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RESEARCH ARTICLE

A RARE CASE OF MIDDLECEREBRAL ARTERYSTROKERESULTING FROM ADISTANT PENETRATINGCHEST TRAUMA

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Abstract

This case report analyses the occurrance of an exceedingly rare and unpredictable complication of remote penetrating chest trauma with arterial injury , which is embolic stroke , leading to a particular combination of hemorrhagic shock hemodynamic profile altering the cerebral autoregulation , with malignant middle cerebral artery stroke in a young patient , causing intracranial hypertension resulting in subfalcine herniation , and compromising the penumbral zone and the chances of survival , and rendering most of the conventional stroke teatments contra indicated or not useful , our purpose is to raise into awareness this type of complication in order to establish efficient salvatory treatments without delay.

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Introduction:-

Ischemic stroke is an extremely rare consequence of remote chest trauma . This case report analyses a particular situation that originated from vascular trauma , causing a combination of hemorrhagic shock and ischemic stroke in a young patient , leading to a pathophysiological mechanism accelerating the evolution towards a vicious cycle , slimming down chances of survival by deducting therapeutic means .

Case report:

An 18 year old 178cm , 70kg caucasian male patient with no medical history was admitted to the emergency department for an hemopneumothorax at the thirtiest minute following a low energy penetrating cold steel chest trauma located in the anterior left fourth intercostal space , medially to the mid clavicular line . The blade was implanted slightly inwards and removed right afterwards , it measured 14 centimeters of length , 4cm of width .

Following admission , christalloid volume expanders were used to treat the resulting hemorrhagic shock .

At this juncture , the patient was conscious , agitated , displayed signs of a class 1 hemorragic shock (ATLS classification [1]) arterial blood pressure was 160/80mmHg , heart rate at 73 beats per minute , respiratory rate at 30 cycles per minute , blood saturation at 99 percent under 6 liters/minute delivered by simple face mask .

No clinical neurologic deficit or abnormality were detectable at this stage .

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Vascular analysis of the contrast thoracic Computed tomography (CT) scan objectified an aortic filling defect next to the ostium of the brachiocephalic arterial trunk(Fig 1 and 2), extravasation of the iodinated contrast material into the anterior mediastinum, in addition to a low abundance left hemomediastinum and pneumomediastinum (Fig 3) and a small pericardial effusion , no images of aneurysm , dissection , nor vascular hematoma were detected

