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MRI EVALUATION OF VENTRICULOMEGALY IN PEDIATRIC PATIENTS

Radiodiagnosis

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ABSTRACT

BACKGROUND: Ventriculomegaly refers to enlargement of the ventricles. It is crucial to differentiate hydrocephalus from ex vacuo enlargement of the ventricles. MRI is an excellent tool to assess the ventricles and aid in diagnosis and management of patients with ventriculomegaly. **AIM:** The purpose of the study was to establish role of MRI as the primary imaging modality in the evaluation of ventriculomegaly and to distinguish it from hydrocephalus along with illustrations of various imaging patterns. **CONCLUSION:** Overall, congenital and developmental causes were the most common causes of ventriculomegaly.

KEYWORDS

MRI, Ventriculomegaly, Hydrocephalus, Pediatric patients.

INTRODUCTION

Ventriculomegaly refers to enlargement of the ventricles.

By convention, ventriculomegaly associated with increased intracranial pressure is termed "hydrocephalus." It is crucial to differentiate hydrocephalus from ex vacuo enlargement of the ventricles as a result of volume loss or from congenital anomalies with associated ventriculomegaly. Hydrocephalus is one of the most common sequelae of any insult to a child's central nervous system. Hydrocephalus is the end result of many different processes that lead to enlarging ventricles with compression of brain parenchyma and subarachnoid spaces, which in turn leads to raised intracranial pressure (ICP)[1].

Ventriculomegaly is defined as lateral ventricular diameter > 10mm across the atria of the lateral ventricles. (The measurement should be in axial plane at the atria of lateral ventricles and glomus of choroid plexus and the ventricle is measured from the inner margin of the medial ventricular wall to inner margin of lateral wall). The severity of ventriculomegaly can be classified as Mild/Borderline (10-12mm), Moderate(12-15mm), Severe (>15mm) based on lateral ventricular diameter[2].

Hydrocephalus is a condition wherein excess of CSF accumulates within the ventricular system and cisterns of the brain leading to increased ICP and related consequences.Numerous definitions of hydrocephalus have been proposed. Summarily, it can be described as an imbalance between production and absorption of CSF[3].

In the majority of cases, the physiological mechanism underlying hydrocephalus is an obstruction of the CSF circulation, reduced re-absorption and, in a few cases, the overproduction of CSF. Hydrocephalus can be classified into communicating and obstructive forms; the difference is whether or not there is a free flow of CSF from the ventricles through the aqueduct and foramina to the spinal compartment (Dandy 1920)[4]. Dandy stated that almost every kind of hydrocephalus could be called obstructive, as it is the CSF absorption that is obstructed in communicating hydrocephalus.

Signsofhydrocephalusin radiology:

CT and MRI are used as primary modalities to assess ventricular size. Cranial USG is used as the initial study in infants with macrocephaly. Several parameters can help differentiate between hydrocephalus & ex-vacuo dilatation of ventricles due to cerebral atrophy, and the most reliable sign of hydrocephalus is enlargement of the anterior and posterior recesses of the third ventricle, this phenomenon does not occur in ex -vacuo ventricular enlargement[5].

The most commonly used radiological criteria in the diagnosis of hydrocephalus are given below:

1. Ventriculomegaly(Evans' index>0.3). Evan's index is the ratio of maximum width of the frontal horns of the lateral ventricles and the maximum internal diameter of the skull at the same level in axial CT/MR images.
2. Enlargement of the third ventricular recesses and lateral ventricular horns. (Most useful sign)
3. Narrowing of Ventricular angle/ Frontal horn angle/ Callosal angle. (The ventricular angle measures the divergence of the frontal horns.The enlargement of the frontal horn radius gives it a rounded configuration of the frontal horns, or a "Mickey Mouse ears" appearance).
4. Commensurate dilatation of the temporal horn with the lateral ventricles.
5. Decreased mamillo-pontine distance (It is measured on MRI from the anterior root of the mammillary body to the top of the pons parallel to the midbrain. The normal average is 3.8mm)
6. Thinning and elevation of the corpus callosum
7. Normal or narrow cortical sulci (effacement)
8. Periventricular white matter hyperintensities (suggesting interstitial edema & acute hydrocephalus)
9. Aqueductal flow void phenomenon in T2W images (a sign of communicating hydrocephalus).

The most significant finding on MRI to discriminate between acute and chronic forms of hydrocephalus is periventricular hyperintensities on T2W or FLAIR images, which is consistent with interstitial edema[6].

