ORIGINAL RESEARCH PAPER
Volume - 11 | Issue - 03 | March - 2022 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

POST TRAUMATIC CEREBRAL FAT EMBOLISM

Radiodiagnosis

Dr. Manjot Kaur* Professor and Head, Dept. of Radiodiagnosis. *Corresponding Author

Dr. Anshul Junior Resident, Dept. of Radiodiagnosis.

ABSTRACT

Cerebral fat embolism is an uncommon type of fat embolism syndrome usually preceded by long bone fractures of lower limbs. It is quite challenging to diagnose with diagnosis based on clinical features and on the exclusion of other diseases. Neuroimaging is helpful to hasten the diagnosis and management. Here, we present a case of young adult presenting to us with traumatic cerebral fat embolism.

KEYWORDS

INTRODUCTION-

Cerebral fat embolism (CFE) is a rare and potentially fatal condition that may occur following a long bone fracture or pelvis trauma, showing an incidence ranging from 0.9 to 11% with a mean mortality rate around 10%.[1-3] Although it is usually self-limiting, it may be fatal (mortality rate of up to 10%).[4] FES diagnosis is only based on clinical features and on the exclusion of other diseases but it remains a difficult task as there are no universal criteria for diagnosis and laboratory tests are nonspecific.[5] When a diagnosis of cerebral fat embolism is suspected, a cerebral MRI must be performed, followed by supportive treatments focused on the clinical symptoms, and hemodynamic and respiratory support.[6]

We report a case of a young male who presented with symptoms of severe cerebral FES after a road traffic accident.

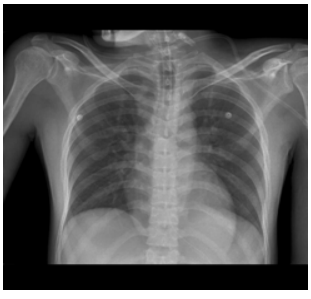
Case Report –

A twenty six year old male presented to Adesh Institute of Medical Sciences in casualty with alleged history of assault in disoriented state with GCS of $E_2V_2M_5$ with history of loss of consciousness after trauma followed by multiple episodes of vomiting although there was no history of any bleed from ear, nose or mouth. On further examination, His PR was 112/min with BP of 120/80 mm of Hg with Spo2 of 98% on oxygen support at 8lt/min with multiple fractures and wounds were present over bilateral lower limbs.

X ray findings –

Comminuted fracture of tibial and fibular shaft (FIG – 1)

X ray chest was done and was grossly normal (FIG -2)



Outside report of NCCT head revealed –

Mild effacement of bilateral cerebral cortical sulci s/o cerebral edema.

As conscious level of patient didn't improve,MRI Brain was planned and done in our institute.

Findings revealed –

Multiple punctate non confluent T2 and FLAIR hyperintensities in deep and periventricular white matter of bilateral cerebral hemispheres in bilateral corona radiata, centrum semiovale, basal ganglia, thalami and corpus callosum.(FIG.3a)

On DWI, these areas are seen as multiple punctate foci showing diffusion restriction and appearing as starfield that is multiple scattered white spots against dark background s/o cytotoxic edema. Hyperintensities on DWI indicate low diffusivity appearing as hypointensities on ADC mapping. (FIG. 3b)

No obvious blooming is seen on SWI. (FIG. 3c)

Ventricular system is normally defined.No midline shift is seen.Findings suggestcerebral fat embolism.

