DE GRUYTER OPEN

Romanian Neurosurgery (2015) XXIX 4: 445 - 457 445

DOI: 10.1515/romneu-2015-0060

Massive cerebellar infarction: a neurosurgical approach

Luis Rafael Moscote Salazar[1], Hernando Raphael Alvis-Miranda[2], Amit Agrawal[3], Gabriel Alcala-Cerra[1], Williem Calderon-Miranda[4], Umamaheswara Reddy V.[5]

[1]Neurosurgeon, University of Cartagena, Colombia
[2]Resident of Neurosurgery, University of Cartagena, Colombia
[3]Professor of Neurosurgery, Department of Neurosurgery, Narayna Medical College Hospital, Chinthareddypalem, Andhra Pradesh (India)
[4]Resident of Radiology, UNAM, Mexico DF, Mexico
[5]Assistant Professor of Radiology, Department of Radiology, Narayana Medical College Hospital, Chinthareddypalem, Nellore, Andhra Pradesh (India)

Abstract: Cerebellar infarction is a challenge for the neurosurgeon. The rapid recognition will crucial to avoid devastating consequences. The massive cerebellar infarction has pseudotumoral behavior, should affect at least one third of the volume of the cerebellum. The irrigation of the cerebellum presents anatomical diversity, favoring the appearance of atypical infarcts. The neurosurgical management is critical for massive cerebellar infarction. We present a review of the literature.
Key words: Cerebellar infarction, stroke, infratentorial infarction

Introduction

Massive cerebellar infarction is less frequent than ischemic strokes in anterior circulation; there is a huge variability on the definition. It is more frequent in younger individuals and those with less comorbidities than those that occur in the anterior circulation, generally having an embolic origin, although principal arteries occlusion of posterior circulation are distinguished. Clinically it will depend on the extension of the cerebellar compromise and the simultaneous injury to the encephalic trunk, although the clinical manifestation is non-specific. The CT and MRI are still the initial imaging studies. The most useful mode to identify the ischemic area are the diffusion sequences (DWI-ADC), given the great sensitivity. The management always emphasizes the patient stabilization, airway protection, appropriate oxygenation, arterial pressure management, glycemic control, euthermia and prevention of deep venous thrombosis and pulmonary thromboembolism. Despite all the diagnostic and therapeutic tools, described in this review, the mortality due to ischemic events in the

posterior fossa is still high, around 43%, this make us follow up to define the etiology, diagnostic and therapeutic processes, specially in the critical stage. Ischemic strokes injuring cerebellum are less frequent that those involving anterior cerebral circulation. According to the findings from different observational studies, it is estimated that infratentorial strokes correspond approximately to 3% of all ischemic events (from 1,9% to 27,2%); (1-7) however, the consequences can be devastating, in many cases, fatal. Nowadays, the exact proportion of patients with cerebellar strokes that will develop massive edema is unknown, however, it is estimated that from 17% to 54% of cases can develop edema and threat life. (8)

Massive cerebellar or space-occupying infarction: in search of a definition

In the actuality there is no concrete definition that includes objective criteria to consider a cerebellar infarction as massive or space-occupying. These criteria are variable among specialized centers, which constitute one of the principal limitations for the design, handling, and generalization of investigation works. Generally, the presence of cerebellar edema, associated to obstructive hydrocephalus, posterior fossa cistern artery or cerebral trunk compression, is a condition infallible to most definitions, which generally, do not consider the clinical state of the patient, leaving the definition to be linked only to the imaging findings. However, not all the patients with these findings will develop clinical deterioration. In a follow up by Koh and coworkers to 35 patients that developed caudal displacement of cerebellar amygdala or rostral displacement of the aqueduct of Sylvius or pontocerebellar junction on medium sagittal MRI, only 50% developed consciousness deterioration, defined by the study as decrease of 2 points in ECG from the initial assessment. (9) This is why a definition that includes only radiologic findings is imprecise and insufficient to determine the therapeutic conducts.

Etiology

The clinical and etiologic characteristics of patients with infratentorial infarction are different from those with injury to anterior circulation. The National Acute Stroke Israeli Survey (NASIS) study showed that these patients are generally younger and have less comorbidities, specially heart failure and atrial fibrillation that predispose to cardioembolic events. (10) Also it has been found that arterial dissections are less frequent. (11,12)

In a follow up of 293 patients by Tohgi and coworkers, demonstrated that approximately 24% of cerebellar infarctions are from embolic origin. Additionally, they found that most of the cerebellar infarctions were caused by obstruction of superior cerebellar artery (SCA) and posterior inferior cerebellar artery (PICA), 52% and 49% respectively; while anterior inferior cerebellar artery is only responsible in 20% of the cases. (7)

Physiopathology

After blood flow decrease to the brainstem and cerebellum, a diminution of neural parenchyma and glia capacity to carry on aerobic metabolism occurs, this causes a reduction of ATP molecules available for the Na/K pump to maintain homeostasis of these

DOI: 10.1515/romneu-2015-0060

electrolytes. At the same time, the osmotic pressure imbalance favors the increase on cellular content of water molecules, generating cytotoxic edema; while hematoencephalic barrier disruption develops vasogenic edema. (13)