AIMS AND OBJECTIVES

- To establish role of magnetic resonance imaging as the primary imaging modality in the evaluation ventriculomegaly in pediatric patients.
- To study the clinico-radiological spectrum of ventriculomegaly on MRI with illustration of the imaging patterns.
- To assess and discuss etiologies of ventriculomegaly in pediatric patients and further categorize them into congenital or acquired with other radiological modalities, if needed.
- To establish an accurate diagnosis so as to aid in the early management of the pediatric patients with ventriculomegaly.

MATERIALS AND METHODS

This study was done in the department of Radio-diagnosis, Maharani Laxmi Bai Medical College, Jhansi (U.P), India, on 0.3 Tesla MRI machine. (HITACHI 0.3T Aris Elite)

The group under study comprised of patients of pediatric age group attending the outdoor and indoor of Department of Pediatrics of this hospital from May 2019 to October 2020 referred to us with neurological signs and symptoms. Sample size- 120 cases

Inclusion criteria:

All symptomatic patients referred from the Department of Pediatrics for brain MRI with neurological signs and symptoms, newly born upto 18 years of age, to diagnose the cause of ventriculomegaly on MRI brain were included after taking parents' consent for the study.

Exclusion criteria:

- Patients with history of trauma.

- Very sick patients.
- Idiopathic intra-cranial hypertension.

Methods:

A detailed history including details of the age of the mother, parity, time of delivery, antenatal, birth and postnatal history were noted.The patients were placed in supine position & head placed in a specifically designed 24cm split, clear receiver coil. Immobilization of the head was achieved by surrounding the head with an air evacuated bag filled with polystyrene balls; following which the scan was performed under the review of radiologists. Relevant investigations like MRI Spine, CT, Transcranial USG, CXR, USG abdomen, CSF examinations, CBC, ESR, ELISA and Interferon Gamma for tuberculosis, Serum electrolytes, blood culture, routine/microscopy/culture of urine were done to aid in the diagnosis. Ocular exams& genetic history was recorded in all patients.

Finally, collected data was analyzed & MRI findings were correlated with clinical and laboratory picture to arrive at final diagnosis.

OBSERVATIONS:

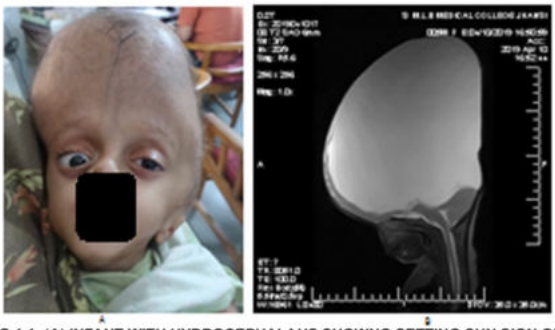


FIG 1.1: (A) INFANT WITH HYDROCEPHALAUS SHOWNG SETTING SUN SIGN OF RAISED ICP B) SAG T2WI MR IMAGE SHOWS MASSIVE HYDROCEPHALUS (WITH CORTICAL THINNING) DUE TO AQUEDUCTAL STENOSIS

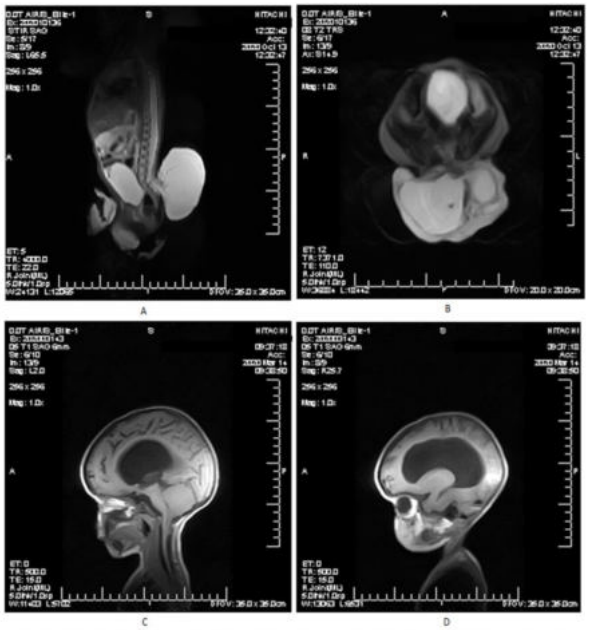
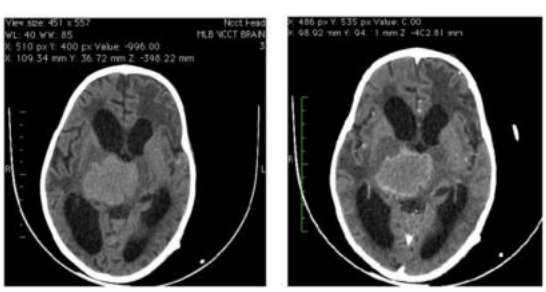