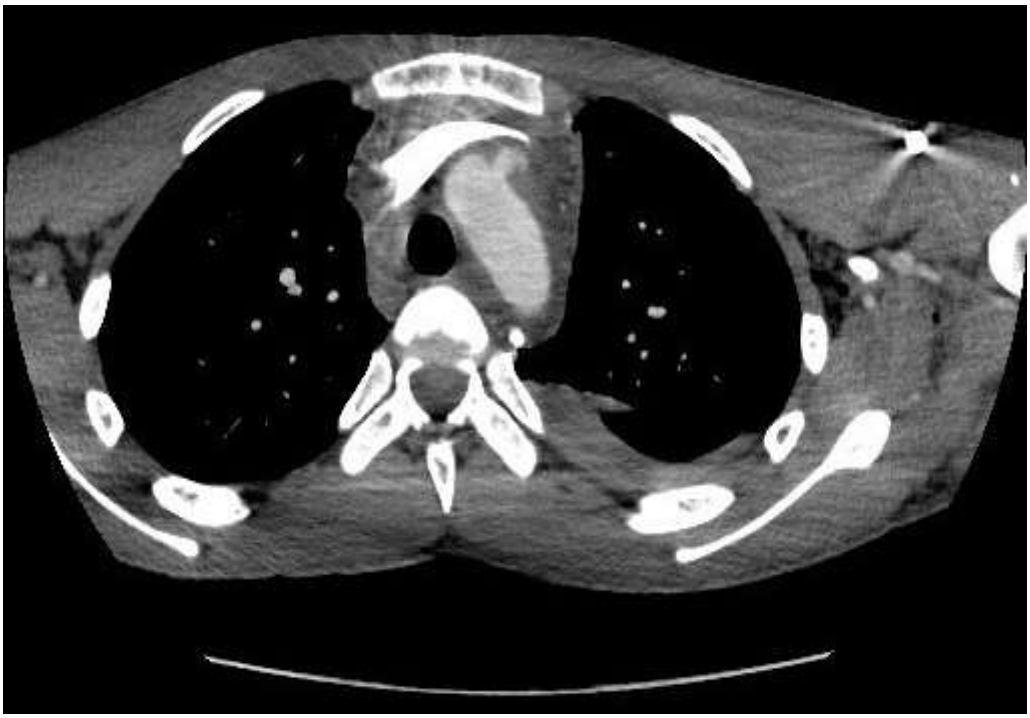

Fig 1:- Transversal slice of chest CT scan displaying the aortic breach.

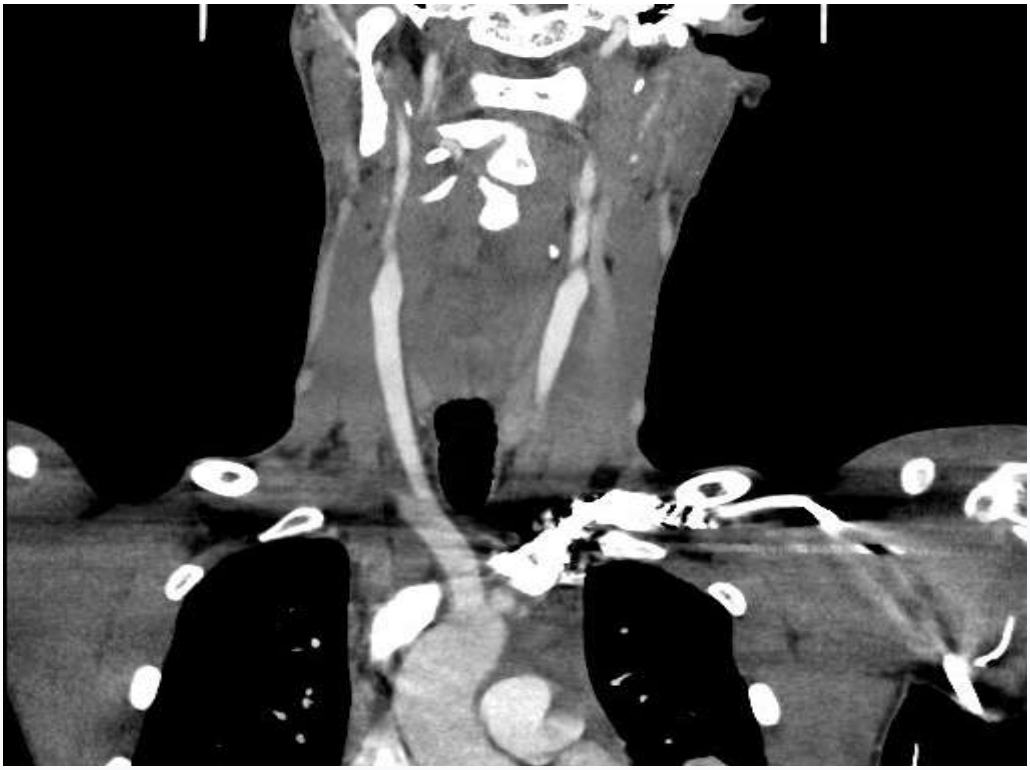

Fig 2:-coronalslice of chest CT scan displaying the aortic breach.

