Research Square

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Case Series Recurrent meningitis: causal management is the final goal

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Case Report

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Abstract

Abstract Recurrent meningitis indicate a host defect either in the local anatomy or in the antibacterial and immunologic defenses,Streptococcus Pneumoniae is by far the most frequent cause of recurrent meningitis, susceptible patients to have recurrent meningitis are those with defects in the dura mater after head injury or surgery,those with chronic infections in the skull e.g. chronic suppurative otitis media or sinusitis, Patients with large nasal polyps specially after polypectomy, individuals who lack spleen e.g. hemolytic diseases, after abdominal trauma or after splenectomy for lymphoma or other malignancies, the anatomic defect may produce CSF leak (rhinorrhea) which may be intermittent. Herein we describe three cases of recurrent meningitis, the purpose is to emphasize that any patient with bacterial meningitis particularly if it is recurrent should be evaluated carefully for any congenital or post traumatic defect.A structured approach and early diagnosis of any underlying pathology are crucial to prevent further episodes and improve the overall outcome for the affected individual.

Case Presentation

Case1:

Female patient 58 years old ,house wife, known diabetic ,hypertensive and chronic asthmatic ,admitted to the ICU in a deep coma state, The patient was quite well until one day before admission when she complained of headache ,fever and vomiting for which she received non- specific medications from a private dispensary, the patient didn't improve ,rather she had also been noticed to be confused few hours before losing her conscious level, no history of head trauma, injury or falling from a height. six years earlier nasal polypectomy was performed because of nasal obstruction and difficult breathing.

Past history: over the last 5 years, the patient admitted to the hospital with 2 similar episodes with a history suggestive of meningitis

There is a history of a unilateral right side feeling of clear salty fluid from the nose and in her throat.

On Examination: the patient was morbid obese with flat decubitus,in a deep coma state, GCS : Severe 7/15 E2M4V1 , Pupils :RRR , no jaundice , neck veins not congested , mild pitting edema in both lower limbs

She was vitally stable ,temperature 37.5C pulse 90/m , Blood Pressure:145/70 mm Hg RR: 22/m. PSo2 :95% . tongue : moist with no injury and no evidence of previous convulsions.

Chest : normal vesicular breathing with occasional expiratory rhonchi. Cardiovascular examination: unremarkable . Abdomen : lax, liver and spleen can not be palpated , no lymphadenopathy, joint examination : normal

Neurological examination : No signs of lateralization , no signs of cranial nerve affection Neck :rigid, Kernig's and Brudisiniki's signs are positive .

Laboratory Investigations:

Complete blood count, biochemical investigations and CSF analysis are shown in table 1.

Test	Result	Unit	Reference range
Complete blood count			
Hemoglobin	15.1	gm/dl	11-16
hematocrite	41.9	%	33-44
Red blood cells	5.200,000	U/L	$3.8\text{-}5.5x10^6$
Platlets	220 $x10^3$	U/L	$150\text{-}450X10^3$
TLC	16.000	U/l	4-11
Neutrophils	88	%	40-60
Lymphocytes	6.3	%	20-40
MCV	80	Fl	78-96
MCH	28.8	Pg	26-32
Chemistry			
Random blood sugar	345	mg/dl	74-106
Serum creatinine	0.7	mg/dl	0.7-1.3
Blood urea	29	mg/dl	17-49
AST	30	U/l	15-37
ALT	23	U/l	14-63
Alkaline phosphatase	81	U/l	44-147
Serum bilirubin	0.9	mg/dl	0.2-1
Total protein	8	gm/dl	6-8.3
Serum albumin	3.9	gm/dl	3.4-5.4
Calcium (total	9.2	mg/dl	9-11
CSF analysis			
Aspect	Purulent		Clear
Cell count	>5.000	Cu mm	0-5
Neutrophils	95	%	0
Glucose	133	mg/dl	
	5	%	

Lymphocytes	292	mg/dl	50-80
Protein	Gram positive diplococcus capsulated S. pneumoniae		0-5
Gram stain	Growth of streptococcus pneumoniae		15-40
			No organism
CSF culture			
			No organism

CSF Culture: Aerobic culture revealed growth of streptococcus pneumoniae, sensitive to penicillin, vancomycin and cefoperazone.

