Oligodendrocyte

Oligodendrocytes are glial cells that form the myelin of the central nervous system and are present in both gray and white matter.

From: Bancroft's Theory and Practice of Histological Techniques (Seventh Edition), 2013

Related terms:

Serositis, Multiple Sclerosis, Lesion, Myelin, Central Nervous System, Astrocyte, Microglia, Demyelination

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Techniques in neuropathology

J. Robin Highley, Nicky Sullivan, in Bancroft's Theory and Practice of Histological Techniques (Seventh Edition), 2013

Oligodendrocytes

Oligodendrocytes are glial cells that form the myelin of the central nervous system and are present in both gray and white matter. As noted above, a single oligodendrocyte may myelinate axons from multiple neurons. In H&E and cresyl violet preparations, oligodendrocytes have small (7 μm) round to oval nuclei with compact chromatin. The cytoplasm is indistinct from the surrounding neuropil although oligodendroglial tumors may show artifactual perinuclear halos in paraffin sections. Silver preparations to demonstrate oligodendroglia (Penfield 1928; Stern 1932) are rarely used, now replaced by immunohistochemistry to myelin basic protein and myelin associated glycoprotein label oligodendrocyte processes (see above). However, these epitopes are not expressed by oligodendroglial tumors (Nakagawa et al. 1986). Olig2 is a transcription factor that regulates oligodendroglial development and is expressed by the nuclei of oligodendrocytes and oligodendroglial tumors (Yokoo et al. 2004). Sadly, it is not specific for oligodendrogliomas as it labels other morphologically similar tumors (Preusser et al. 2007) and is also expressed by astrocytomas (Ligon et al. 2004). A reliable immunohistochemical marker to distinguish oligodendroglioma from astrocytomas has not yet been found. Finally,

deletion of chromosomes 1p and 19q (most commonly investigated by fluorescence *in situ* hybridization) is a well-recognized molecular feature of oligodendrogliomas and appears to be associated with a better prognosis and response to treatment (Bourne & Schiff 2010).

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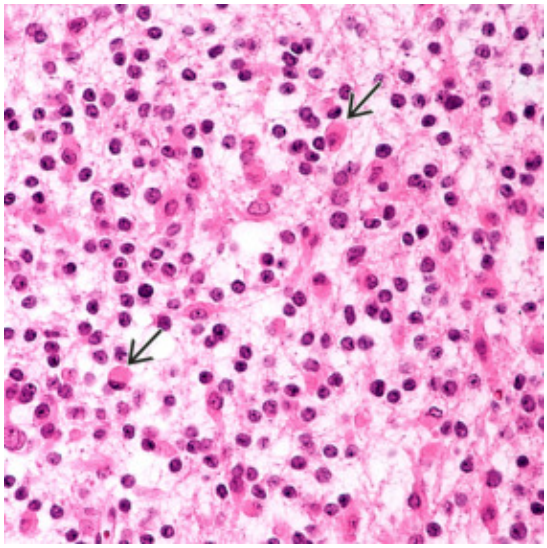
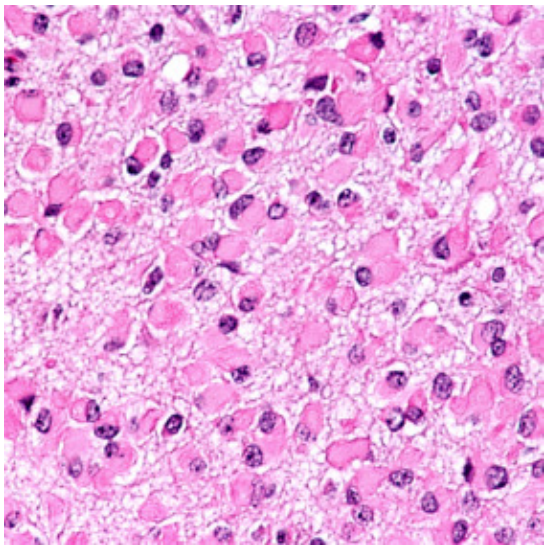
Oligodendroglioma