Posterior fossa is a non-distensible space, whose approximate volume is between 165cm3 and 196cm3 and therefore, the tolerance limit is reduced by any expansive process, this may lead to accelerated increase in infrantentorial pressure. (14) Because of this, around 81% of cerebellar infarctions may develop radiologic signs of mass effect. (15)

Increase of edematous cerebellar tissue volume causes compression of adjacent structures, especially of fourth ventricle and brainstem; this can lead to acute obstructive hydrocephalus and major compromise to arterial flow. As the clinical picture get worse and the infratentorial pressure increases, cerebellar amygdala herniation through the foramen magnum may be present, which present clinically with deep coma, ataxic breathing, irregular shape of pupils in medial position, loss of motor response to pain stimuli and absence of ocular cephalic and ocular vestibular reflexes. The other type of cerebellar herniation is the displacement of superior vermis to supratentorial space through the tentorial foramen, also known as ascending or transtentorial herniation. This phenomenon is causes by increase of pressure gradient between anterior fossa and supratentorial space, presenting clinically as consciousness quantitative compromise, ocular motor compromise, ascending conjugated eye movement paresis is frequent, or more characteristic, paresis of conjugated descending eye movements (looking upwards spontaneously). Decerebrate and decorticate rigidity are also seen. It may lead to infarction of posterior cerebral artery. (16-18) This event may present as a complication related to the performance of procedures that decrease supratentorial pressure, such as ventricular punctures, ventriculostomies or ventriculoperitoneal shunt, however, the complication of any of these procedures is in low. Both types of cerebellar herniation conctitutes terminal stages of the onfratentorial hypertension process, and its prognosis is generally fatal. It is the most frequent cause of dead in the acute state. (8, 19-21)

Clinical presentation

The clinical manifestations will depend on the extension of the cerebellar compromise and especially of the simultaneous injury to the brainstem. The majority of distal arterial infarctions that compromise the cerebellar parenchyma cause few specific symptoms, such as vomit, walking abnormalities, cephalea and vertigo. Furthermore, the clinical signs such as dysarthria, ataxia and nystagmus are often hard to detect or may be confused with other benign disorders of peripheral vestibular system. (3) Thus, it is not surprising that about 30% of patients receive a wrong diagnosis at the moment the symptoms begin, especially when the patients are younger than 60 years old or have vertebral artery dissections or medial branch obstruction of the posterior inferior cerebellar artery, whose neurologic symptoms and signs may be subtle. (22-24) Among the clinical variables that must

increase the suspicion of a cerebellar infarction there is the presence of moderate to severe unbalance, the presence of vertigo for more that 72 hours since the beginning of the symptoms and abnormalities in the neurologic examination. (25) Nevertheless, up to 10% of cases may present vertigo as the only symptom and in these cases, more than 90% are secondary to infarction in the medial branch of the posterior inferior cerebellar artery. (26) For proper identification of these cases, one of the most useful maneuvers is cephalic impulse test, in which, the absence of a corrective shake highly decreases the probability that symptoms are caused by peripheral injuries of the vestibular system (vestibular neuritis, Ménière's disease, etc). (23, 27) Another sign with highly diagnostic value is spontaneous nistagmus, especially if it has a vertical or rotational component, or variable direction. (25) These signs have to be systematically examined in all patients with acute vertigo in order to rule out cerebellar infarctions, because when overlooked, there is a high risk of complications related to poor clinical examination, with a mortality that can go as high as 40%. (24)

TABLE 1

Syptom	SCA	AICA	PICA
Deafness	Absent	Frequent	Absent
Dizziness	Frequent	Absent	Frequent
Abnormalitis of consciousness	Frequent	Frequent	Rarely
Tinnitus	Rarely	Frequent	Absent
Vertigo	Frequent	Frequent	Very common
Nausea/vomiting	Frequent	Very common	Very common
Hallucinations	Rarely	Rarely	Absent
Cephalea/facial pain	Rarely	Absent	Rarely
Pain in extremities and trunk	Absent	Frequent	Absent

AICA: anterior inferior cerebellar artery; SCA: superior cerebellar artery; PICA: posterior inferior cerebellar artery
Modified from Manto, et al. (28)

Radiologic assessment

When a cerebellar infarction is suspected, a computed tomography scan without contrast material is still the first election imaging study; it allows differentiation of ischemic events from hemorrhagic events. (Image 1) Nevertheless, it must be considered that bone structures that form the base of skull highly decrease the sensibility to assess the posterior fossa. (3, 29) This explains why in a significant group of patients with ischemic events no abnormality is detected in the initial evaluation, therefore when a cerebellar ischemic event is suspected a magnetic resonance imaging (MRI) scan without contrast material should be performed to complete the diagnostic approach. (9, 29) However, the sensibility of standard sequences (T1, T2 and FLAIR) to detect early ischemic