FIG1.2: CHIARI II MALFORMATION(A) SAG T2WI (B) AXIAL T2WI OF LS SPINE &SAG T1WI OF BRAIN (C, D)SHOWS LUMBOSACRAL MYELOMENINGOCELE WITH SMALL POSTERIOR FOSSA, TONSILLAR AND BRAINSTEM HERNIATION WITH HYDROCEPHALUS



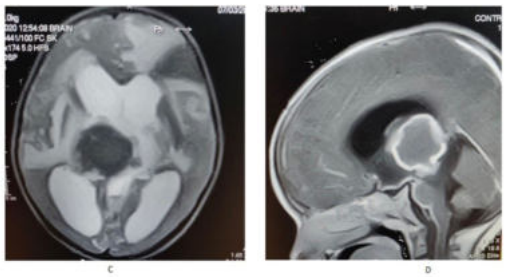
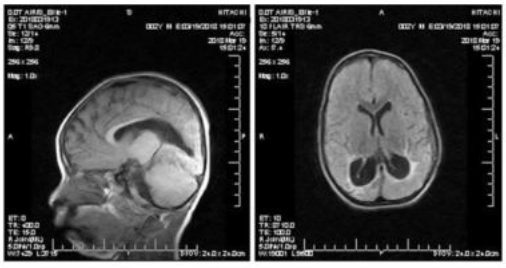


FIG 1.3: ATYPICAL TUBERCULOMA WITH MENINGITIS (A) AXIAL NCCT, (B) AXIAL POST CONTRAST CT IMAGE SHOWING A LARGE HYPERDENSE RING ENHANCING LESION IN RIGHT THALAMUS WITH EDEMA CAUSING MASS EFFECT ON 3RD VENTRICLE AND CAUSING NON-COMMUNICATING HYDROCEPHALUS. (C) T2WI IMAGE SHOWING THE LESION IS HYPOINTENSE (D) T1+C IMAGE SHOWING RING ENHANCEMENT OF THE LESION WITH LEPTOMENINGEAL ENHANCEMENT. FEW SIMILAR LESIONS WERE NOTED IN LEFT FRONTAL LOBE TOO (NOT SHOWN IN IMAGES)



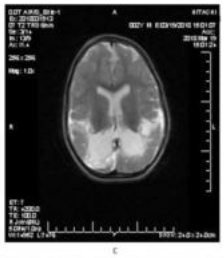


FIG 2.4: HYPOXIC ISCHEMIC ENEPHALOPATHY WITH CALLOSAL DYSGENESIS: (A) SAG T1WI AND (B) AXIAL FLAIR IMAGE SHOWING CORPUS CALLOSAL DYSGENESIS WITHCOLPOCEPHALY& ADJACENT GLIOSIS (C) AXIAL T2WI SHOWING B/L PERIROLANDIC GLIOSIS AND WHITE MATTER PAUCITY

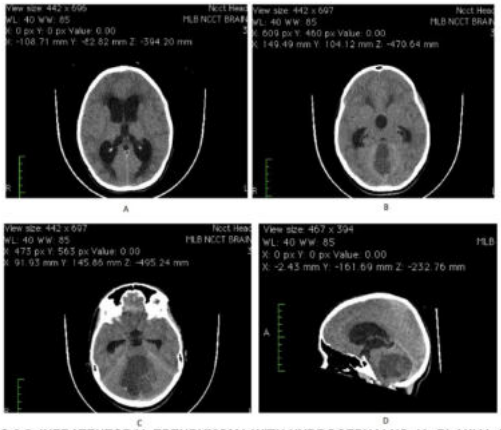


FIG 2.5: INFRATENTORAL EPENDYMOMA WITH HYDROCEPHALUS (A, B) AXIAL CT SCAN SHOWS DILATED LATERAL VENTRICLES & 3RD VENTRICLE WITH PERIVENTRICULAR INTERSTITIAL EDEMA ; (C, D) AXIAL AND SAGITTAL CT SECTIONS SHOWING HETEROGENEOUS MASS ARISING FROM 4th VENTRICLE, WITH FEW HEMORRHAGIC FOCI, CAUSING MASS EFFECT IN THE FORM OF OBSTRUCTIVE NON-COMMUNICATING HYDROCEPHALUS. [NOTE: BULGING CHIASMATIC AND INFUNDIBULAR RECESSESOF 3RD VENTRICLE IN IMAGE (D)]