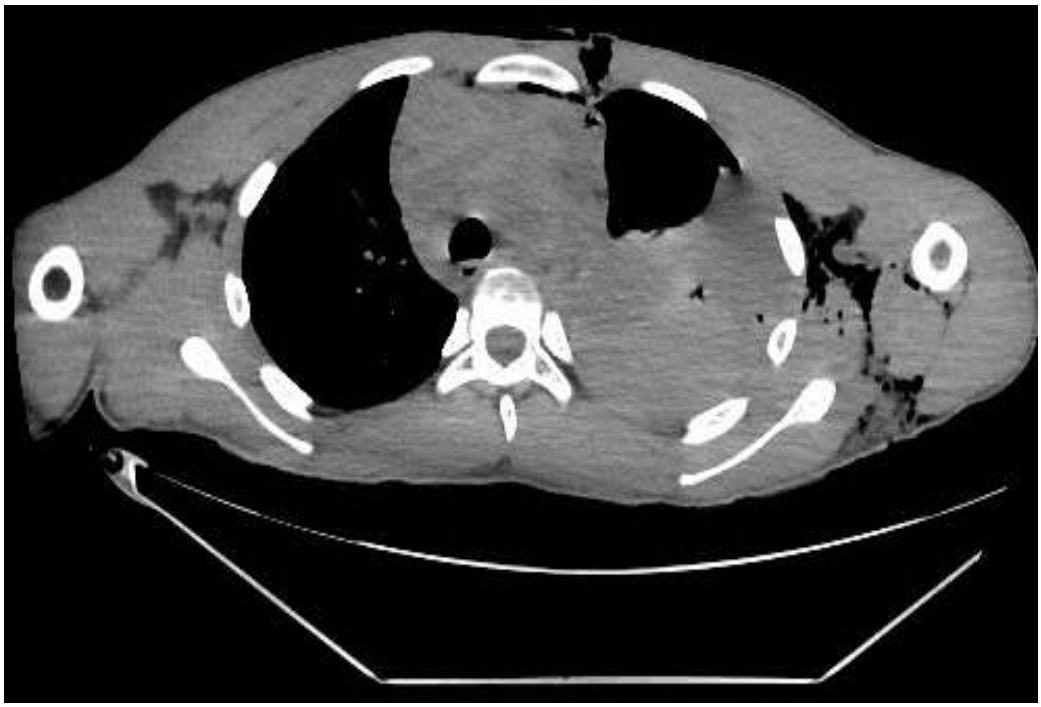

Fig 3:- Transversal slice of chest CT scan demonstrating hemopneumothorax and pneumomediastinum.

The hemopneumothorax was evacuated successfully by chest tube insertion, the complete blood count revealed a 16980/mm3 hyperleucocytosis including 80 percent neutrophils , and a 10 g/dl normocytic anemia.

An antibiotic tritherapy encompassing metronidazole, ciprofloxacine and gentamycine was installed as a probabilist treatment .

This succession of events happened 48 hours prior to the brutally occuring neurological impairement that manifested in a right hemiplegia followed by a sudden drop of the Glasgow Coma Scale was estimated at 6/15 (eye response at 1 , motor response at 4 with a right hemiplegia , and verbal response at 1) toxic and metabolic causes of acute onset coma were eliminated , and a hyperglycemia without ketosis was noted .

Another concomittent event was the onset of bradycardia and a widened pulse pressure (increasing systolic at 180mmHg , and decreasing diastolic at 55mmHg) that happenend right before intubation suspecting Cushing's reflex**.**

An anticipated rapidly progressive neurological decline warranted the performance of emergency neurological life support protocol that consisted of Intubation , control mode mechanical ventilation , and sedation using benzodiazepine and opioids .The neurological intervention was resquest right afterwards

Neurological clinical findings after the instauration of this protocol were as follows :

1. Absent brain stem reflexes includingfrontoorbicular reflex , vertical oculocephalic , photomotor , horizontal oculocephalicreflexes,excepting the oculocardiac reflex whichwaspresent ,
2. asymetricalunresponsivebilateralmydriasis pupilles
3. leftforcedconjugateeyedeviation signaling hemispheric topography ,
4. Generalizedflaccidity partlycaused by the administration of benzodiazepines , deep tendon reflexes abolition
5. Right Banbiski sign

A vascular mecanism involving the left hemisphere was suspected , however , The evaluation of severity of the stroke using the NIHSS **(**National Institutes of Health Stroke Scale**)** score was equal to 35 .