DOI: 10.1515/romneu-2015-0060

changes is also low. (30) (Image 2) At the present time the most useful modality to identity the ischemic area is the diffusion sequences (DWI-ADC) which have a sensibility around 88% and 100%, even when performed during the first 24 hours after the symptoms have started. Its sensibility is also high, between 95%-100%, because of this it must be performed specifically when a cerebellar infarction is suspected. (3, 13, 30, 31) The total extension to the cerebellar tissue with diffusion restriction correlates to the final infarction territory, allowing the distinction of vascular territory that is compromised; it also allows estimating the risk of deterioration, related to the addition of hydrocephalus and/or conversion to hemorrhagic event, in the early stages. (31) Additionally, these sequences help to identify small cortical and subcortical injuries that make us suspect a cardioembolic etiology. (30)

Besides the diagnostic significance, some findings in neurologic imaging allow to estimate the risk of neurological deterioration in the acute phase. Koh and coworkers identified several factors related to adverse clinical outcomes, with their respective odds ratio (OR), among these there is brainstem deformities, (OR=15,1) and the presence of obstructive hydrocephalus (OR=26). Also, a displacement greater that 5 mm of the fourth ventricle from the middle-clival line may be helpful to estimate the clinical evolution, however, the impact seems to be lower that the one of those previously mentioned signs. (9)

The assessment of the cisterns in the posterior fossa is also a strong factor to predict deterioration. It has been demonstrated that those patients in whom they are compressed, the probability of presenting consciousness deterioration is 20 times higher. (9) Taneda and coworkers developed a scanning scale to classify the severity of compression based on the appearance of the quadrigeminal cistern in the patients with cerebellar hemorrhage, this scale has also been used for ischemic injury, including: grade I (normal), grade II (compresses) or grade III (absent). This scale correlates to the prognosis and not necessarily to the volume of affected tissue or hematoma; for this it is an indicator of infratentorial hypertension, useful in the selection of therapeutic conducts. (32)

Other imaging characteristics that predict adverse outcome are the compromise of more than two thirds of a cerebellar hemisphere and the conversion to a hemorrhagic event. (9, 33, 34) The risk of conversion to a hemorrhagic event is closely related to the diameter and total cerebellar tissue with infarction. Sakamoto and coworkers showed that infarctions with a diameter bigger than 2,7 cm or a volume greater that 4,5 cm3 have a probability of hemorrhagic conversion of 7-11 times superior to those whose extension is smaller. (31) Therefore, a strict follow-up by imaging is advisable during hospitalization, since up to 55% of them will develop a hemorrhagic conversion that is clinically significant. (3, 31)

Because of the different etiologies associated with cerebellar infarctions, different authors have recommended an imaging evaluation of the arterial system in search of underlying injuries when identification of a cardioembolic etiology is not possible in the

initial evaluation. (3) However, other study groups recommend performing them systematically, knowing that alone or coexisting alterations may be found along with other potential etiologies in up to 20% of the cases. (12,35) The most recent guidelines from the American Stroke Society suggest the anatomic vascular assessment as integral component of the initial evaluation of patients with neurologic symptoms that may be attributed to the posterior circulation or those with subclavial steal syndrome (Class I Recommendation). (35)

In the actuality, there are many diagnostic modalities available to identify arterial injuries; among these the digital subtraction angiography stands out; as well as other non-invasive methods such as ultrasonography and CT or MRI guided angiography. (36-38) The usefulness of Doppler ultrasonography is very limited, specially to assess the ostium and V1 segment of vertebral arteries, where the atherosclerotic injuries are more frequent, thus, it is not recommended for the assessment of posterior circulation. (12, 35)

Digital subtraction angiography is still the gold standard to assess the intra- and extra-cranial arterial system; however, it has several deficiencies such as availability, opportunity, requirement of contrast material, ionizing radiation exposure, costs and the risks related to the procedure itself. (39,40) Therefore, there have been several advances in non-invasive techniques with CT and MRI, which have shown that the specificity and sensibility of imaging obtained by CT are good enough to be recommended as first election method in the assessment of patients with cerebellar infarction. (35-38, 41) Although CT angiography is widely available and it is performed rapidly, the patient is exposed to high doses of ionizing radiation and contrast material and may be potentially toxic; this may be a very important limitation, specially for patients in risk of developing contrast material induced nephropathy. Despite these limitations, the spatial resolution of CT angiography is way superior to that of MRI, especially in the assessment of infratentorial arteries. (42) In the other hand, acquisition of imaging through MRI does not require exposure to contrast material and ionizing radiation, making it an alternative choice when other modalities represent an unacceptable risk. However, it must be considered that most of the studies have demonstrated their sensibility is not higher than 70%. (35-38)

Therapeutic approach

The medical treatment of patients with cerebellar infarction is very similar to that recommended for supratentorial infarction, it includes airway protection, proper oxygenation, arterial pressure management, glycemic control, euthermia, prevention of deep venous thrombosis and pulmonary thromboembolism. Also, the conventional treatment with antiaggregant and statints, is also recommended in these patients. (30, 35) The intensive control of patients with massive cerebellar infarction is similar to the one of patients with infarct in the middle cerebral artery, which has been deeply reviewed previously. (13)

