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Review Article

Posterior cerebral artery stroke with visual manifestation during COVID pandemic

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ABSTRACT

The posterior cerebral artery (PCA) stroke represents 5% to 10% of total strokes. The number of stroke cases has increased during the Global pandemic of Covid 19. It can be challenging to diagnose PCA stroke presenting as a visual manifestation, as symptoms are nonspecific and variable over due course of time. This is more complex in patients who are not aware of their symptoms, and therefore difficult to establish a timeline of events. Mostly PCA stroke with only visual symptoms visits an ophthalmologist rather than an emergency attendee. Knowledge of the neurovascular anatomy of the brain is required to understand PCA stroke and identify the stroke's mechanism to select appropriate therapies. In this article, we provide an overview of PCA stroke and focused on visual manifestations, diagnosis, and management of PCA stroke.

1. Introduction

A cerebrovascular accident is an injury to the brain due to vascular cause.[1] Its refer as a transient ischemic attack (TIA) if symptoms last < 24 hours without findings on brain imaging.[2] It's called a stroke if symptoms last > 24 hours with findings on brain imaging.[1,2] Neuroimaging help to differentiate the ischemic or hemorrhagic cause of strokes.[1] Ischemic strokes were further divided depending upon the anatomical distribution of vessels such as anterior cerebral artery (ACA), middle cerebral artery (MCA), and posterior cerebral artery (PCA) stroke.[1,3] An embolus or thrombus may block blood vessels within the brain leading to ischemic stroke and symptoms depend upon the area supplied by that vessels.[1] TIA also includes Amaurosis fugax; momentary visual loss due to transient retinal ischemia without retinal infarct confirmed by fundus examination and/or retinal imaging.[2]

2. COVID-19 and Cerebrovascular Strokes

The global pandemic of COVID-19 is reported to be linked with neurological disorders including cerebrovascular disease(CVD).[3] CVD affects about 3% of hospitalized COVID patients & up to 6% of those in ICU due to COVID.[4,5] The most common cause of cerebrovascular manifestation is ischemic stroke.[1,3] The risk of ischemic stroke increases 3.6 times if infected with COVID-19.[3] The severity of COVID-19 does not affect the frequency of ischemic stroke.[4] All age groups are affected by COVID stroke and Males are more affected.[4,5] It is postulated due to the high levels of inflammation (vascular endotheliopathy) and Coagulopathy.[3–5] Virus gets attached to the angiotensin-converting enzyme ACE-2 receptor on endothelium and arterial smooth muscle, resulting in inflammation.[3] All body cell expresses ACE2-receptors that is directly invaded by the corona virus.[4] ACE2 is said to be the door for corona virus to enter the body.[6] This binding of the virus leads to a decrease in the expression and activity of ACE2.[5] The protective

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effect of ACE2 decreases with a decrease in expression and simultaneously the injurious actions of angiotensin II increase.[3,5] This further leads to the stimulation of immune cells by producing cytokines, comprising IL-6 and IL-1ß.[5] The complement-mediated endothelial injury may also play an important role in this pathogenesis.[4] Hypercoagulability, which plays a very crucial role in the pathogenesis of stroke, is not defined clearly in most of the studies.[4] The spectrum of hypercoagulability includes elevated fibrinogen, prolonged prothrombin time, prolonged PTT, factor VIII, von Willebrand factor (vWF), or elevated D-dimer.[3,4] Other than hypercoagulability, viral infection or bacterial superinfection can also elevate D-dimer.[4]

The hypercoagulable state can also manifest as pulmonary embolism (PE), deep venous thrombosis (DVT), and paradoxical embolism stroke.[3] Virchow's triad also plays a definite role in COVID-19 patients.[3,4] The endothelial injury is either by direct invasion of the virus and/or increased cytokines release.[3] The hypercoagulable state depends upon disseminated intravascular coagulation (DIC), cytokine storm, complement activation, macrophage activation syndrome & hyperferritinemia, and renin-angiotensin system overactivation.[7] Stasis is due to the immobilization of hospitalized patients, especially in the ICU.[3]