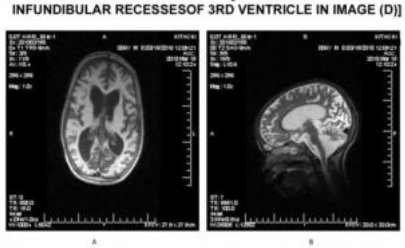


FIG 2.6: DIFFUSE BRAIN ATROPHY (A) AXIAL T1WI AND (B) SAG T2WI SHOWING DILATED SULCAL SPACES, SYLVIAN FISSURE, CEREBELLAR FOLIA WITH EX-VACUO DILATATION OF ALL VENTRICLES

RESULTS:

Table 3.1: Age wise distribution of study subjects (n=120)

Age group	No.	%
<1 month	14	11.7
1-12 months	52	43.3
1year-5 years	30	25.0
5-12 years	12	10.0
12 years-18 years	12	10.0

Table 3.2:Gender wise distribution of study subjects (n=120)

Gender	No.	%
Male	68	56.7
Female	52	43.3

Table 3.3:Maternal folic acid intake in study subjects (n=120)

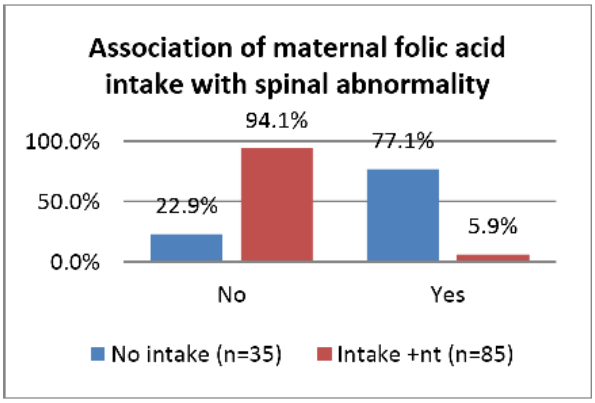
Maternal folic acid intake	No.	%
Yes	85	70.8
No	35	29.2

Table 3.4:Spinal abnormality in study subjects (n=120)

Spinal abnormality	No.	%
Yes	32	26.7
No	88	73.3

Table 3.5: Association of maternal folic acid intake with spinal abnormality

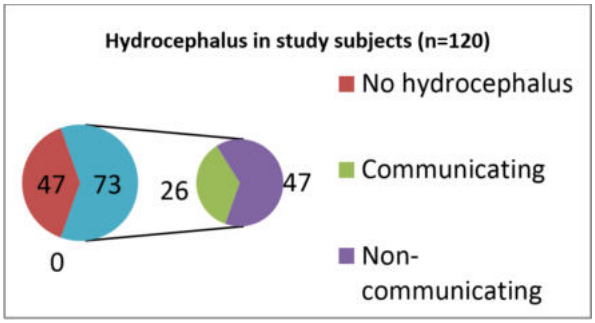
Spinal abnormality	No intake (n=35)		Intake +nt (n=85)		P-value
	Number	%	Number	%	
No	8	22.9	80	94.1	<0.001
Yes	27	77.1	5	5.9	



Graph 1: Association of maternal folic acid intake with spinal abnormality

Table 3.6:Hydrocephalus in study subjects (n=120)

Hydrocephalus	No.	%
Yes	73	60.8
No	47	39.2
Type of hydrocephalus (n=73)		
Communicating	26	35.6
Non-communicating	47	64.3



Graph 2:Hydrocephalus in study subjects (n=120)

Table 3.7:Causes of ventriculomegaly in study subjects (n=120)

Causes of ventriculomegaly	No.	%
Congenital/developmental	56	46.7
Infective	25	20.8
Neurovascular	12	10.0
Neoplastic	4	3.3
Ex-vacuo	20	16.7
Normal variant	3	2.5