1A: Level of consciousness	**Unresponsive = +3**
1B: Ask month and age	**Intubated = +1**

1C: 'Blink eyes' & 'squeeze hands'	**Performes 0 tasks = +2**
2: Horizontal extraocular movements	**forced gaze palsy / cannot be overcome = +2**
3: Visual fields	**Patient bilaterally blind = +3**
4: Facial palsy	**Unilateral complete paralysis : lower and upper face = +3**
5A: Left arm motor drift	**No effort against gravity = +3**
5B: Right arm motor drift	**no movement = +4**
6A: Left leg motor drift	**No effort against gravity = +3**
6B: Right leg motor drift	**No movement = +4**
7: Limb Ataxia	**Does not understand = 0**
8: Sensation	**Coma/unresponsive = +2**
9: Language/aphasia	**Coma/unresponsive = +3**
10: Dysarthria	**Intubated/unable to test = 0**
11: Extinction/inattention	**Extinction to >1 modality = +2**

Table 1:- The NIHSS (National Institutes of Health Stroke Scale)of 35.

Non contrast brain CT scan was carried out subsequently , displaying a large wedge shaped region of low density within the left middle cerebral artery distribution , presicely the horizontal M1-segment (Fig 4 and 5) . Contrast CT scan revealed an occlusion of the right middle cerebral artery (Fig 7) .

Other findings were the enlargement of the controlateralventricule consistent with hydrocephalus , and a subfalcine hernia (Fig 6) causing 6.8 milimeters midline shift , no border zone infarcts were found .

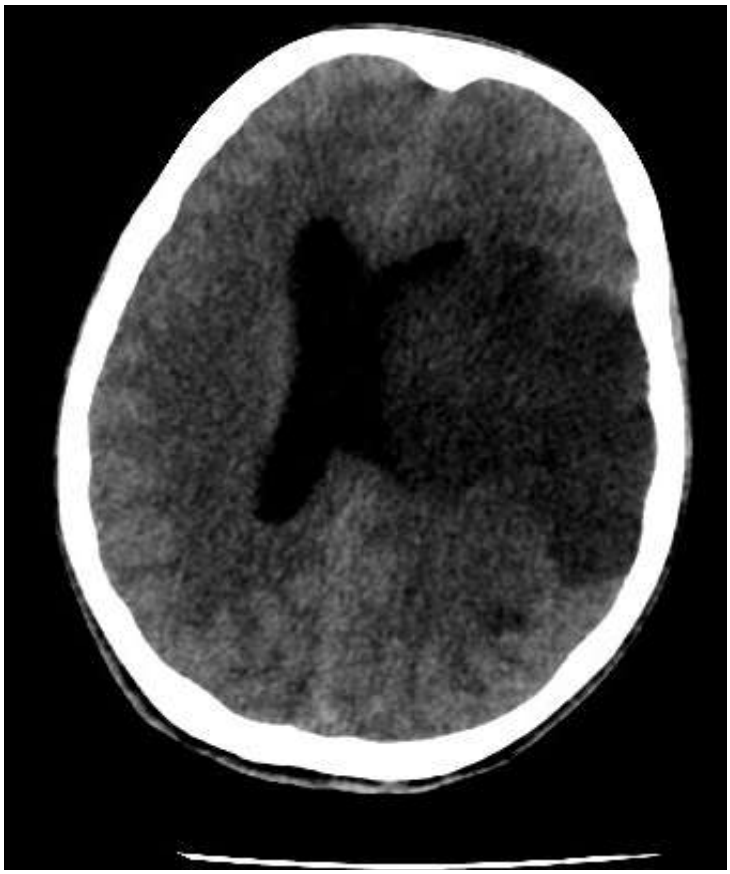

Fig 4:- Non contrast CT axial slice at the basal ganglia level demonstrating a large low density within the left middle cerebral artery distribution (caudate , internal capsule , lentiform nucleus , insular cortex , M2 (MCA cortex lateral to the insular ribbon) and M3(posterior MCA cortex)