Despite the start of an appropriate complementary medical treatment, the

DOI: 10.1515/romneu-2015-0060

available observational studies have demonstrated that the conservative treatment as the only measure is associated with a high mortality rate, close to 43% and can go as high as 85% in those patients that reach deep coma. (8) In the other hand, the result of a complementary treatment that includes appropriate required surgical procedures tends to have favorable results. The surgical treatment is the cornerstone of massive cerebellar infarction management. According to a review by Neugebauer et al. the survival rate of patients treated surgically was superior that 75%. The subgroup analysis found that for those who were treated only with ventriculostomy the survival rate was 81,6%, for those who underwent decompression of the posterior fossa, 76,8%, an 77,5% for those who underwent both procedures simultaneously. (8) Because of the mentioned previously, multidisciplinary assessment and management is recommended, ideally in a unit that specializes in the management of patients with infarction whose characteristics allow early surgical intervention if needed. When there is no infarction specialized unit available, an alternative choice is intensive care unit. The rigorous monitoring is essential, especially during the acute stage of the event, because about 50% of patients will deteriorate during the infarction, especially in the first five to seven days after the beginning of symptoms. (2, 6, 8, 43, 44) The neurological complications that may present during this stage are: recurrent ischemic events, large brainstem infarction, brainstem-encephalon compression by the infarcted hemisphere, obstructive hydrocephalus, and descending or ascending cerebellar herniation.

The surgical treatment of patients with cerebellar infarction has as main goal to control supratentorial obstructive hydrocephalus and infratentorial intracranial pressure. Although there are several therapeutic approaches, the superiority of each of them is not know for sure, because there are no clinical trials that allow to clear the uncertainty. Thanks to this, there is a remarkable variability between the therapeutic approaches used in each medical center. (45)

We propose an algorithm for the clinical management of massive posterior infarct that can be used by the treating medical team. (See algorithm below). In the actuality, there are no comparative studies that evaluate the effectiveness of surgical decompression and cerebrospinal fluid diversification procedures, alone or in combination, and the conservative treatment in each particular circumstance. Therefore, only through a detailed analysis, the information obtained from the clinical examination and the findings from neurologic imaging may determine the best therapeutic option for each patient. In the neurologic images it must be evaluated carefully the aspect of peritruncal cisterns, the appearance of fourth ventricle, the size and morphology of supratentorial ventricular system.

Acute obstructive hydrocephalus is one of most frequent complications of cerebellar infarctions, presenting in 10,9 to 27,2% of cases. (2, 44) Its evolution is generally progressive; reaching abrupt changes in the state of consciousness whose consequences may even be lethal if treatment is not started

promptly. The management of hydrocephalus must be aggressive; as soon as dilation of temporal horns of lateral ventricles becomes evident. In the actuality, control of hydrocephalus may be accomplished through several techniques; among these techniques there is ventriculoperitoneal shunting, external ventricular shunting, ventriculostomy, endoscopic ventriculocisternostomy of third ventricle. However, some authors have recommended surgical decompression of posterior fossa as treatment, based on the concept that the cause of hydrocephalus is the asymmetrical deformation of the fourth ventricle. (30, 34, 36) In the absence of comparative studies, the most accepted conduct in the majority of centers is to perform a cerebrospinal fluid shunt. Also, the latest guidelines from The American Stroke Association recommend the implantation of catheters for ventricular draining of patients with acute hydrocephalus secondary to ischemic infarction (Class I Recommendation). (30) Nevertheless, the clinical status posterior to the control of hydrocephalus must be monitored strictly, specially during the first hours after surgery, because in case that there is no clinical improvement additional exams should be performed in order to determine whether there is compression of infratentorial structures and the functional status. (8, 30, 47)

In the absence of hydrocephalus, the neurologic causes of persistent deterioration in the state of consciousness are:

1. Brainstem compression by the infarcted hemisphere.

2. Repetitive ischemic events, supra- or infratentorial.

3. Extensive infarctions of brainstem.

4. Descending (transforaminal) or ascending (transtentorial) herniation.

The importance of performing an accurate differential diagnosis is preponderant for subsequent decision making in the therapy. For this, there are basically 2 approaches:

1) Decompressive craniectomy of posterior fossa. The American Stroke Society recommends this procedure in combination with aggressive medical treatment to relieve brainstem compression and as therapeutic strategy in those cases that show clinical or imaging signs of cerebral herniation (Class I Recommendation; Evidence level B). (30) The ideal indication for the procedure is those cases that represent deterioration in the state of consciousness, associated with radiological signs of hypertension within the posterior fossa, such as compression of basal cisterns or displacement from the middle-clival midline, in those who do not have active hydrocephalus and no irreversible injury to the brainstem is present. (6, 8)

Identification of brainstem reflexes in the clinical examination.

2) Perform control MRI, which is very useful to identify if whether there are changes in the intensity in the brainstem that confirm the presence of ischemic injury in the anatomical region that contains the reticular activating substance. Also, it allows identification of new ischemic events, which are specially frequent in those patients whose embolism has a cardiogenic origin. It has been shown that in the cases that MRI confirms an extended infarction in the brainstem, the

DOI: 10.1515/romneu-2015-0060

survival and functionality prognosis is adverse, therefore, the literature recommends limiting therapeutic effort. (6, 8)

However, performing a MRI in the critical patient may be unpractical because it requires great acquisition time and wide electromagnetic field, which is incompatible with several life support and monitoring elements. To avoid this, an alternative that is becoming highly acceptable is the functional evaluation of brainstem through neurophysiologic exams, which can be performed even bedside in the intensive care unit. (48, 50) In patients with cerebellar and brainstem infarction, the brainstem auditory evoked response and somatosensory evoked response generally give accurate estimations about survival and functional prognosis. Brainstem auditory evoked response measure transmission through the cochlear nerve, superior olive, lateral lemniscus and inferior colliculus; these allow extension of ischemic compromise in the auditory pathways that are found in the pons. (51) The somatosensory evoked response is more reliable than brainstem auditory evoked response.