Table 3.8: Distribution of causes of ventriculomegaly in study subjects (n=120)

	Causes of ventriculomegaly	No.	%
Congenital/ Developmental Causes (n=56)	Aqueductal stenosis	5	4.2
	MMC	19	15.4
	• MMC with meningtis • MMC with aqueductal stenosis • MMC only	6 12 1	5.0 10.0 8.3
	Chiari Malformations	15	12.5
	• Chiari 1 • Chiari 2 • Chiari 3	1 13 1	0.8 10.8 0.8
	Tuberous sclerosis complex	4	3.3
	Dandy walker continuum	4	3.3
	Encephalocele	3	2.5
	Meningoencephalocele	1	0.8
	CC Dysgenesis	1	0.8
	BESS	2	1.7
	Pachygyria	1	0.8
	Porencephalic cyst	1	1.7
Infective causes (n=25)	Bacterial meningitis	12	10.0
	Meningoencephalitis	3	2.5
	Cerebral Abscesses	4	3.3
	Tuberculoma and tubercular meningitis	4	3.3
	TORCH infection	2	1.7
Neurovascular causes (n=12)	HIE	7	5.8
	IVH	1	0.8
	Porencephalic cyst	2	1.7
	Encephalomalacia (Left ICA insult)	2	1.7
Neoplastic Causes (n=4)	Ependymoma	1	0.8
	Medulloblastoma	1	0.8
	Colloid cyst	1	0.8
	Post op case of Recurrence/Residual of primary brain tumour in right parietal lobe	1	0.8
Causes of ex-vacuo ventriculomegaly (n=20)	Diffuse cerebral atrophy	3	2.5
	B/L Fronto-Parieto-Temporal atrophy	7	5.8
	Diffuse brain atrophy	1	0.8
	Dyke Davidoff Masson syndrome	4	3.3
	B/L frontal atrophy	1	0.0
	Encephalomalacia/Gliosis	4	3.3
Normal Variants (n= 3)	Asymmetrical Lateral Ventricles	3	2.5

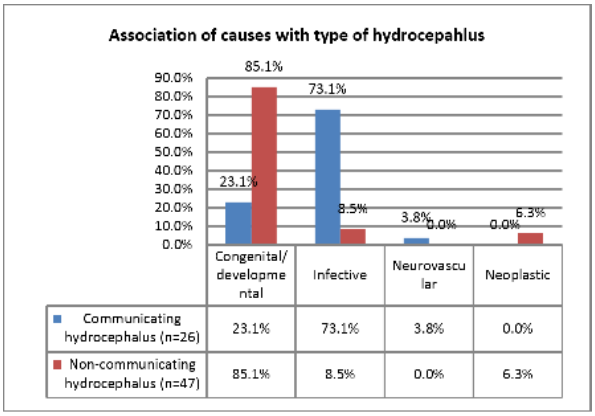
Table 3.9:Causes of hydrocephalus in study subjects (n=73)

Cause of hydrocephalus	No.	%
Congenital/developmental	46	63.0
Infective	23	31.5
Neurovascular	1	1.4
Neoplastic	3	4.1
Ex-vacuo	0	0.0
Normal variant	0	0.0

Table 3.10:Association of causes with type of hydrocephalus

Causes of Ventriculomegaly	Communicating hydrocephalus (n=26)		Non-communicating hydrocephalus (n=47)		P value
	No.	%	No.	%	
Congenital/ developmental	6	23.1	40	85.1	<0.001
Infective	19	73.1	4	8.5	<0.001

Neurovascular	1	3.8	0	0.0	0.35
Neoplastic	0	0.0	3	6.3	0.54
Ex-vacuo	0	0.0	0	0.0	-
Normal variant	0	0.0	0	0.0	-



Graph 3:Association of causes with type of hydrocephalus

DISCUSSION

Our study included 120 pediatric patients, who had ventriculomegaly on MRI. Although newer techniques and modern MRI sequences has immensely aided in the diagnosis of such patients by beautifully depicting the structures of the brain, it is still not available everywhere, especially in India. Thus, we have to rely on the basic techniques and sequences.

After thoroughly studying all the cases including the clinical, biochemical, histopathological aspects we have divided the causes of ventriculomegaly in 6 categories on the basis of the **main** etiology behind it:

1. Congenital/Developmental,
2. Infective,
3. Neurovascular,
4. Neoplastic,
5. Ex-vacuo dilatation and
6. Normal Variants.