In Diagnostic Pathology: Neuropathology (Second Edition), 2016

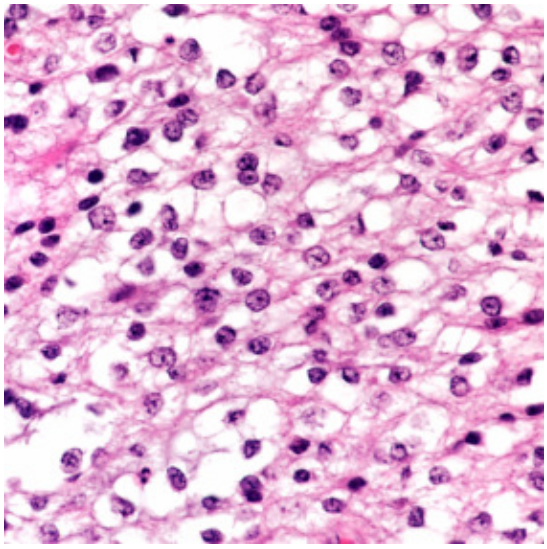


Gliofibrillary OligodendrocytesCells with features suggesting astrocytic differentiation , gliofibrillary oligodendrocytes in this case, are not uncommon in low-grade oligodendrogliomas, but are more frequent when grade III.

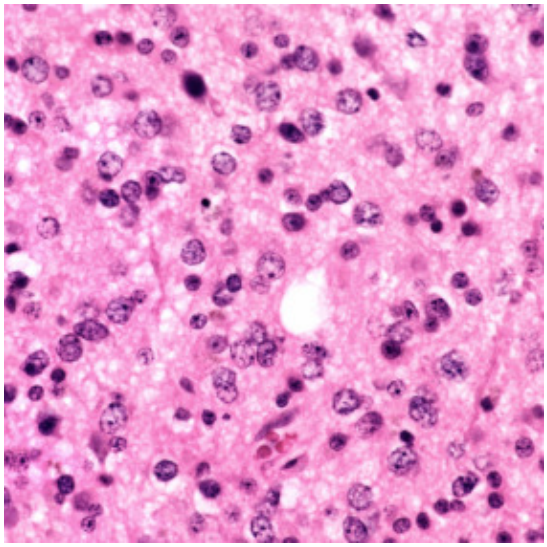


MinigemistocytesAs small versions of gemistocytes, the globular rather than polygonal mini version has hyaline cytoplasm and short processes. Such cells overlap with

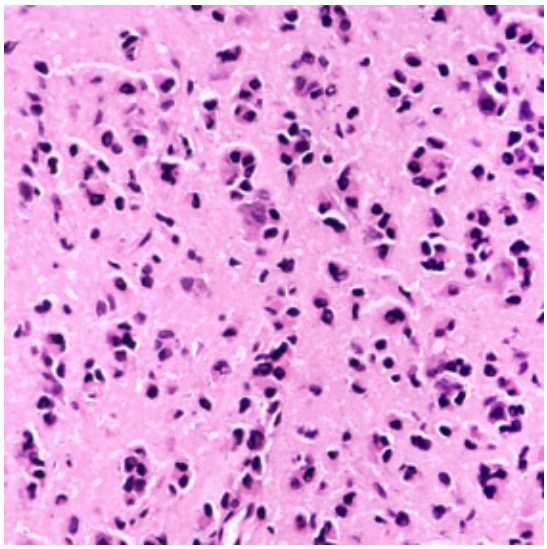
gliofibrillary oligodendrocytes, and the term minigemistocyte is often used for both. The cells will be GFAP(+).