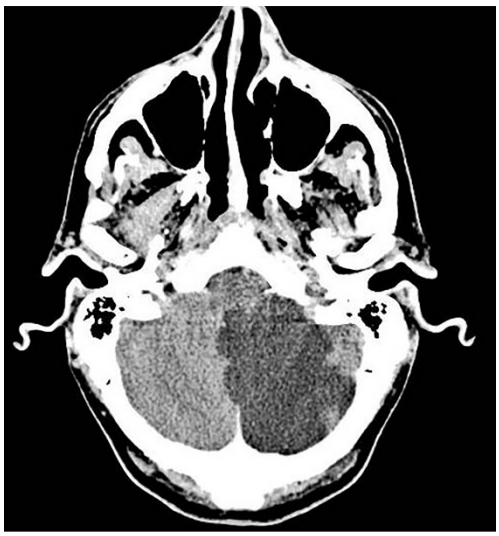


Figure 1 - Brain CT scan showing infarction in the posterior fossa

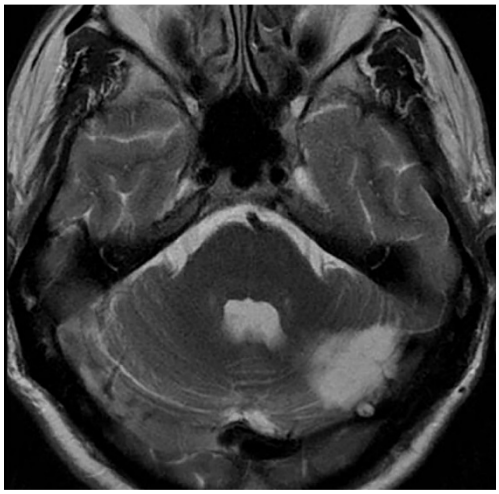
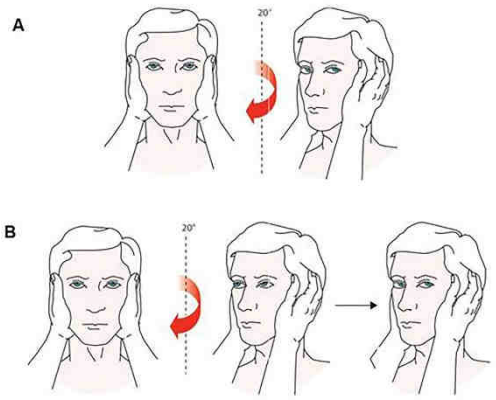


Figure 2 - MRI showing cerebellar infarction



Cephalic impulse test. A) Normal response. When rapid cephalic rotation is performed, conjugated look remains focused on the look's target. B) Abnormal response or "positive test".

Conclusions

Despite the large variability of massive cerebellar infarction definitions, this is characterized by cerebellar edema associated with obstructive hydrocephalus, compression of cisterns in the posterior fossa or brainstem; however, it does not consider the patient's clinical status. Massive cerebellar infarction is more frequent in younger individuals than those infarctions involving anterior circulation, in patients with less comorbidity, and generally they have an embolism as etiology, although obstructions in the principal arteries of posterior circulation are distinguished. The clinical presentation will depend on the extension of cerebellar injury and specially, the simultaneous injury to the brainstem, although the clinical picture is non-specific. CT and MRI are still the first choice imaging studies, considering bone findings in the posterior fossa when assessed by CT scan. The most useful mode to identify the ischemic territory is the diffusion sequence (DWI-ADC), due to the high sensibility even when performed during the first 24 hours after the symptoms have started. The management is similar to that in supratentorial ischemic events, emphasizing at all time patient stabilization, airway protection, proper oxygenation, arterial pressure control, glycemic control, euthermia, prevention of deep venous thrombosis and pulmonary thromboembolism. Despite the knowledge of all these diagnostic and therapeutic tools, previously discussed in the review, the mortality caused by ischemic events in the posterior fossa is still high, around 43%, this force us to follow up to establish an etiology, diagnostic and therapeutic processes, specially in the critical patient.