3. Vascular Anatomy of the Brain

The knowledge of the blood supply of the brain is important to understand the pathophysiology of stroke. The primary blood circulation of the brain is from the internal carotid artery (ICA) supplying the anterior & middle portions of the brain and the basilar artery (BA) supplying the posterior portion of the brain.[8,9] ICA supplies the anterior and middle portions of the brain through the right & left anterior cerebral artery (ACA) and right & left middle cerebral artery (MCA) respectively.[8] BA is formed by the fusion of the right & left vertebral arteries (VA) within the cranium and then divides into the right & left posterior cerebral artery (PCA) to supply the posterior portion.[9] The posterior communicating artery (PCOM) which is a branch of PCA, connects ACA & MCA to PCA to form the circle of Willis.[8] The circle of Willis is completed with seven arteries, Right & left PCA, Right & left PCOM, Right & left ICA, Right & left ACA, and Anterior communicating artery (ACOM) (Figure 1). The circle of Willis and collateral circulation act as an alternate pathway to prevent ischemia in case of occlusion in the cerebral vasculature.[8,9] Posterior circulation is contributed by various arteries including VA, BA, PCA, etc.[8]

Posterior circulation strokes with only visual manifestation usually have nonspecific presentation as symptoms may change with the passage of time and many times patient is not aware of its severity unless vision is severely affected.[8] This nonspecific presentation can be

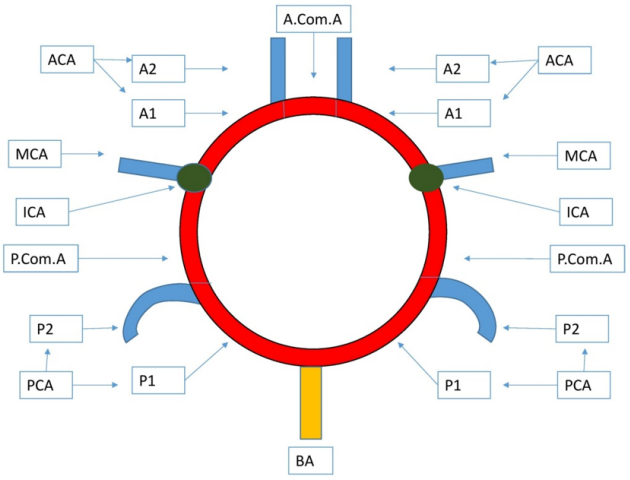

Fig. 1: Schematic diagram of segments of the circle of Willis ACA (Anterior cerebral artery A1 & A2), A. Com. A (Anterior communicating artery), MCA (Middle cerebral artery), ICA (Internal carotid artery), P. Com. A (Posterior communicating artery), PCA (Posterior cerebral artery P1 & P2), BA (Basilar artery)

challenging to diagnose and manage.[8] The PCA stroke comprises 5% to 10% of all strokes.[1] The PCA originates from the basilar artery and terminates by supplying the occipital lobe.[8] Its full course can be divided into 5 segments (P1, P2, P3, P4 & P5).[9] (Figure 2) The P1 comprises from basilar artery to the starting of the PCOM artery.[8] The P1 passes over the oculomotor nerve.[9] Lesion to this segment causes bilateral paramedian thalamic lesions and Its symptoms may include memory impairment, altered sensorium and verticle gaze palsy.[8,9] The P2 begins from PCOM, the proximal part is P2A within the crural cistern and the distal part is P2P within ambient cistern.[9] Lesion to this part affects the supply of the occipital lobe resulting in a visual deficit.[1] The P3 segment runs through the quadrigeminal cistern and gives off anterior and posterior inferior temporal arteries.[8] The P4 segment runs through the calcarine sulcus and supplies areas bordering the calcarine sulcus and the medial surface of the occipital lobe.[9] The P5 segment is the terminating branch of P4, that is parieto-occipital & calcarine arteries.[9] Some literature combines P5 into P4 segment only.[8,9]

The visual cortex is the primary cortical region of the brain that receives, integrates, and processes visual information relayed from the retinas.[10] The branches of the PCA supply the visual cortex.[8] Based on the function and structure, the visual cortex can be divided into five different areas (V1 to V5).[10] V1 is also known as the primary visual cortex and V2 – V5 is known as the secondary visual cortex. The information first reaches to V1 centers around the calcarine sulcus. The left eye hemifield information is processed by the right cortical areas and the right eye

