Case Report

Acute Non-Haemorrhagic Contusion: A Case Report

Abstract:

Introduction: The most common type of brain injury is traumatic brain injury (TBI). severe of all traumatic wounds, typically keeping physical, cognitive, behavioral, and emotional deficits. TBI is estimated to cost $60 billion a year including medical care in the United States expenses and lost productivity costs making it one of the cost healthcare expenses. Thurman et al., 1999; Langloisetal.,2006; Selassieetal.,2008; Langlois and others. Langlois .and others, 2006). To begin, we'll go at the theory, which states that a latent or explicit coagulopathy causes Bleeding from microvessels that were fractured during the primary injury may continue or be delayed. **Main symptoms and the important clinical findings:** - A male patient was admitted to Acharya Vinoba Bhave rural hospital on dated 17-07-2021 with the chief complaint of sudden paralysis in right upper limb and lower limb since 11/7/2021 exact time not know H/O RTA 9 years back with head trauma. After a physical examination and investigation, the doctor diagnoses a case of acute non-hemorrhagic contusion since at the 3 years back for which he was hospitalized for 20 days after the investigation was observed he took treatment for that and his outcome was good. acute non-hemorrhagic contusion depending on the severity of injury Headache confusion dizziness loss of nausea. Vomiting seizures, difficulty with coordination and moment lightheadedness, tinnitus, and spinning sensation. **Main Diagnostic therapeutic intervention and outcome:** After a physical examination and investigation doctor diagnoses a case of acute non-hemorrhagic contusion Tablet Levepsy 500mg BD, Tablet Dolo 650 mg TDS, Tab pantoprazole 40 mg BD, Tab Atorva40 mg given to a patient. The patient response to all medication and his outcome was good. trying to reserve any signs and symptoms that have appeared Doctor advised follow-up after 1 month a Sonography, blood investigation, and other examination to know the further disease progression. trying to reserve any signs and symptoms that have appeared Doctor advised follow up after 1 month a Sonography, blood investigation, and other examination to know the further disease progression **Conclusion:** trying to reserve any sign and symptoms that have appeared Doctor advised follow up after 1 month a Sonography, blood investigation, and other examination to know the further disease progression.

Keyword: - Acute Non-Haemorrhagic Contusion, Coagulopathy, Contusion, Traumatic Brain Injury, Haemorrhage, Cerebral Contusion

Introduction: -

The initial injury is caused by the kinetic energy created during the collision. Following a head injury, the severity of damage to cerebral tissue is characterized by several secondary injury reactions that almost always exacerbate the underlying injury.[1]

When there is a result of a concussion of the brain Traumatic brain injury when a concussion in the brain occurs as a result of a concussion During the first few hours after impact, the hemorrhagic lesion frequently progresses, either growing or creating new, no contiguous hemorrhagic lesion, a condition known as a contusion's hemorrhagic progression (HPC). As a result of a hemorrhagic contusion causes a complete and irreversible loss of function in tissues, as well as the truth that blood is one of the most corrosive substances on the planet.[2]

Nearly half of TBI survivors who are hospitalized have long-term disabilities. TBI is a term that refers to a variety of brain injuries. The hemorrhagic cerebral contusion is one of the most serious injury mechanisms. Nearly half of TBI survivors who are hospitalized have long-term disabilities. TBI is a term that encompasses a wide range of injuries. One of the most severe forms of harm is one of the mechanisms is hemorrhagic cerebral congestion. In TBI patients, TBI combined with brain contusions raises the risk of impairment and death. Cerebral contusions result in irreversible injury to the cerebrum's tissues. The original harm is a result of kinetic energy absorbed by the effect, as well as the c arcade of secondary injury reactions. pound the primary injury, determine the degree of the damage.[3]