Non contrast CT Scan of the paranasal sinuses (figures 1,2) revealed : small bony defect at the right aspect of floor of the anterior cranial fossa (arrows) associated with cystic like opacification of the right ethmoidal air cells,mild right maxillary sinusitis.

Fig. 1, 2: Non contrast CT Scan of the paranasal sinuses revealed: small bony defect at the right aspect of floor of the anterior cranial fossa (arrows)

The patient immediately received on admission at the first dose of dexamethasone 0.15 mg /kg/dose then continued for 4 days.vancomycin and ceftriaxone are initially started as empirical treatment then replaced ceftriaxone by cefoperazon after the result of CSF culture and sensitivity, fluids and supportive treatment are given. After 2 days the patient regained consciousness, the fever subsided and discharged after 2 weeks without any neurological sequalae and referred for nasal endoscopic closure of the cranial defect .

Diagnosis : Recurrent bacterial meningitis after nasal polypectomy.

Case 2

A 9 year old girl,Medically free with Normal developmental milestones Normal vaginal delivery with no NICU admission, admitted to the ICU because of altered sensorium, generalized tonic- clonic convulsions and high grade fever, Preceded by symptoms suggestive of upper respiratory tract infection five days before admission.There is a history of episodic attacks of feeling a salty clear fluid running from the left side of the nose suggested to be CSF rhinorrhea, no history suggestive of immune suppression, head trauma, or previous nasal operation, history suggestive of previous meningitis one and half years before admission

Laboratory investigations

Complete blood count, biochemical investigations and CSF analysis are shown in table 2.

Test	Result	Unit	Reference range
Complete blood count			
Hemoglobin	9.3	gm/dl	11-16
TLC	18.900	U/L	4-11
Platlets	136x 10^3	U/L	150-450x 10^3
Hematocrite	30	%	33-44
Red blood cells	4.300x10^3	U/l	3.8-5.5x 10^3
Neutrophils	90	%	40-60
Lymphocytes	8	%	20-40
MCV	72	Fl	78-96
MCH	20	Pg	26-32
CSF analysis			
At time of admission			
Cells	2500	Cu mm	0-5
Neutrophils	90	%	0
Lymphocytes	10	%	0-5
Glucose	73	mg/dl	50-80
Protein	266	mg/dl	15-40
CSF analysis after two days			
Cells			
Glucose	1700	Cu mm	0-5
Protein	90	mg/d	50-80
Neutrophils	182	mg/dll	15-40
Lymphocytes	50	%	0
CSF culture	50	%	0-5
CSF analysis after one	No growth		No organism
week of admission			
Cells			
Glucose	20	Cu mm	0-5
Protein		mg/dl	

Chemistry	60	mg/dl	50-80
ALT	100		15-40
AST		U/L	
Serum creatinine	39	U/L	14-63
BUN	40	mg/dl	15-37
Serum sodium	0.24	mg/dl	0.7-1.3
Serum potassium	3	meq/l	6-24
	137	mmol/l	135-145
	4.3		3.5-5

Radiological investigations:

CT brain examination revealed the presence of 2.2 x 4.4 cm soft tissue mass having heterogenous density seen totally obliterating and expanding the left nasal cavity with related bone defect

Fig. 3: Multislice- non contrast CT examination revealed soft tissue mass 2.2x 4.4 cm (arrow) oblitrating the left nasal cavity with related bone defect at the left side of cribriform plate, the mass is rarefying the bony nasal septum which is seen deviated to the right side, features of left cribriform bony defect with fronto ethmoidal encephalocele.