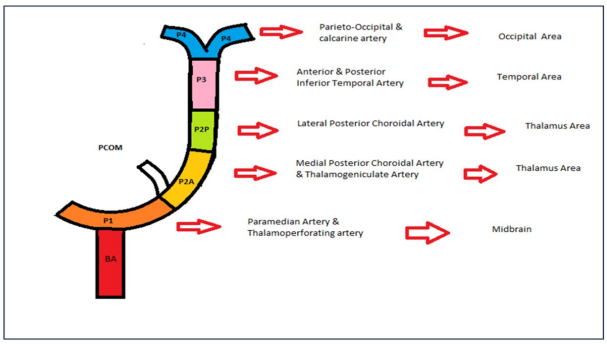

Fig. 2: Schematic diagram of segments of posterior cerebral artery, its branches and brain area supplied by them BA (Basilar artery), P (Posterior cerebral artery P1, P2A, P2P, P3, P4), PCOM (Posterior communicating artery)

hemifield information is processed by the left cortical area.[10] Based on the structure (simple & complex cell) and function V1 is further divided into six distinct layers. The highest concentration of simple cells is present in Layer 4 of V1 which processes information from the lateral geniculate body. Complex cells can be found in layers 2, 3, and 6 of V1. Visual components of orientation and direction are responded by V1. Integrated information from V1 is passed to V2. The V2 processes the information and sends feedback to V1 and/or feeds forward to a higher level of the secondary visual cortex. As the information passes from V1 to V5, there is an increase in the level of complexity and response patterns to objects.[10]

4. Etiology

The most common etiology for PCA strokes are atherosclerosis, embolism, and small artery disease.[8] TIA can be classified as cerebral TIA due to cardio-embolism, hypercoagulable state & arterial dissection and AF (retinal TIA) including retinal arterial/arteriolar vasospasm & retinal hypoperfusion.[2] When the etiology is unidentifiable after comprehensive and systemic clinical evaluation then its termed as cryptogenic.[2] About 40% of acute strokes among COVID-19 patients are diagnosed as cryptogenic strokes.[5] Most cryptogenic Stroke/TIA seems to be of embolic origin and named as an embolic stroke of an undetermined source (ESUS).[2]

COVID-19 leads to a large number of systemic complications and is frequently found be to associated with coagulopathy and endotheliopathy.[3,5,11] Direct viral invasion causes endothelium inflammation leading to an immune response from the body in the form of cytokine storm causing hypercoagulability predisposes to thrombus formation and stroke.[3,4] There is a 7.6 times increase in the complication of cerebrovascular disease with COVID-19 virus than with the influenza virus.[5]

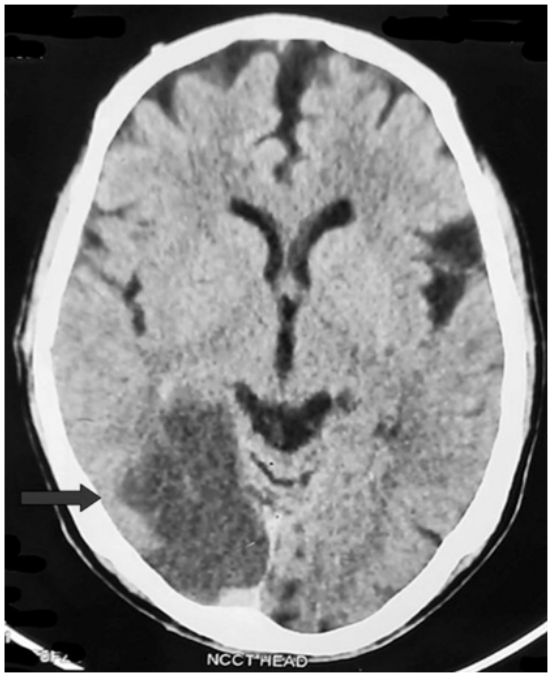

Fig. 3: Computerized tomography of brain and skull shows infarct area in right occipital lobe