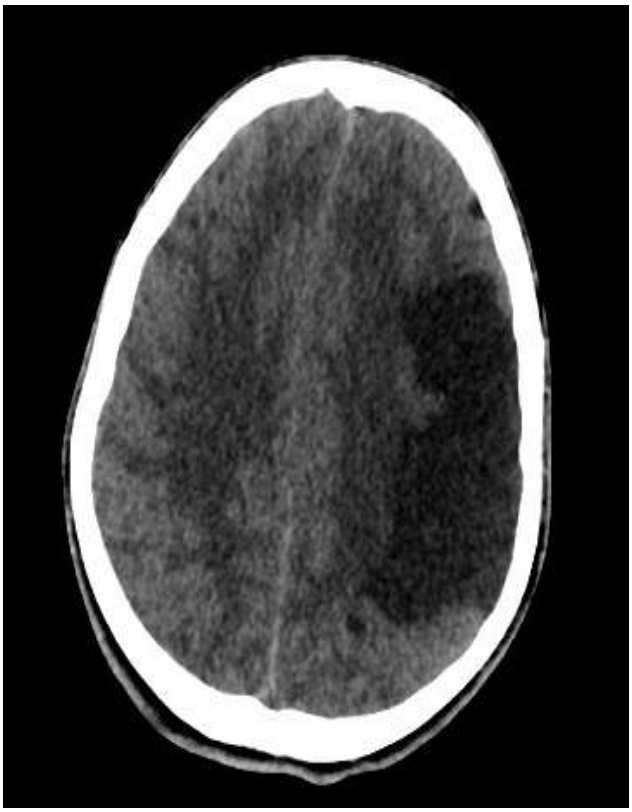

Fig 5:-Non contrast CT axial slice at the corona radiata level showinglow density within M5 (lateral MCA supperior territory) and M6 (posterior MCA superior territory).

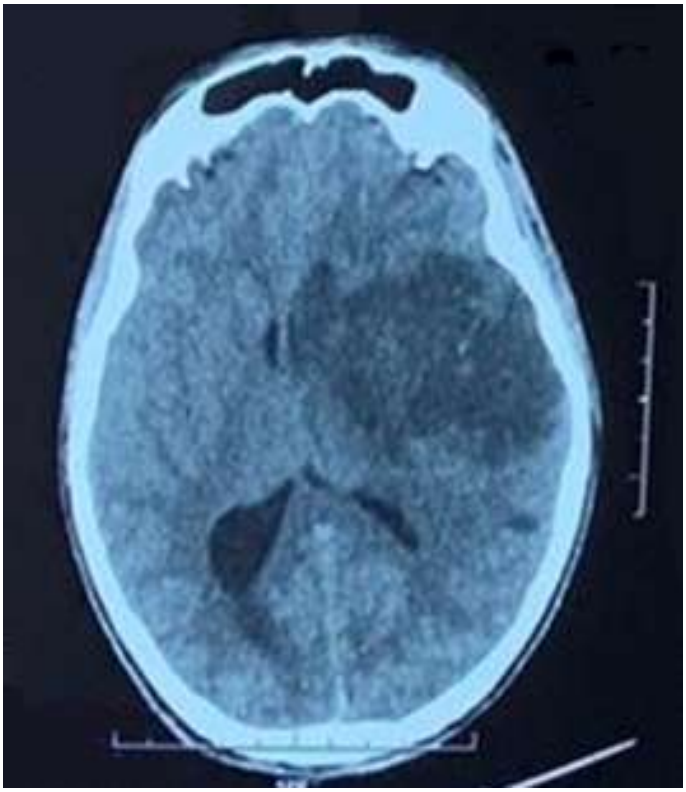

Fig 6:-Non contrast CT axial slice demonstrating subfalcine hernia.

