lesions to the retina or certain inflammatory conditions that cannot be consistently differentiated from primary uveal melanomas. Doppler sonography may help detect vascularity within an intraocular tumor and help differentiate such entities from nonvascular choroidal, subretinal or subhyloid effusions, or hematomas.

CLINICAL ALGORITHM(S)

Algorithms were not developed from criteria guidelines.

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The recommendations are based on analysis of the current literature and expert panel consensus.

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

Appropriate selection of radiologic exams to diagnose disorders of the orbit and optic nerve in patients because some patients may benefit from treatments, such as corticosteroid therapy.

Subgroups Most Likely to Benefit:

For example, radiation-induced optic neuropathy and patients with high T2 values on magnetic resonance imaging may respond to treatments such as corticosteroid therapy.

POTENTIAL HARMS

Not stated

QUALIFYING STATEMENTS

QUALIFYING STATEMENTS

An American College of Radiology (ACR) Committee on Appropriateness Criteria and its expert panels have developed criteria for determining appropriate imaging examinations for diagnosis and treatment of specified medical condition(s). These criteria are intended to guide radiologists, radiation oncologists and referring physicians in making decisions regarding radiologic imaging and treatment. Generally, the complexity and severity of a patient's clinical condition should dictate the selection of appropriate imaging procedures or treatments. Only those exams generally used for evaluation of the patient's condition are ranked. Other imaging studies necessary to evaluate other co-existent diseases or other medical consequences of this condition are not considered in this document. The availability of equipment or personnel may influence the selection of appropriate imaging procedures or treatments. Imaging techniques classified as investigational by the U.S. Food and Drug Administration (FDA) have not been considered in developing these criteria; however, study of new equipment and applications should be encouraged. The ultimate decision regarding the appropriateness of any specific radiologic examination or treatment must be made

by the referring physician and radiologist in light of all the circumstances presented in an individual examination.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better
Living with Illness

IOM DOMAIN

Effectiveness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

Hasso AN, Drayer BP, Anderson RE, Braffman B, Davis PC, Deck MD, Johnson BA, Masaryk T, Pomeranz SJ, Seidenwurm D, Tanenbaum L, Masdeu JC. Orbits, vision, and visual loss. American College of Radiology. ACR Appropriateness Criteria. Radiology 2000 Jun; 215(Suppl):579-87. [23 references]

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

1999

GUIDELINE DEVELOPER(S)

American College of Radiology - Medical Specialty Society

SOURCE(S) OF FUNDING

The American College of Radiology (ACR) provided the funding and the resources for these ACR Appropriateness Criteria™.

GUIDELINE COMMITTEE

ACR Appropriateness Criteria™ Committee, Expert Panel on Neurologic Imaging

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

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FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

Not stated

GUIDELINE STATUS

This is the current release of the guideline.

The ACR Appropriateness Criteria™ are reviewed after five years, if not sooner, depending upon introduction of new and highly significant scientific evidence. The next review date for this topic is 2004.

GUIDELINE AVAILABILITY

Electronic copies: Available from the [American College of Radiology \(ACR\) Web site](#).

Print copies: Available from ACR, 1891 Preston White Drive, Reston, VA 20191. Telephone: (703) 648-8900.

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

This summary was completed by ECRI on July 31, 2001. The information was verified by the guideline developer as of August 24, 2001.

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