5. Risk Factor

Common risk factors for stroke in the general population include high blood pressure, heart disease, diabetes, dyslipidemia, smoking, heavy drinking, high salt, high-fat diet, and lack of exercise.[8] Pro-coagulant state heightened the risk of stroke among Covid patients.[3,5] Patent Foramen Ovale (PFO) which is present in up to 35% of the general population, has been reported to complicate embolus-related complications.[11] Embolus/clot can easily travel to the brain from the right atrium through PFO causing an ischemic stroke. Pro-coagulant state of Covid-19 and PFO is a major challenge for stroke physicians worldwide as Covid cases with acute infarction, associated with PFO have been reported and it is difficult to certain whether it is causative when found in a patient with a stroke, regardless of Covid status.[11]

The risk factors can be of two types, non-modifiable and modifiable.[8] Non-modifiable risk factors that we cannot change are age, gender, race, history of or family history of TIA, CVA, and hypercoagulable states.[8] Modifiable risk factors that we can change are hypertension, diabetes mellitus, hyperlipidemia, smoking, heart disease (atrial fibrillation, endocarditis) oral contraceptives, substance abuse, poor diet, obesity, immobility and sleep apnea.[8] ABCD2 score can be used to identify a high risk of subsequent stokes as a person with the history of a stroke or TIA is at risk of having another episode.[2] Age, BP, Clinical presentation, Duration of symptoms, and DM are

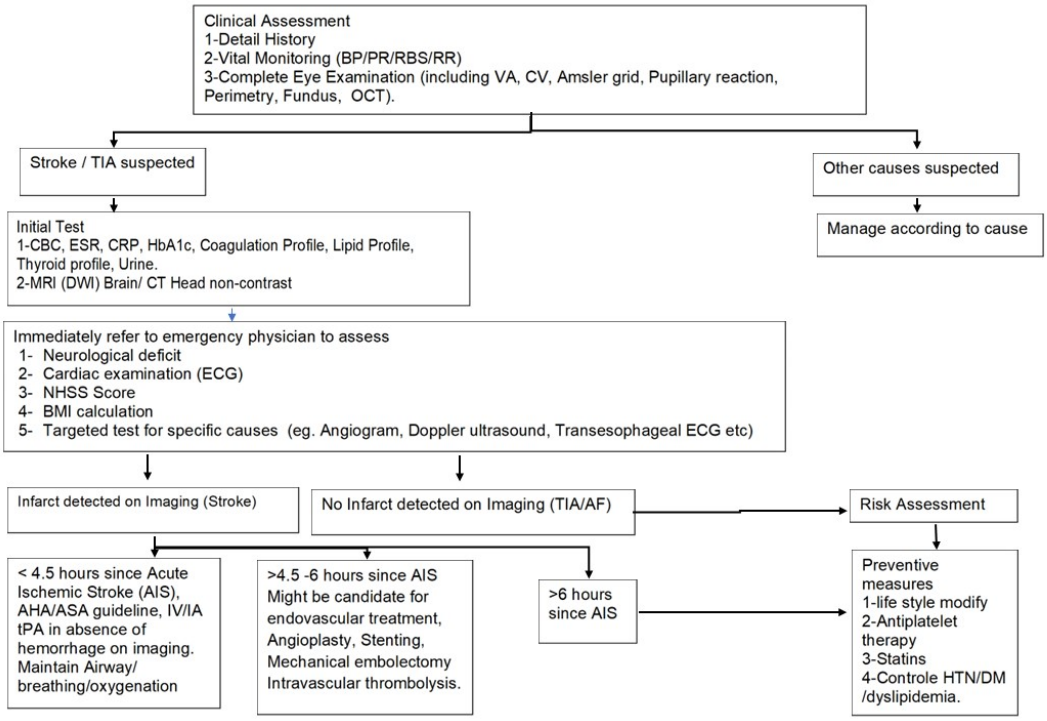

Fig. 4: Flow chart for the evaluation of posterior cerebral artery stroke / transient ischemic attack

used to calculate the score and graded as mild risk (0-3), Moderate risk (4-5) and high risk (6-7).[2] ABCD3 which also includes recent history of TIA and ABCD3-i includes imaging finding in scores that have been found superior to ABCD2.[2] (Table 1)