Widespread axonal damage (DAI) is a type of form of tissue injury caused by abrupt rotational acceleration-deceleration stresses that causes stretching, disruption, and eventual separation of axonal fibers of fibers. Corticomedullary connections within the temporal and frontal lobes areas, the corpus callosum, upper brainstem, and deep brainstem are all part of the corpus callosum. grey matter, among others, are all common anatomical sites influenced by corticomedullary connections based on their neuropathological studies, Adams et al.3) described three grades of DAI: Grade 1 DAI is defined by axonal damage that is widespread and microscopic in anyplace; DAI 2nd grade is defined by grade 3 DAI, and localized anomalies in the corpus callosum are defined by lesions in the head and neck rostra brainstem. In comparison to prior X-ray or computer tomography approaches Because advancement in radiological procedures or Magnetic resonance imaging (MRI) is widely used in traumatic brain injury (TBI) patients to detect non-hemorrhagic injuries. localized the National Heart, Lung, and Blood Institute (NHL) has improved its efficiency. (CT). NHLs have been identified as a kind of DAI8,13) after head trauma. Since advancements in radiological techniques or the diagnosis the prevalence of localized non-hemorrhagic lesions (NHL) has

increased. been more effective thanks to traumatic brain injury (TBI) patients, magnetic resonance imaging (MRI) is commonly used. The 3diagnosis the prevalence of localized non-hemorrhagic lesions (NHL) has increased. been more effective thanks to the Magnetic resonance imaging (MRI) are commonly used in traumatic brain injury (TBI) patients. in contrast to an X-ray or computer tomography scan procedures (CT). Following a concussion, NHLs have already been identified as a considerate DAI8,13) is a subset of DAI8.[4]

NHL is defined by Shear injury, traumatic white matter damage, and the cytoskeleton option all are possibilities. Retraction ball, delayed taxonomy, and cytoskeleton disruption are all pathogenic features in NHL. The distribution, evolution, and developmental mechanisms of NHL, on the other hand, are completely unknown.[5]

HPCs stand for hemorrhagic progression of contusions. the. After initial imaging, the hemorrhagic component of brain contusions enlarges. traumatic brain injury (TBI) (TBI). HPC, commonly called “blossoming” concussions, can cause neurological deterioration as a result of the mass effect, cerebral edema, and necrosis of parenchymal tissue HPC has been reported in TBI patients at rates ranging from 18 to 51 percent in previous studies done. Early diagnosis of high-risk patients HPC may lead to solutions that minimize pollution. TBI mortalities and morbidity.[6]

Patient-specific information: A 50 yrs old male was admitted to AVBRH on the date 17 -7-21 with a chief complaint of sudden paralysis in Rt ul and LL since 11/7/2021axact time not know H/O RTA 9 years back with head trauma. After a physical examination and investigation, the doctor diagnoses a case of acute non-hemorrhagic contusion since at the 3 years back for which he was hospitalized for 20 days after the investigation was observed he took treatment for that and his outcome was not good

Primary concerns and symptoms of the patient: -chief complaint of sudden paralysis in right UL and LL since 11/7/21axact time does not know H/O RTA 9 years back with head trauma. were the primary symptoms which were observed at the time of admission.

Medical, family, and psychosocial history: -the present case had a history of any medical history of acute non-hemorrhagic contusion he had maintained a good relationship with doctors and nurses as well as to patients also.

Relevant past intervention with the outcome: - History of acute non-Haemorrhagic contusion he was hospitalized 20 days after the investigation was observed he took treatment for that and his outcome was not good after blood urine tests, radiography. ultrasound. Computerized tomography scan rt said x-ray after investigation acute non Hemorrhagic contusion and his outcome was good.

Clinical findings: - The patient was conscious and well oriented to date time and place his body build was moderate and he had maintained good personal hygiene, his vital states of health were unhealthy thick body build the height of the patient 127 cm and weight is 140kg vital signs normal RT, UL AND LL paralysis

Timeline: -. History of acute non-Haemorrhagic contusion he was hospitalized for 20 days after the investigation was observed he took treatment for that and his outcome was not good currently she was admitted for the treatment of acute non-hemorrhagic contusion, tab, levepsy 500mg BD. Tab. Dolo 650mg TDS drug is given and calcium and multivitamin supplementary was given for 7 days to enhance immune function he was taking all treatment and the outcome was good his sign and symptoms were reduced.

Diagnostic assessment: -

Diagnostic challenges: No challenges during diagnostic evaluation

Diagnosis: -After a physical examination and investigation doctor diagnose a case of acute hemorrhagic contusion**.**

Prognosis: was good

Therapeutic intervention: -medical management was provided to the patient calcium and multivitamin supplementary was given for 8 days to enhance immune function tab. Levepsy 500 mg BD drug is given and calcium and multivitamin supplementary was given for 7 days to enhance immune function he was taking all treatment and the outcome was good.

Follow-up and outcome: -

Clinical and patient assessment outcome: -patient condition was improved.