Correspondence
Dr. Luis Rafael Moscote
University of Cartagema Colombia
E-mail: mineurocirujano@aol.com

References

1. Shenkin HA, Zavala M. Cerebellar strokes: mortality, surgical indications, and results of ventricular drainage. Lancet [Internet]. 1982 Aug 21 [cited 2014 Jul 10];2(8295):429–32. Available from: http://www.ncbi.nlm.nih.gov/pubmed/6124817
2. Baldauf J, Oertel J, Gaab MR, Schroeder HWS. Endoscopic third ventriculostomy for occlusive hydrocephalus caused by cerebellar infarction. Neurosurgery [Internet]. 2006 Sep [cited 2014 Jul 10];59(3):539–44; discussion 539–44. Available from: http://www.ncbi.nlm.nih.gov/pubmed/16955035
3. Edlow JA, Newman-Toker DE, Savitz SI. Diagnosis and initial management of cerebellar infarction. Lancet Neurol [Internet]. 2008 Oct [cited 2014 Jul 10];7(10):951–

DOI: 10.1515/romneu-2015-0060

64. Available from: http://www.ncbi.nlm.nih.gov/pubmed/18848314
4. Jauss M, Krieger D, Hornig C, Schramm J, Busse O. Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. J Neurol [Internet]. 1999 Apr [cited 2014 Jul 10];246(4):257–64. Available from: http://www.ncbi.nlm.nih.gov/pubmed/10367693
5. Andoh T, Sakai N, Yamada H, Hattori T, Miwa Y, Hirata T, et al. [Cerebellar infarction: analysis of 33 cases]. No Shinkei Geka [Internet]. 1990 Sep [cited 2014 Jul 10];18(9):821–8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/2234303
6. Raco A, Caroli E, Isidori A, Salvati M. Management of acute cerebellar infarction: one institution's experience. Neurosurgery [Internet]. 2003 Nov [cited 2014 Jul 10];53(5):1061–5; discussion 1065–6. Available from: http://www.ncbi.nlm.nih.gov/pubmed/14580272
7. Tohgi H, Takahashi S, Chiba K, Hirata Y. Cerebellar infarction. Clinical and neuroimaging analysis in 293 patients. The Tohoku Cerebellar Infarction Study Group. Stroke [Internet]. 1993 Nov [cited 2014 Jul 10];24(11):1697–701. Available from: http://www.ncbi.nlm.nih.gov/pubmed/8236346
8. Neugebauer H, Witsch J, Zweckberger K, Jüttler E. Space-occupying cerebellar infarction: complications, treatment, and outcome. Neurosurg Focus [Internet]. 2013 May [cited 2014 Jul 10];34(5):E8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23634927
9. Koh MG, Phan TG, Atkinson JL, Wijdicks EF. Neuroimaging in deteriorating patients with cerebellar infarcts and mass effect. Stroke [Internet]. 2000 Sep [cited 2014 Jul 10];31(9):2062–7. Available from: http://www.ncbi.nlm.nih.gov/pubmed/10978030
10. Korn-Lubetzki I, Molshatzki N, Benderly M, Steiner I. The relatively good outcome of cerebellum-brainstem ischemic strokes. Eur Neurol [Internet]. 2013 Jan [cited 2014 Jul 10];69(1):8–13. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23146821
11. Putaala J, Haapaniemi E, Kaste M, Tatlisumak T. How does number of risk factors affect prognosis in young patients with ischemic stroke? Stroke [Internet]. 2012 Feb [cited 2014 Jul 10];43(2):356–61. Available from: http://www.ncbi.nlm.nih.gov/pubmed/22052508
12. Glass TA, Hennessey PM, Pazdera L, Chang H-M, Wityk RJ, Dewitt LD, et al. Outcome at 30 days in the New England Medical Center Posterior Circulation Registry. Arch Neurol [Internet]. 2002 Mar [cited 2014 Jul 10];59(3):369–76. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11890839
13. Godoy D, Piñero G, Cruz-Flores S, Alcalá Cerra G, Rabinstein A. Malignant hemispheric infarction of the middle cerebral artery. Diagnostic considerations and treatment options. Neurologia [Internet]. 2013 Apr 16 [cited 2014 Jul 10]; Available from: http://www.ncbi.nlm.nih.gov/pubmed/23601756
14. Bagci AM, Lee SH, Nagornaya N, Green BA, Alperin N. Automated posterior cranial fossa volumetry by MRI: applications to Chiari malformation type I. AJNR Am J Neuroradiol [Internet]. 2013 Sep [cited 2014 Jul 10];34(9):1758–63. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3773013&tool=pmcentrez&rendertype=abstract
15. Amarenco P. The spectrum of cerebellar infarctions. Neurology [Internet]. 1991 Jul [cited 2014 Jul 10];41(7):973–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/2067660
16. Adamson DC, Dimitrov DF, Bronec PR. Upward transtentorial herniation, hydrocephalus, and cerebellar edema in hypertensive encephalopathy. Neurologist [Internet]. 2005 May [cited 2014 Jul 10];11(3):171–5. Available from: http://www.ncbi.nlm.nih.gov/pubmed/15860139
17. Lankford J, Butler IJ. Transient tonic upgaze deviation: a neuro-ophthalmological complication of acute cerebellitis. J Child Neurol [Internet]. 2013 Jul [cited 2014 Jul 10];28(7):942–4. Available from: http://www.ncbi.nlm.nih.gov/pubmed/22914381
18. Gurol ME, St Louis EK. Treatment of cerebellar masses. Curr Treat Options Neurol [Internet]. 2008 Mar [cited 2014 Jul 10];10(2):138–50. Available from: http://www.ncbi.nlm.nih.gov/pubmed/18334136
19. Amarenco P, Hauw JJ. Cerebellar infarction in the territory of the superior cerebellar artery: a clinicopathologic study of 33 cases. Neurology [Internet]. 1990 Sep [cited 2014 Jul 10];40(9):1383–90. Available from: http://www.ncbi.nlm.nih.gov/pubmed/2392223
20. Amarenco P, Hauw JJ. [Edematous cerebellar infarction. A clinico-pathological study of 16 cases]. Neurochirurgie [Internet]. 1990 Jan [cited 2014 Jul 10];36(4):234–41. Available from: http://www.ncbi.nlm.nih.gov/pubmed/2277660
21. Keidel M, Galle G, Wiedmayer J, Taghavy A. [Malignant cerebellar infarct]. Fortschr Neurol Psychiatr