6. Clinical Evaluation

Evaluation begins with a detailed history and examination.[2] Diagnosis heavily relies on history as symptoms may change at the time of assessment.[1,2] The time of onset of symptom is a very important clue and help to decide the mode of treatment in an emergency.[8] In some cases of posterior circulation stroke patients may be unaware of their symptoms, so ask when the patient starts behaving in a manner that appears to be abnormal for them.[8] A brief history with a focus on risk factors should be obtained from the patient's past medical history. PCA stroke patients can present with headaches with mild visual changes that may include vision loss, diplopia, visual field defect, color vision defect, or difficulty in recognizing familiar faces.[8]

A full physical examination should be performed as early as possible with the help of an emergency physician.[8] A complete neurological examination is required to identify any residual deficits and the National Institutes of Health Stroke Scale (NHSS) can be helpful.[2] Comprehensive ocular examination including visual acuity, color vision, contrast sensitivity, Amsler grid, visual field testing, pupillary examination and dilated fundus examination should be done in the ophthalmology department as general emergency room evaluation from an ophthalmological point of view may be suboptimal.[2]

The presentation of PCA stroke patients depends upon the vessel involved, the area affected, and the severity of occlusion.[2,8] When P1& P2 are involved than thalamus, midbrain and cortex will be affected.[8] P1 involvement may present as hypersomnolence, cognitive deficits and vertical gaze paresis whereas P2 involvement may present as severe contralateral hypesthesia and ataxia.[12] But when P3& P4 involve than only cortical structures are affected.[8,12] The presentation of P3 & P4 involvement is nonspecific and may include only headache and visual impairment.[8]

Cortical blindness (CB) is a rare neurological condition.[13] Visual impairment is a common sequela after stroke affecting the primary visual cortex.[14] It presents as contralesional homonymous hemianopia in unilateral lesions or less commonly cortical blindness in bilateral lesions.[8,14] CB is defined as vision loss without any ocular cause and with normal pupillary reaction.[15] Bilateral lesions of the occipital cortex are responsible for CB.[15] While it has been widely assumed that after a brief period of spontaneous improvement, vision loss becomes stable and permanent but accumulating data suggest that visual rehabilitation training may produce some modest recovery of vision in selected cases, even long after the stroke.[14] Cortical blindness comprises loss of all visual

Table 1: ABCD scores to access the risk of future stroke in a stroke patient

Variables	Parameters	ABCD2	ABCD3	ABCD3-i
Age	<60yrs	0	0	0
	≥60yrs	1	1	1
Blood pressure	<140/90 mm Hg	0	0	0
	≥140/90 mm Hg	1	1	1
Clinical features	Others	0	0	0
	Speech impairment without weakness	1	1	1
	Unilateral weakness	2	2	2
Duration	≥60 min	2	2	2
	10–59 min	1	1	1
	<10 minutes	0	0	0
Diabetes mellitus	Absent	0	0	0
	Present	1	1	1
Dual TIA	TIA prompting medical attention plus at least another TIA in the preceding 7 days	NA	2	2
Imaging	Ipsilateral ≥50% stenosis of internal carotid artery	NA	NA	2
	Acute diffusion-weighted imaging hyperintensity	NA	NA	2
Total range of score		0-7	0-9	0-13

TIA indicates transient ischemic attack; and NA, not applicable

sensations (including the perception of light and dark), loss of menace reflex, normal papillary reaction, normal fundus examination, and normal ocular movements.[13] Other features of visual cortex lesion include Anton-Babinski syndrome (Anton syndrome or ABS), Riddoch phenomenon and formed visual hallucination.[15]

ABS is a rare complication of cortical blindness which present as visual anosognosia and confabulation.[13] Visual anosognosia is defined as denial of loss of vision by the patient in the presence of obvious visual loss, and confabulation is defined as the patient explaining events, surrounding, and experiences which is not present with obvious visual loss.[13,16] Patients may appear to have normal vision due to their detailed confabulation and description of surroundings, often leading to delayed diagnosis of vision loss.[13] The defect becomes apparent when patients are found describing people or surroundings that are not present and try to walk into objects in their way.[15] Patients with ABS often give excuses of dim light for their inability to see and insist to deny their vision loss even when presented with evidence otherwise, thereby putting themselves in danger.[13] The Riddoch phenomenon is statokinetic dissociation, which means the patient can only identify mobile objects in the blind field.[9,15] One of the explanations for this is motion processing fibers reaching V5 (middle temporal area) from the lateral geniculate nucleus and bypassing the V1 cortex.[13]