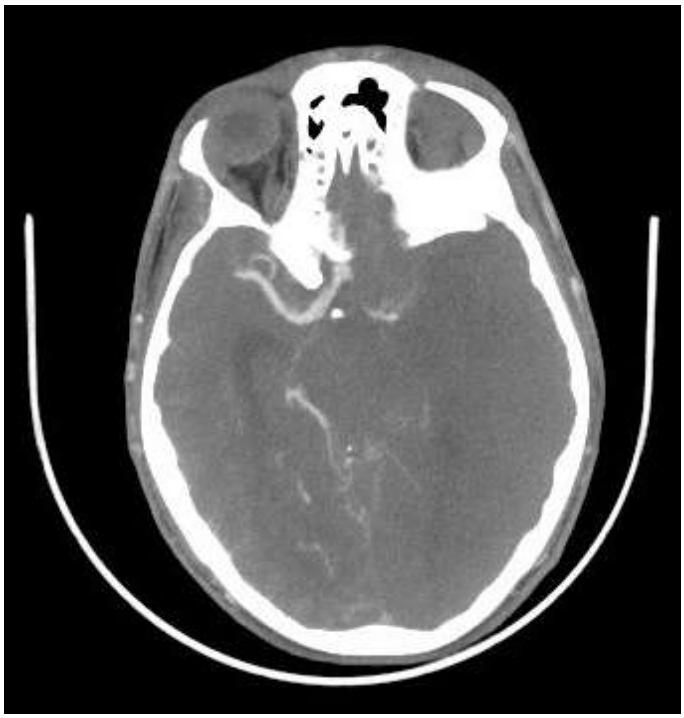

Fig 7:-Contrast CT scan showing the occlusion of the middle cerebral artery.

Thus , a preventive dose of low molecular weight heparin and an antiplatelet aggregation dose of acetylsalicylic acid were indicated . secondary systemic insults to the brain prevention was established . elevation of the head to the bed to 30 degrees was performed . The ventilation was set to aim normoxia and normocapnia .

Cardiological examination findings were poor , electrocardiography didn't detect any arrhythmia , transthoracic echocardiogram showed pericardial effusion .

The hemoglobin concentration decreased to 8 g/dl inspite of volume expansion and repeated red blood cell transfusions .

The rapid deterioration of hemodynamic parameters was attributed to the persistance of bleeding after 48 hours , the preventive anticoagulation was interrupted, and the patient reached class 4 hemorragic shock [1] implying a blood volume loss of 40 percent , arterial pressure reached 50/30mmHg , heart rate ranged between 63 and 130 beats per minute , the mean arterial pressure didn't exceed 54mmHg . Fluid resuscitation and vasoactive drugs were administered consequently (starting with epinephrin at 8gamma/kg/min) failing to reverse the refractory shock , therefore a decompression craniotomy and thrombolysis were unfeasible .

The patient passed away 4 days after the admission .

Discussion:-

The prognosis following MCA stroke primarily depends on the size of the necrosis and whether the patient received thrombolytic therapy and/or thrombectomy[7]

Studies have shown that younger patients are more prone to malignant MCA syndrome or fatal brain edema ,which is the leading cause of early death after stroke , than older patients who present cerebral atrophy [14 15 16].

The semiology of the focal neurological findings evoked an extensive hemispheric lesion , the acute onset directs towards a vascular aetiology , the initial neurological state rose the question of toxic or metabolic causes of coma , which were eliminated by blood tests .

Knowing that arterial dissection was eliminated , the most likely mecanism was a migrated sizable blood clot that generated from the aortic breach located next to the ostium of the brachiocephalic arterial trunk, that reached the intimal layer leading to the activation of the coagulation cascade , another factor of its formation was the non linear flow caused by the extravasation of blood through the arterial wound and the changing intrathoracic pressures caused by the hemopneumothorax and its drainage . The other suspected mechanism was air embolism by entering the arterial bed directly from the pneumothorax or the pneumomediastinum [9] .

Studies have shown that the Maximum velocities of 120 cm/s are found in the ascending aorta in supine position in males[2], which explains the upward migration of the clot towards the closest arterial branch to the ostium of the brachicephalic artery, which is the left common carotid artery , the internal carotid artery , then following the least resistance pathway , aiming at the middle cerebral artery , resulting in the occlusion of its horizontal M1 portion , therefore including lenticulostriate arteries , causing a massive large infarction comprising the surface cortex including peri-rolandic cortex , insular ribbon and striatocapsular territories containing petechial hemorrhages that are often associated with an embolic mechanism[3 4 5 6] , sparing the thalamus which is supplied by the posterior cerebral artery (PCA) translating into hypodensity in CT-scan . These findings showed that the stroke may have happened at least 24 hours after the beginning of the cerebral ischemia , in other words , during the acute phase[12] which probably was accelerated due to the pathophysiological mecanisms describe further below, this delay was caused because of the circumstances and history of the patient causing a setback regarding the resquest of neurological intervention , as well as the precise determining of chronological sequence of neurological findings .