[Internet]. 1984 Aug [cited 2014 Jul 10];52(8):277–83. Available from: http://www.ncbi.nlm.nih.gov/pubmed/6479828
22. Masuda Y, Tei H, Shimizu S, Uchiyama S. Factors associated with the misdiagnosis of cerebellar infarction. J Stroke Cerebrovasc Dis [Internet]. 2013 Oct [cited 2014 Jul 10];22(7):1125–30. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23186911
23. Lee H. Neuro-otological aspects of cerebellar stroke syndrome. J Clin Neurol [Internet]. 2009 Jun [cited 2014 Jul 10];5(2):65–73. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2706413&tool=pmcentrez&rendertype=abstract
24. Savitz SI, Caplan LR, Edlow JA. Pitfalls in the diagnosis of cerebellar infarction. Acad Emerg Med [Internet]. 2007 Jan [cited 2014 Jul 10];14(1):63–8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/17200515
25. Casani AP, Dallan I, Cerchiai N, Lenzi R, Cosottini M, Sellari-Franceschini S. Cerebellar infarctions mimicking acute peripheral vertigo: how to avoid misdiagnosis? Otolaryngol Head Neck Surg [Internet]. 2013 Mar [cited 2014 Jul 10];148(3):475–81. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23307911
26. Nelson JA, Viirre E. The clinical differentiation of cerebellar infarction from common vertigo syndromes. West J Emerg Med [Internet]. 2009 Nov [cited 2014 Jul 10];10(4):273–7. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2791733&tool=pmcentrez&rendertype=abstract
27. Maranhão ET, Maranhão-Filho P. Vestibulo-ocular reflex and the head impulse test. Arq Neuropsiquiatr [Internet]. 2012 Dec [cited 2014 Jul 10];70(12):942–4. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23295423
28. Manto M. Cerebellar stroke. Cerebellar disorders A practical approach to diagnosis and management. Cambridge: Cambridge University Press; 2010. p. 88–101.
29. Simmons Z, Biller J, Adams HP, Dunn V, Jacoby CG. Cerebellar infarction: comparison of computed tomography and magnetic resonance imaging. Ann Neurol [Internet]. 1986 Mar [cited 2014 Jul 10];19(3):291–3. Available from: http://www.ncbi.nlm.nih.gov/pubmed/3963774
30. Jauch EC, Saver JL, Adams HP, Bruno A, Connors JJB, Demaerschalk BM, et al. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke [Internet]. 2013 Mar [cited 2014 Jul 10];44(3):870–947. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23370205
31. Sakamoto Y, Kimura K, Iguchi Y, Shibazaki K, Aoki J. Hemorrhagic transformation in acute cerebellar infarction. Cerebrovasc Dis [Internet]. 2011 Jan [cited 2014 Jul 10];32(4):327–33. Available from: http://www.ncbi.nlm.nih.gov/pubmed/21921595
32. Taneda M, Hayakawa T, Mogami H. Primary cerebellar hemorrhage. Quadrigeminal cistern obliteration on CT scans as a predictor of outcome. J Neurosurg [Internet]. 1987 Oct [cited 2014 Jul 10];67(4):545–52. Available from: http://www.ncbi.nlm.nih.gov/pubmed/3655893
33. Reddy AK, Saradhi V, Panigrahi M, Rao TN, Tripathi P, Meena AK. Decompressive craniectomy for stroke : indications and results. Neurol India [Internet]. 2002 Dec [cited 2014 Jul 10];50 Suppl:S66–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/12665881
34. Michel P, Arnold M, Hungerbühler H-J, Müller F, Staedler C, Baumgartner RW, et al. Decompressive craniectomy for space occupying hemispheric and cerebellar ischemic strokes: Swiss recommendations. Int J Stroke [Internet]. 2009 Jun [cited 2014 Jul 10];4(3):218–23. Available from: http://www.ncbi.nlm.nih.gov/pubmed/19659825
35. Brott TG, Halperin JL, Abbara S, Bacharach JM, Barr JD, Bush RL, et al. 2011 ASA/ACCF/AHA/AANN/AANS/ACR/ASNR/CNS/SAIP/SCAI/SIR/SNIS/SVM/SVS guideline on the management of patients with extracranial carotid and vertebral artery disease: executive summary. Stroke [Internet]. 2011 Aug [cited 2014 Jul 10];42(8):e420–63. Available from: http://www.ncbi.nlm.nih.gov/pubmed/21282494
36. Nguyen-Huynh MN, Wintermark M, English J, Lam J, Vittinghoff E, Smith WS, et al. How accurate is CT angiography in evaluating intracranial atherosclerotic disease? Stroke [Internet]. 2008 Apr [cited 2014 Jul 10];39(4):1184–8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/18292376
37. Bash S, Villablanca JP, Jahan R, Duckwiler G, Tillis M, Kidwell C, et al. Intracranial vascular stenosis and occlusive disease: evaluation with CT angiography, MR angiography, and digital subtraction angiography. AJNR