Age:

In our study, we included newborns to children upto 18 years of age. We divided them into 5 age groups as illustrated in Table 3.1. Congenital or developmental causes of ventriculomegaly was most common in the age groups < 1months [11(78.6%)] and 1-12 months [34(65.4%)]. Neurovascular causes were the commonest in the age group 1-5 years [9 (30%)]. In the age groups 5-12 years and 12-18 years, Ex-vacuo dilatation and infectives causes predominated.

Gender:

The distribution of patients is illustrated in Table 3.2. There was no statistical significance (p value > 0.05) in the distribution of causes of ventriculomegaly amongst males and females i.e. no gender predilection for ventriculomegaly.

Socioeconomic status:

In our study, majority of patients with ventriculomegaly [81(67.5 %)] were from low socioeconomic strata followed by middle [27 (22.5 %)] and then high socioeconomic status [12 (10%)].

Need of sedation:

It was observed that most of the children did not need any sedation. Out of 120 patients, only 41 patients (34.2 %) needed sedation while in 79 cases (65.8%) there was no need of sedation.

Maternal folic acid intake during pregnancy:

It was observed that out of 120 paediatric patients in this study with ventriculomegaly, 85 patients (70.8 %) gave history of complete course of maternal folic acid intake during pregnancy whereas 35 patients (29.2%) gave history of no or incomplete intake during pregnancy. It was found that 27 (77.1%) patients with no history of folic acid intake developed MMC and out of the 85 patients with complete history of folic acid intake, only 5 patients (5.9%) developed MMC suggesting a strong association between maternal folic acid intake with prevention of neural tube defects which was statistically significant (p- value < 0.001).

Clinical features:

In our study it was observed that patients with ventriculomegaly presented with a vast array of neurological signs and symptoms viz fever, vomiting, developmental delay, mental retardation, loss of consciousness, increasing head size, seizures, headache, neck rigidity, limb paresis, cranial nerve palsies and other neurological deficits. Younger patients presented with rather vague symptoms like poor feeding, irritability, poor response to touch, excessive crying, failure to thrive, etc. Similar findings were also mentioned by NK Venkatarman et al (2011)[7].

Head circumference:

In our study, 56 (46.7%) patients had macrocephaly out of which 54 (96.42%) patients had hydrocephalus (51 of them were under 2 years of age). This data was statistically significant suggesting a positive correlation between hydrocephalus and increasing head size under 2 years of age. This commensurates with Kristopher T Kahle et al (2015)[8] who stated that infants with hydrocephalus commonly present with progressive macrocephaly whereas children older than 2 years of age generally present with other signs and symptoms of intracranial hypertension.

Hydrocephalus:

It was found that out of 120 patients with ventriculomegaly, 73 (60.8 %) patients had clinical as well as radiological evidence of hydrocephalus whereas 47 (39.2%) patients had no hydrocephalus. Out of these 73 patients with hydrocephalus, 26 (35.6%) patients had communicating hydrocephalus while 47 (64.3%) patients had non-communicating hydrocephalus.

Ophthalmological findings:

Out of 73 cases of hydrocephalus in our study, 36 (49.31%) patients had ophthalmological signs and symptoms. Overall, the commonest ophthalmological sign in hydrocephalus was papilledema seen in 24 patients (32.87 %). Poor vision was seen in 11 cases (15.06%), this commensurates with Heinsbergen et al (2002)[9]who found decreased visual acuity in 13% cases with hydrocephalus. Other findings included sunset sign, strabismus, etc. Long standing cases also showed optic atrophy.

Anatomical structures involved:

It was observed that the most common anatomical structure involved were Ventricles– {Lateral Ventricles [120 (100%)] followed by 3rd [93 (77.5%)] & 4th ventricle [54 (45%)]}, then cerebral hemispheres 61 (50.8%), followed by cerebellum 26(21.7%) and brainstem 15 (41.7%).

Spinal abnormalities:

In our study, out of 120 patients of ventriculomegaly, 32 patients (26.7 %) had spinal abnormalities. Out of these 32 patients, the commonest abnormality was Lumbosacral MMC [24 (75%)]. Our result was similar to the results of Ectstein and Macnab et al (1966)[10]who stated that 82% of meningomyeloceles are lumbar or sacral in site.