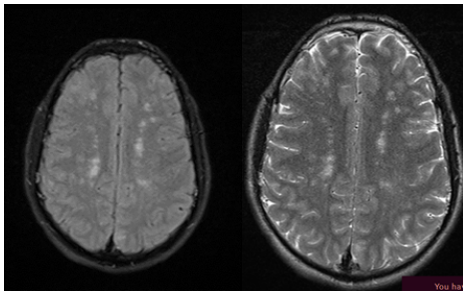


FIG. 3a

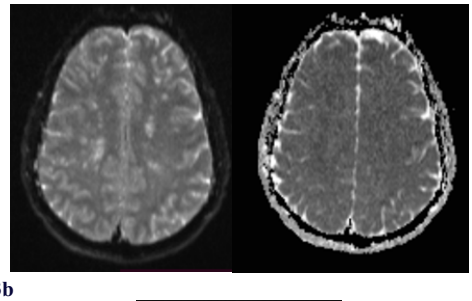


FIG. 3b

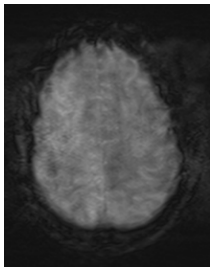


FIG. 3c

DISCUSSION:-

Cerebral fat embolism is commonly reported in younger patients with long bone fractures, especially femoral and tibial fractures.[7] Patients with isolated cerebral fat embolism have no respiratory dysfunction but exhibit a change of consciousness, which is usually accompanied by new-onset neurological deficit, apathy, convulsion, hallucination, facial palsy, or hemiplegia. It typically manifests with petechial rash, deteriorating mental status, and progressive respiratory insufficiency, usually occurring within 24–48 h of trauma with long-bone fractures or an orthopedic surgery.[8] Gurd and Wilson's criteria are the diagnostic criteria widely used today but they are nonspecific and have never been validated in large cohorts.[9] Although the patient in the current case did not initially meet Gurd and Wilson's criteria (only one major and one minor criteria were met: cerebral symptoms in a patient with non-head injury and tachycardia). MR imaging of the brain by using T2-weighted, diffusion-weighted, gadolinium contrast-enhanced images, and susceptibility-weighted imaging has been applied to CFE and has improved the ability to make a diagnosis.[10]

In addition, the fluid-attenuated inversion recovery sequence is useful in detecting these lesions in their acute stage and revealing their watershed distribution.[11] On diffusion-weighted imaging (DWI), the areas of cytotoxic edema appear as a "starfield," i.e., multiple scattered white spots against dark background.[12] In addition, hyperintensities on DWI indicate low diffusivity, appearing as hypointensities on apparent diffusion coefficient map.[13]

CONCLUSION:

CFE is an uncommon but potentially life threating condition with a challenging diagnosis due to variable and non specific signs and symptoms. However, neuroimaging can be quite helpful in early diagnosis and management.

REFERENCES

1. Bulger EM, Smith DG, Maier RV, Jurkovich GJ. Fat embolism syndrome: a 10-year review. Archives of surgery. 1997 Apr 1;132(4):435-9.
2. Fabian TC, Hoots AV, Stanford DS, Patterson CR, Mangiante EC. Fat embolism syndrome: prospective evaluation in 92 fracture patients. Critical care medicine. 1990 Jan 1;18(1):42-6.
3. Aman J, van Koppenhagen L, Snoek AM, van der Hoeven JG, van der Lely AJ. Cerebral fat embolism after bone fractures. The Lancet. 2015 Oct 3;386(10001):e16.
4. Kosova E, Bergmark B, Piazza G. Fat embolism syndrome. Circulation. 2015 Jan 20;131(3):317-20.
5. Shaikh N, Parchani A, Bhat V, Kattren MA. Fat embolism syndrome: clinical and imaging considerations: case report and review of literature. Indian journal of critical care medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine. 2008 Jan;12(1):32.
6. Kuo KH, Pan YJ, Lai YJ, Cheung WK, Chang FC, Jarosz J. Dynamic MR imaging patterns of cerebral fat embolism: a systematic review with illustrative cases. American Journal of Neuroradiology. 2014 Jun 1;35(6):1052-7.
7. Gupta B, Kaur M, D'souza N, Dey CK, Shende S, Kumar A, Gamangatti S. Cerebral fat embolism: A diagnostic challenge. Saudi journal of anaesthesia. 2011 Jul;5(3):348.
8. Talbot M, Schemitsch EH. Fat embolism syndrome: history, definition, epidemiology. Injury. 2006 Oct 1;37(4):S3-7.
9. Gurd AR. Fat embolism: an aid to diagnosis. The Journal of bone and joint surgery. British volume. 1970 Nov;52(4):732-7.
10. Suh SI, Seol HY, Seo WK, Koh SB. Cerebral fat embolism: susceptibility-weighted magnetic resonance imaging. Archives of neurology. 2009 Sep 1;66(9):1170-.
11. Brant-Zawadzki M, Atkinson D, Detrick M, Bradley WG, Scidmore G. Fluid-attenuated inversion recovery (FLAIR) for assessment of cerebral infarction: initial clinical experience in 50 patients. Stroke. 1996 Jul;27(7):1187-91.
12. Parizel PM, Demey HE, Veeckmans G, Verstreken F, Cras P, Jorens PG, De Schepper AM. Early diagnosis of cerebral fat embolism syndrome by diffusion-weighted MRI (starfield pattern). Stroke. 2001 Dec 1;32(12):2942-4.
13. Kim HJ, Lee CH, Lee SH, Moon TY. Magnetic resonance imaging and histologic findings of experimental cerebral fat embolism. Investigative radiology. 2003 Oct 1;38(10):625-34.