DOI: 10.1515/romneu-2015-0060

Am J Neuroradiol [Internet]. 2005 May [cited 2014 Jul 10];26(5):1012–21. Available from: http://www.ncbi.nlm.nih.gov/pubmed/15891154

38. Skutta B, Fürst G, Eilers J, Ferbert A, Kuhn FP. Intracranial stenoocclusive disease: double-detector helical CT angiography versus digital subtraction angiography. AJNR Am J Neuroradiol [Internet]. 1999 May [cited 2014 Jul 10];20(5):791–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/10369348

39. Leffers AM, Wagner A. Neurologic complications of cerebral angiography. A retrospective study of complication rate and patient risk factors. Acta Radiol [Internet]. 2000 May [cited 2014 Jul 10];41(3):204–10. Available from: http://www.ncbi.nlm.nih.gov/pubmed/10866072

40. Willinsky RA, Taylor SM, TerBrugge K, Farb RI, Tomlinson G, Montanera W. Neurologic complications of cerebral angiography: prospective analysis of 2,899 procedures and review of the literature. Radiology [Internet]. 2003 May [cited 2014 Jul 10];227(2):522–8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/12637677

41. Hirai T, Korogi Y, Ono K, Nagano M, Maruoka K, Uemura S, et al. Prospective evaluation of suspected stenoocclusive disease of the intracranial artery: combined MR angiography and CT angiography compared with digital subtraction angiography. AJNR Am J Neuroradiol [Internet]. 2002 Jan [cited 2014 Jul 10];23(1):93–101. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11827880

42. Graf J, Skutta B, Kuhn FP, Ferbert A. Computed tomographic angiography findings in 103 patients following vascular events in the posterior circulation: potential and clinical relevance. J Neurol [Internet]. 2000 Oct [cited 2014 Jul 10];247(10):760–6. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11127530

43. Cano LM, Cardona P, Quesada H, Mora P, Rubio F. [Cerebellar infarction: prognosis and complications of vascular territories]. Neurologia [Internet]. [cited 2014 Jul 10];27(6):330–5. Available from: http://www.ncbi.nlm.nih.gov/pubmed/22341984

44. Khan M, Polyzoidis KS, Adegbite AB, McQueen JD. Massive cerebellar infarction: "conservative" management. Stroke [Internet]. [cited 2014 Jul 10];14(5):745–51. Available from: http://www.ncbi.nlm.nih.gov/pubmed/6658959

45. Mostofi K. Neurosurgical management of massive cerebellar infarct outcome in 53 patients. Surg Neurol Int [Internet]. 2013 Jan [cited 2014 Jul 10];4:28. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3604818&tool=pmcentrez&rendertype=abstract

46. Adams HP, Adams RJ, Brott T, del Zoppo GJ, Furlan A, Goldstein LB, et al. Guidelines for the early management of patients with ischemic stroke: A scientific statement from the Stroke Council of the American Stroke Association. Stroke [Internet]. 2003 Apr [cited 2014 Jul 10];34(4):1056–83. Available from: http://www.ncbi.nlm.nih.gov/pubmed/12677087

47. Kudo H, Kawaguchi T, Minami H, Kuwamura K, Miyata M, Kohmura E. Controversy of surgical treatment for severe cerebellar infarction. J Stroke Cerebrovasc Dis [Internet]. 2007 [cited 2014 Jul 10];16(6):259–62. Available from: http://www.ncbi.nlm.nih.gov/pubmed/18035243

48. Itoh A, Kim YS, Yoshioka K, Kanaya M, Enomoto H, Hiraiwa F, et al. Clinical study of vestibular-evoked myogenic potentials and auditory brainstem responses in patients with brainstem lesions. Acta Otolaryngol Suppl [Internet]. 2001 Jan [cited 2014 Jul 10];545:116–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11677723

49. Deftereos SN, Panagopoulos G, Eleftheriadou A, Korres S, Georgonikou D, Kandiloros D, et al. Using vestibular evoked myogenic potentials to localise brainstem lesions. A preliminary report. B-ENT [Internet]. 2008 Jan [cited 2014 Jul 10];4(4):215–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/19227026

50. Burghaus L, Liu W-C, Dohmen C, Bosche B, Haupt WF. Evoked potentials in acute ischemic stroke within the first 24 h: possible predictor of a malignant course. Neurocrit Care [Internet]. 2008 Jan [cited 2014 Jul 10];9(1):13–6. Available from: http://www.ncbi.nlm.nih.gov/pubmed/17982737

51. Sand T, Kvaløy MB, Wader T, Hovdal H. Evoked potential tests in clinical diagnosis. Tidsskr Nor Laegeforen [Internet]. 2013 May 7 [cited 2014 Jul 10];133(9):960–5. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23652144