Normal vs abnormal cases:

It was observed that in our study, out of 120 patients with ventriculomegaly, only 3 patients (2.5 %) had isolated mild ventriculomegaly. Rest of the patientsi.e.117 patients (97.5 %) had some pathological cause of ventriculomegaly.

Etiology of ventriculomegaly:

The distribution of causes is listed in Table 3.8

- **Normal variants-**Out of 120 patients with ventriculomegaly on MRI, 3 cases, isolated asymmetrically mildly dilated lateral ventricles were noted & diagnosed as Asymmetrical Lateral ventricles, a normal variant.
- **Congenital and developmental** causes were noted in 56 cases (46.7%) forming the largest group. The most common congenital cause was MMC which was seen in 19 cases- 15.4% (12 cases with aqueductal stenosis, 6 cases with meningitis and 1 case of MMC alone). This was followed by Chiari Malformations in 15 cases (13 cases of Chiari II, 1 case of Chiari 1 and 1 case of Chiari 3 malformation).This result was similar to the study of **Salem-Memouet al**[11] who found that the most common cause of hydrocephalus in newborns and infants is myelomeningocele (23.8 %).
- **Infective** causes were noted in 25 patients(20.8%).The most common infective cause was found out to be Bacterial Meningitis

(12 cases).

- **Ex-vacuo dilatation** was seen in 20 cases (16.7 %). The most common cause in this group was B/L Fronto-parieto-temporal atrophy (7cases)followed by Encephalomalacia and Gliosis (4 cases) and Dyke Davidoff Masson syndrome (4 cases) which is a rare neurological condition resulting in hemiatrophy of cerebral hemispheres. Due to its rarity, it is often misdiagnosed or underreported by majority of clinicians.
- **Neurovascular causes** were noted in 12 cases (10%) with HIE (7 cases) being the commonest cause in this group. 5 cases out of 7 cases of HIE were associated with corpus callosal dysgenesis.
- **Neoplastic causes** was the least common pathological group constituting only 4 cases (3.3%).

Etiology of hydrocephalus and its association with type of hydrocephalus:

In our study it was found that out of 120 cases of ventriculomegaly, 73 patients had hydrocephalus. 26 (35.6%) patients had communicating hydrocephalus whereas 47 (64.3%) patients had non-communicating hydrocephalus]. The distribution of causes of hydrocephalus is illustrated in Table 3.9:

- Overall, congenital and developmental causes were the commonest cause of hydrocephalus, seen in 46 patients(63%).
- Out of 47 cases of non-communicating hydrocephalus, Congenital/Developmental causes were noted in 40 cases (85.1%).
- Out of 26 cases of communicating hydrocephalus, Infective causes were noted in 19 cases (i.e. 73.1%).
- It was observed that out of 23 cases of infective causes of hydrocephalus, majority of them were causing communicating hydrocephalus (82.60 %) and similarly out of 46 cases Congenital/developmental causes of hydrocephalus, majority of them were causing non-communicating hydrocephalus (86.9 %). This association was stastistically significant (p-value <0.001).

Limitations of our study:

Our study only included patients presenting to us in our institution, i.e. in the Bundelkhand region of India therefore the data extrapolated from our study may not be able to closely represent the other parts of the world.

Controversies in the results and inability to compare results could be attributed to various factors such as sample size, geographical variation, varying techniques and machines, different materials and methods, various inclusion and exclusion criteria, etc.

Advantages of our study:

Previously, we have had studies and research papers on hydrocephalus in children but there are barely any studies on ventriculomegaly in pediatric patients. Thus, our study highlights a whole new edge in this context, adding to the sparse literature.

Pictorial illustration: Our study, beautifully illustrates the imaging patterns on CT/MRI while discussing the etiology of ventriculomegaly in all the patients.

CONCLUSION:

MRI is the best radiological investigation for evaluation of ventriculomegaly. Overall, congenital and developmental causes were the most common causes of ventriculomegaly and non-communicating hydrocephalus whereas infective causes comprised majority of cases with communicating hydrocephalus. Overall, the single most common cause of ventriculomegaly was Myelomeningocele and majority of them had no or incomplete history of maternal folic acid intake.

Abbreviations: Cerebrospinal fluid -**CSF,** Myelomeningocele-**MMC,** Intracranial pressure-**ICP,** Magnetic Resonance Imaging -**MRI,** Computed Tomography-**CT,** Ultrasonography-**Usg**

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