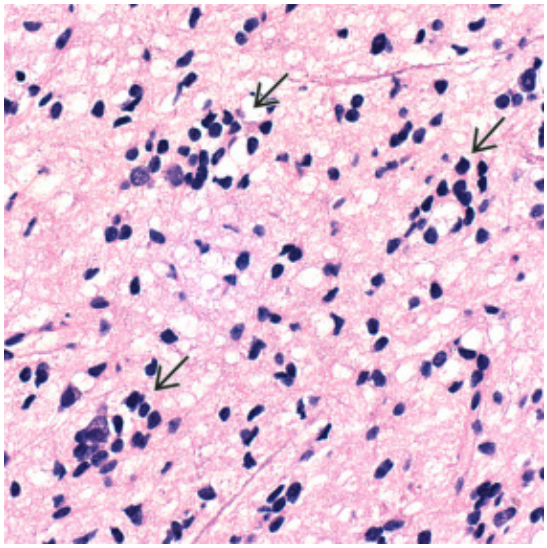
Cobweb AppearanceIn combination, perinuclear halos and parenchymal edema can produce a cobweb effect.



Minor Nuclear PleomorphismThe minor nuclear pleomorphism of this oligodendroglioma overlaps with that of diffuse astrocytoma. Nonetheless, it has the open nuclei and distinct nucleoli of oligodendroglioma. Molecular studies, 1p/19q, are often applied, even to classic lesions.



Freezing ArtifactNuclear condensation and pleomorphism are consequences of prior freezing. The prominent perineuronal satellitosis is a clue as to the oligodendroglial nature of this infiltrating neoplasm.



Freezing ArtifactNuclear angulation and hyperchromatism are artifacts of freezing. Oligodendrogliomas in frozen section controls often resemble astrocytomas. Perineuronal satellitosis is more suggestive with oligodendroglioma.

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Techniques in Neuropathology

Scott L. Nestor, in Theory and Practice of Histological Techniques (Sixth Edition), 2008

Techniques for demonstrating oligodendrocytes

The oligodendrocytes are the cells that form myelin within the white matter of the central nervous system. A single oligodendrocyte may be responsible for the myelin of numerous nerve fibers. Oligodendrocytes are also seen in gray matter, where they are thought to act as support cells for neurons. In H&E-stained sections and Nissl-stained sections, the oligodendrocyte is identified by its small (7 μm), dense, rounded nucleus. The cytoplasm is not distinguishable from the surrounding tissues but may form an artifactual 'halo' around the nucleus after formalin fixation and paraffin processing.

The demonstration of oligodendroglial cells is usually an anatomical/histological exercise and their demonstration by metal impregnation techniques is rarely called upon for diagnostic purposes. The silver carbonate method of Penfield (see p. 387) uses frozen sections of formalin-fixed tissue and will show oligodendroglial processes in very fresh human tissue. Unfortunately, autolytic processes in autopsy material frequently lead to a poor result. Weil & Davenport's method (see below) may be applied to paraffin sections but suffers from the same problem of rapid autolysis of oligodendroglia, leading to a poor result. Oligodendroglia may be demonstrated by immunohistochemical methods using antibodies to galactocerebroside, myelin basic protein, or carbonic anhydrase C (Sternberger et al 1977; Raff et al 1978; Ghandour et al 1980). Tumors derived from oligo-dendrocytes generally do not react with these antibodies (Schwechheimer et al 1992). Oligodendroglial tumors may, however, demonstrate variable immunoreactivity for some of the same stains demonstrated in astrocytic tumors (GFAP, vimentin, S100, and aberrant p53 expression), but at the present time there is no adequate marker that is sufficiently sensitive and specific enough to demonstrate tumors derived from oligodendrocytes. There is a tumor marker identified in low-grade oligodendroglioma and oligoastrocytoma which, unlike many other markers, allows one to predict the response to a specific combination chemotherapy treatment with procarbazine, lomustine, and vincristine (PCV). This marker is the 1p/19q deletion which is readily demonstrated with the FISH technique (Buckner et al 2003).