The brain CT scan displayed an malignant MCA infarction , however , due to the persistant hemorrhage , fibrinolysis , thrombectomy and decompression craniotomy were contraindicated .

The first peak of the blood brain barrier permeability caused by its breakdown as a result of sudden hypoxia of neurons occurs as fast as a few hours of the onset , ie the hyperacute stage , the second peak is observed after 72h to 96h due to neuroinflammation processes motivated by the first cytotoxic events[11] , macromolecules are released from the vasculature to the extracellular space, pulling water according to the osmotic gradient .The result is a rapid generation of an extensive vasogenic edema [13] , causing mass effect on the brain. The cerebrospinal fluid volume and cerebral blood volume decrease as an attempt to maintain a normal intracranial pressure(ICP) .

The lesion's volume overwhelmed the compensatory mechanism to maintain a normal intracranial pressure (ICP) , causing subfalcine or cingulate hernia as shown in the imagery , proving that the ICP exceeded 28 cm H2O for more than 5 minutes[8] prior to brain CT scan , transcending the mean arterial blood pressure , thus , leading to a drop in cerebral perfusion pressure (CPP) , causing cerebral ischemia , therefore jeopardizing the penumbral region . Another outcome of brain ischemia was the activation of sympathetic response of the α1 adrenergic reaceptors causing hypertension and β1 adrenergic receptors causing an initial tachycardia that stimulated the baroreceptors resulting in bradycardia therefore a lower diastolic blood pressure , by activation of muscarinic M2 receptors of the heart , the intracranial hypertension would have caused a press on respiratory centers of the brain stem causing Cheyne-Stokes respirations [17]signaling the start of brain stem dysfunction. Clinically , these mechanisms were exhibited by two signs of the cushing reflex triad [10] since the patient was under controlled mode ventilation , masking the irregular breathing .The cingular hernia could have caused an added ischemia of the anterior cerebral artery (ACA) territory by trapping mechanism .

After the onset of Cushing's reflex , the mean arterial pressure (MAP) didn't exceed 54 mmHg as the patient entered the sympathoinhibitory phase of the hemorrhagic shock because of the persistent bleeding , surpassing the cerebral autoregulatory capacities , further compromising the chances of penumbral zone salvation , resulting in a accelerated evolution towards vasogenic edema and necrosis .

Vasoactive drugs were added in order to enhance the MAP, and elevation of the head to the bed to 30 degrees to achieve gravity induced caudal flow of CSF [18 19] was performed , these two measures sought the elevation of the CPP and the lowering of ICP respectively .

As for ventilation , available studies don't support the induction of hypercapnia to increase CPP [20]

Watershed stroke linked to this hemodynamic profile was likely to happen but the timing of effectuation of CT-scan was enough to only show the embolic stroke , because the later manifests itself chronologically earlier than the first .

Magnetic resonance imaging (MRI) would have determined this complication along with the probable ischemia caused by trapping of the ACA , however , it wasn't accessible .

In short , the hemodynamic instability figured as a contra indication to acute ischemic stroke treatments including thrombolysis , and its consequences counting decompression craniotomy and antiedematous agents . this refractory shock caused a dysfunction in cerebral autoregulation , causing arterial collapse further lowering the cerebral metabolic rate of oxygen (CMRO2) transport to the penumbral zone , accelerating the speed of generation of an extensive vasogenic edema contained in the ischemic territory of the MCA , triggering a faster onset of subfalcine hernia and non communicating hydrocephalus , As a result , a vicious cycle is created , dramatically worsening the prognosis .

This Particular combination of hemodynamic instability with malignant middle cerebral artery stroke resulted in an unfortunate prognosis leading to the decease of the patient .

Conclusion:-

The unpredictability of ischemic stroke in patients presenting remote trauma involving large vessels is the main cause of its late diagnosis . A coma following this circumstance should raise the question of the occurrence of ischemic stroke in order to shorten the timeframe preceding curative interventions thus reducing the risk of decease .

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