Although vision is severely impaired but central vision may be spared in some cases as the area in the occipital lobe representing the fovea is supplied by MCA, which is spared in PCA stroke.[9] Visual field defect can be small scotoma, quadrantanopia, or hemianopia depending upon the severity of brain damage.[13] One side infarction of the occipital lobe may cause opposite side hemianopia with macular sparing (rare to have both MCA & PCA stroke simultaneously).[15] Both side occipital lobes infarction may cause CB and achromatopia (color blindness).[8] Infarction in the temporal lobe may present as superior quadrantanopia.[9] Infarction of parietal lobe may present as Inferior quadrantanopoia.[9]

Among 30% of all stroke patients suffer from HH.[17] In the majority of cases, it is congruent.[11,17] Among all HH, about 40% of cases are due to occipital lobe lesions.[17,18] HH is contralateral hemianopsia (unilateral involvement at the optic tract, lateral geniculate nucleus, optic radiations, or occipital cortex opposite to the side of field loss) or bilateral HH (seen in bilateral occipital lobe lesions causing cortical blindness).[17,18] Most HH are Incomplete and among them, homonymous quadrantanopia is more common.[18] Hemianopic patients may notice flickering movement in the area of their blind field, known as blindsight.[15] This can be explained as V5 handles the recognition of motion independent of V1.[17] Some subcortical pathways directly reach V4 & V5 without the involvement of V1[17] Reverse to this is sightblindness, in which the hemianopic patient may notice a deficit in the seeing visual half of their visual field.[18]

Achromatopsia refers to difficulty perceiving colors.[8] It can be congenital or acquired.[19] Acquired Achromatopsia is mostly due to damage in the visual area V4 of the visual cortex.[8,19] Center for perceiving color information lies in the V4 area in the ventral occipitotemporal cortex.[19] If the infarction is unilateral then the patient may present

as hemiachromatopsia.[8] Some patient color vision may preserve as some fibers directly reach V4 & V5 and bypass V1.[17]

7. Investigations

Ocular investigation requires comprehensive eye examination, which comprises visual acuity, color vision, contrast sensitivity, Amsler chart, visual field testing, pupillary examination and dilated fundoscopic examination in the ophthalmology department.[2] Visual acuity may be markedly decreased in case of bilateral lesion with normal pupillary reaction and in case of unilateral lesion, perimetry may show visual field defect in the presence of normal pupillary reaction and normal fundoscopy.[8] Ocular motility should also be evaluated as the deep /proximal segment of PCA may cause vertical motility defects.[8]

Systemic investigation requires a level of consciousness and vital monitoring with blood workup that includes CBC, ESR, hemoglobin A1C, hypercoagulable status (PT, PTT), lipid profile & urine drug screening (eg Amphetamine, cocaine), etc.[2,8] Targeted tests can be considered when specific cases of suspicion exist. Stroke with COVID patients need to check C-reactive protein (C-RP), D-dimer, ferritin, IL-6, etc. Brain MRI with diffusion-weighted imaging/apparent diffusion coefficient sequences is the most effective investigation to identify acute stroke within the first 24 hours.[2,9] MRI imaging showing enhancement of arterial vessel wall may suggest inflammation due to endotheliopathy.[5] If MRI is unavailable or contraindicated then a non-contrast-enhanced computed tomogram (NCCT) scan can be considered.[2] (Figure 3) NCCT is preferred over CECT or MRI as its quicker and especially when the issue is to distinguishing hemorrhage from ischemia, a distinction that does not require contrast.[9] Contrast provides enhance quality of image on CT, but it's mostly used to rule out Vascular anomalies or tumors.[2,9]We can classify stroke based on duration as acute (less than 24 hours), subacute (24 hours to five days), or chronic (weeks).[9] We also required a baseline cardiovascular evaluation for all patients with TIA or Stroke. Electrocardiogram to identify atrial fibrillation & myocardial infarction and Transesophageal echocardiogram (TEE) to identify the cardiac source of emboli as they are one possible cause of stroke and particularly common in PCA stroke.[1,2]