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Visual Loss

Stacy L. Pineles, Laura J. Balcer, in Liu, Volpe, and Galetta's Neuro-Ophthalmology (Third Edition), 2019

Myelinated Retinal Nerve Fibers

Lacking oligodendrocytes, the retina is usually unmyelinated. Myelinated retinal nerve fibers are anomalous white patches of myelin which are often contiguous with the optic disc and typically contained within the nerve fiber layer (see Figs. 4.34B and 6.2C).[150] When viewed with high magnification, the edges appear serrated or feathery as the myelination aligns along the nerve fibers. This feature distinguishes these lesions from cotton-wool spots. Occasionally the myelinated nerve fibers are large, extending along the temporal vascular arcades. They can also be located away from the disc in the retinal periphery. Although usually asymptomatic, some involved eyes are myopic with associated anisometropic amblyopia.[151] When the myelinated area is extensive, visual field defects may occur. With rare exceptions,[152] myelinated retinal nerve fibers are static and, when found incidentally, require no treatment. Although most cases of myelinated nerve fibers are congenital, acquired cases have been reported in disease states with disruption of the lamina cribrosa, such as optic nerve tumors,[153] optic disc drusen,[152] and papilledema.[154] It is important to recognize these benign retinal lesions in the differential diagnosis of retinal ischemia and pseudopapilledema.

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Neuronal Proliferation, Migration, Organization, and Myelination

Joseph J Volpe, in Neurology of the Newborn (Fifth Edition), 2008

Glial Proliferation and Differentiation

Astrocytes, oligodendrocytes, and microglia are the major glial cells of the CNS. Glial proliferation and differentiation are of major importance in developing brain; glial cells clearly outnumber neurons in the CNS. In fact, in human cerebral cortex, glial cells outnumber neurons by approximately 1.25 to 1 and are almost the exclusive cell type in white matter.[675]

Astrocytic and oligodendrocytic lineage, proliferation, and differentiation have been the topics of intense investigation in experimental systems in recent years,[237,676-711] and initial data also are emerging from studies of human brain.[577,676,700,712-725] The observations are not entirely consistent, but my best attempt at a synthesis is shown in Table 2-27. In general, astrocytes are generated primarily before oligodendrocytes. The progenitors of both astrocytes and oligodendrocytes initially are cells of the subventricular zone and probably radial glia (see the earlier discussion of proliferation). Radial glial progenitors may give rise to a glial restricted progenitor that

then generates astrocytes or oligodendrocytes. Proliferation of glia, unlike that of neurons, also may occur locally, during and after migration.

Astrocytes play a variety of complex nutritive and supportive roles in relation to neuronal homeostasis and in the reaction to metabolic and structural insults. For example, astrocytes avidly take up glutamate and convert it to glutamine by the action of the astrocyte-specific enzyme glutamine synthetase; this removal of glutamate from the extracellular space is crucial for protection against excitotoxic injury with ischemia, seizures, or hypoglycemia (see Chapters 5, 6, 8, and 12Chapter 5Chapter 6Chapter 8Chapter 12). Other functions include a wide variety of roles in inflammation, immune responses, production of trophic and neuroprotective factors (e.g., antioxidants), and tissue remodeling after injury.[572]

Oligodendroglial proliferation and differentiation are crucial for myelination and thus are discussed later in relation to that major developmental event. *Microglia* comprise the resident and immune cells of the brain and originate principally if not entirely from bone marrow–derived monocytes.[572] These cells enter the CNS (especially brain stem and spinal cord) in the first trimester, and in the cerebrum microglia become apparent in the second trimester within the marginal zone, the boundary of the cortical plate and subplate, and the ventricular-subventricular zones (see Table 2-27).[726-732a] A study of developing human cerebrum from 20 weeks of gestation made the striking observation that microglial cells during the second and third trimesters were primarily in the active (ameboid morphology) state and could be seen to migrate progressively from ventricular-subventricular zones to the cerebral white matter (20 to 35 weeks) and then to the cerebral cortex.[733] Migration may occur along white matter tracts, radially oriented vasculature, and residual radial glial cells.[729,733] Although the prevailing notion is that these cells *enter* the ventricular-subventricular zones through the circulation, whether any of these cells may *originate* in the ventricular-subventricular zones is unresolved. *The critical point is that the cerebral white matter is heavily populated with activated microglia during a period when developmental events are active and a variety of insults can lead to white matter injury* (see Chapters 6 and 8Chapter 6Chapter 8).