Important checkout investigation and other test results: - To prevent the progression of disease trying to reserve any signs and symptoms that have appeared Doctor advised follow-up after 1 month a Sonography, blood investigation, and other examination to know the further disease progression.

Discussion: -

DAI was described as Injury caused by shearing that causes harm to a large area of the white matter of the brain. of instantaneous Diffuse white matter shearing8 damage, impact type, and 8cerebral inner trauma. Adams et al.3 is a group of researchers who came up with

a novel way to question whether focused damage to the stem of the brain could occur as a consequence of a single head blow and claimed that DAI constituted a primary brain injury clinical syndrome, classifying it into three classes based on the involvement of the brain stem. Grades are assigned based on the areas that are involved Their study was based on the histopathological findings as they pointed out, head injuries, which are becoming more common, could have been disguised by other people types or brain contusions and hematomas injury-related due to a rise in intracranial pressure. based on hypoxia, or infarctions, and their research focused on this. done The identification and classification of less severe crimes in forensic work injuries could have been more difficult. Grades are assigned based on the areas that are involved. Their investigation was based on the histological findings of There were 434 cases of non-muscle head injuries in this research, and as they pointed out, DAI could have been concealed by other sorts of comorbid conditions. [7,8]

Although have found and described NHLs, however, they are not clearly defined. Five examples of DAIs were described by Topal et al.28). with a GCS score of 13 to 15 who had suffered a moderate head injury. They stated that one of the five cases exhibited a hyperintense lesion the lesion was "non-hemorrhagic," with no signal On FLAIR and DW images, there is a signal change, and not on GRE images. On FLAIR and DW images, there is a signal shift, and not on GRE images. However, there has been no change in the GRE signal. However, there has been no change in the GRE signal. images Acute callosal lesions were discovered. frequently non-hemorrhagic in nature origin, accordingtoGentryetal.8, because 21 out of 31 instances (67.7 percent) revealed nature (non-hemorrhagic) on MRI. The lesion was "non-hemorrhagic," with no signal change on DW and FLAIR images, However, there has been no change in the GRE signal. images. Acute callosal lesions were discovered. Frequently non-hemorrhagic in origin, accordingtoGentryetal.8, revealed reason 21 from out 31 cases (67.7 percent) nature (non-hemorrhagic). on MRI. They stated that one of the five instances' Modes: [9,10]

had a blemish that wasFLAIR and DW hyperintense images but did not show any change in the GRE signal images and that the lesion was 'non-hemorrhagic. Acute callosal lesions were discovered. typically, non-hemorrhagic in nature on MRI characteristics, according to Gentry et al.8, because 21 of 31 cases (67.7%) indicated nature (non-hemorrhagic) However, edema surrounding the hemorrhagic lesion appeared to be present in these cases, as they stated, "Usually, substantial zones of non-hemorrhagic of damage encircled the bloody foci" he Brain stem injuries were also recorded by the same authors, but they were only cited once. that T2-weighted MRI is more detectable than T1-weighted MRI or CCT7.[11]

had a hyperintense lesion on DW and FLAI Rimage, However, there has been no change in the GRE signal. images, and that the lesion was 'non-hemorrhagic.' According to Acute callosal lesions were found in Gentry et al.8, usually, is non-hemorrhagic based on MRI findings as 21 of 31 patients (67.7%) were non-hemorrhagic. "Usually, vast zones of non-hemorrhagic damage surrounding the blood foci, "surrounding the blood foci," they wrote, "but in few cases, edema appeared to surround the hemorrhagic lesion. " T2-weighted MRI is more detectable than T1-weighted MRI or CT7), according to the same writers who also reported brain stem damage.[10]

We identified factors associated with HPC In a group of 286 people individuals Those who have had a mild or severe blunt TBI, developed the to stratify risk for HPC. This HPC Score ranges from 0 to 4, and it's 2 points for SAH, 1 point for SDH, and 1 point for SDH for the existence of a skull fracture.HPC incidence was with scores of 0–4, the percentages were 0, 3.7, 7.7, 28.9, and 39.5 percent, respectively. The presence of SAH, skull fracture, and SDH were identified as important risk factors for HPC. as we had previously reported.[11]

Conclusion: -

Trying to reserve any signs and symptoms that have appeared Doctor advised follow-up after 1 month a Sonography, blood investigation, and other examination to know the further disease progression.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly used products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by the personal efforts of the authors.

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