8. Treatment

Treatment of PCA stroke or TIA is based on the etiology.[13] 2018 AHA/ASA guidelines explain pre-hospital care and hospital care clearly.[20] It guides us for quick evaluation and treatment for acute ischemic stroke (AIS).[20] It explains us to maintain the airway for breathing and to check for adequate oxygenation. The guideline tells us about the use of Intravenous therapy in the form of tissue plasminogen activator (tPA) if the patient reached within 4.5 hours after AIS.[8,9] The guidelines for administering IV tPA include a blood pressure < 180/110, finger-stick glucose > 50 mg/dl, and no hemorrhage on initial NCCT.[8,20] The dose of tPA is 0.9 mg/kg, the maximum dose is 90 mg over 60 min with an initial 10% of the dose given as a bolus over 1 min.[8] The use of Intravenous tPA among COVID patients and in the case of AIS is reported to be safe and effective.[3] A patient presenting between 4.5 hour to 6 hour of AIS, might still be a candidate for endovascular treatment (EVT).[8]

For a patient presenting after 6 hour of AIS, the aim should be preventing further stroke.[8] This can be categorized into general treatment that is applicable to all TIA/stroke patients and specific treatment based on identifiable etiology. General measures include lifestyle modification with regular consumption of fruit, fish, and vegetables, moderate-intensity aerobic activity, encouraging tobacco cessation, alcohol use moderation, weight management, and medical compliance.[2] Antiplatelet, anticoagulation, or statins should be used depending upon etiology.[2,9] Other secondary risk factors for example hypertension, cholesterol, and diabetes should be dealt with accordingly.[2,8] (Figure 4)

9. Rehabilitation

Apart from standard therapy for stroke, rehabilitation helps you regain as much independence and quality of life as possible. This can be achieved by vision training. In case of visual impairment due to stroke, this goal can be reached by restitution therapy, compensatory therapy and substitution therapy. Restitution therapy helps us to stimulate the retina in a nonseeing area of the visual field through a perimeter-like device that has multiple light spots. The compensation therapy trains the patient for saccadic eye movement that would help the retina to receive stimulus that would fall in the blind area of the visual field. Substitution therapy takes the help of devices like a prism to deviate the visual stimulus path and make it fall on the normal retina.[15,21]

10. Prognosis

The prognosis of PCA stroke depends on the age of the patient, etiology, severity, duration, initial recovery time, and medical history.[13] Pure PCA stroke has a lower risk of disability and long-term mortality.[22] Association of stroke with COVID-19 have more severe and unfavorable neurological outcomes than stroke without COVID-19.[3] Younger patients (<50 yr) have a chance for visual recovery but complete homonymous hemianopia is a poor prognosis predictor at any age.[23] The patient suffering a minor stroke has a good chance for recovery compared to the patient who has gross neurological deficits at presentation and usually has poor or no guarantee of full recovery.[24]

11. Complications

Visual impairment leads to poor quality of life (QOL), depending upon the extent of the Visual field defect.[25] Patients suffering from PCA stroke with visual impairment may have problems with orientation and moving around with daily activities like driving, reading, and are prone to trauma.[8,25] It makes the patient handicapped and may lead to social isolation.[25] PCA stroke patients may have aggressive behavior especially when they are stimulated by the environment.[8]

12. Conclusion

A multidisciplinary approach is required for the treatment of PCA stroke with visual impairment. The team includes emergency physicians, ophthalmologists, neurologists, and other specialists depending on the deficit. There is a potential for recovery and rehabilitation. This article provides us with an overview of PCA stroke, especially focusing on visual manifestations, diagnosis, and management. This article helps us to refresh guidelines and awareness about visual problems in acute situations. The goal is to achieve minimum deficit with good quality of life, if possible. The prognosis of stroke with COVID-19 appears to be worse than stroke without COVID-19.

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None.

14. Conflict of Interest

None.

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