Microglial cells play key roles during brain development, involving vascularization,[729] apoptosis,[644] axonal development,[586] and later myelination.[734] In addition to these key beneficial roles, these cells, when activated by such insults as hypoxia-ischemia or infection-inflammation, can release such substances as cytokines and reactive oxygen and nitrogen species, which could injure, as "innocent bystanders," differentiating oligodendrocytes of the premature infant or neurons of the term infant (see Chapter 6).

> Read full chapter

Hypoxic-Ischemic Encephalopathy: Biochemical and Physiological Aspects

Joseph J Volpe, in Neurology of the Newborn (Fifth Edition), 2008

Maturation-Dependent Intrinsic Vulnerability of Premyelinating Oligodendrocytes in Cerebral White Matter

An intrinsic vulnerability of pre-OLs in the cerebral white matter of the human premature infant is suggested by the experimental studies, by the rarity of the lesion at later ages, by the concentration of these cells in human cerebral white matter during the peak time period for occurrence of PVL, and by their specific involvement in the lesion. To address and to clarify the issue of the maturation-dependent vulnerability of oligodendroglial precursors, we and others studied this cell lineage, identified by immunocytochemical criteria (see Chapter 2), in experimental systems, both in culture and in vivo, and in human postmortem brain. Within this lineage, as stated earlier, the data indicate that the principal cellular target in PVL is the *pre-OL, a term that includes both the* O4+ pre-oligodendrocyte and the O1+ *immature oligodendrocyte*. The data indicate that these cells are particularly vulnerable to the *two principal downstream events* in PVL (i.e., *ROS/RNS toxicity* and *excitotoxicity;* see Fig. 6-35), as discussed next.

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Molecular basis of diseases of the nervous system

Margaret Flanagan, ... Thomas J. Montine, in Essential Concepts in Molecular Pathology (Second Edition), 2020

Gliogenesis

Glial cells include astrocytes, oligodendrocytes, microglia, and NG2-positive oligodendrocyte precursor cells. Generally, gliogenesis follows neurogenesis with relatively little temporal overlap. Astrocytes appear to be produced directly from radial glia and by continued division of astrocytes and glial progenitors that maintain the potential to proliferate throughout life. In terms of progenitor lineages and molecular expression, oligodendroglia in most regions are more closely related to neurons than to astrocytes. NG2-positive cells are a more recently identified type

of glia that serve as oligodendrocyte precursors and may have special progenitor properties, such as the ability to generate neurons under certain conditions.

> Read full chapter

Normal Aging Brain

In Diagnostic Imaging: Brain (Third Edition), 2016

Microscopic Features

- Degeneration of neurons and oligodendrocytes
- Decreased myelinated fibers in subcortical WM
- Increased extracellular space, gliosis
- Iron deposition in globus pallidus, putamen
- WM capillaries lose pericytes, have thinner endothelium
- Dilated perivascular spaces of Virchow-RobinoExtension of subarachnoid space that accompanies penetrating vessels into brain to level of capillaries
- Senile plaquesoExtracellular amyloid deposits in cerebral gray matter
- Lewy bodiesoIntraneuronal clumps of □-synuclein and ubiquitin proteinso-Found in 5-10% of cognitively intact individuals
- Neurofibrillary tangles (NFTs)oTau phosphorylation, mitochondrial dysfunction may precede full NFT formationoNFTs appear in small numbers in entorhinal and transentorhinal cortices early in aging (patients ~ 60 years old)o-NFTs may induce neural dysfunction, destruction of synapses, and, eventually, neuronal death

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