MRI Brain with contrast revealed:

Ill defined defect noted at the left cribriform plate with herniation of the meninges and left frontal lobe inferior aspect into the ethmoidal air cells and the left nasal cavity suggesting meningoencephalocele

Fig. 4: MRI brain Axial T1 without contrast showing Ill defined defect noted at the left cribriform plate with herniation of the meninges and left frontal lobe inferior aspect into the ethmoidal air cells and the left nasal cavity (arrows)

Fig.5,6: MRI Brain Axial T2 and FLAIR showing Ill defined defect noted at the left cribriform plate (arrows,)

ENT consultation done Recommended surgical repair after resolution of infection

Upon admission the patient immediately received vancomycin, ceftriaxone, dexamethazone and IV fluids, after two days, the fever subsided, the general condition improved, and she regained consciousness and discharged after 10 days without sequlae for surgical repair of the dural defect.

Diagnosis: recurrent bacterial meningitis due to defect in the cribriform plate of ethmoid

Case 3

A young female child 1year and 3 months presented to the emergency department with high fever and repeated convulsions .

The condition started 4 days before admission when she suffered frequent attacks of vomiting associated with fever and loose stools for which she has been managed by a private dispensary with antibiotics , anti-emetics , and IV fluids for presumed diagnosis of gastroenteritis Over the next 2 days the patient general state worsened more fever spiked again she passed into coma , and she convulsed 3 times before admission to the hospital .

Eight months earlier she exposed to car accident with her mother and open laparotomy revealed traumatic injury of the spleen and she was subjected to urgent splenectomy.There is a history of previous transfusion related to the operation, She received only the compulsory vaccinations schedule, history of similar attack three months after the operation she was diagnosed and treated as bacterial meningitis,The family history was not contributory , normal obstetric history , normal vaginal delivery with no fetal or maternal complications .

CNS infection was suspected and Lumbar puncture was immediately performed after stabilization of the patient .

On examination : the patient was in a deep coma state , looks toxic , with no jaundice or cyanosis , no visible signs of cranial trauma , no visible skin rash , temperature : 39c , pulse : 110/m , RR: 28/m ,no pedal oedema .

pupils : Round ,Regular,Reactive (RRR) , GCS : severe 7/15 (E2V2M3) , neck: rigid
Chest :bilateral clear air entry with normal vesicular breathing

Heart : normal S1,S2 Abdomen : soft , visible transverse scar of splenectomy about 8 cm healed by primary intention(Fig. 7), liver was not palpable .
There was normal joint examination with no lymphadenopathy
Initial investigations : CSF analysis : Aspect : hazy , Cells : 3500/cc ,

N: 70% L. : 30%
Glucose: 1mg/dl Protein : 113mg/dl , No pathogenic organism detected by gram stain and no acid fast bacilli by Zeil Nelsen stain .

CSF and blood cultures revealed no growth
CBC : WBCs : 31,000 , RBCs :3.88 , Hct: 28.7 , MCV:74 fl , MCH: 22.7 Pg , Platlets: 1.222,000/ul , Ly: 53.3 M.: 5.3 Gr.: 42.4
Normal serum electrolytes , normal renal and liver profile .

RBS: 88mg/dl , PT: 10.8
Hepatitis markers : negative .

Test	Result	Unit	Reference range
Complete blood count			
WBCs	31.000	U/L	4-11.000
RBCs	3.880x 10^3	U/L	3.8-5.5X 10^3
Hematocrite	28.7	%	33-44
MCV	74	Fl	78-96
MCH	22.7	Pg	26-32
Platlets	1.222 x 10^3	Ul	4-11 x 10^3
Lymphocytes	53.3	%	20-40
Granulocytes	42.4	%	40-75
Monocytes	5.3	%	1-10
Chemistry			
Random blood sugar	88	mg/dl	74-106
Serum sodium	138	Meq/l	135-145
Serum potassium	3.7	mmol/l	3.5-5
Ionized calcium	1.1	mmol/l	1.2-1.4
ALT	23	U/L	14-63
AST	28	U/L	15-37
Serum urea	37	mg/dl	17-49
Serum creatinine	0.8	mg/dl	0.7-1.3
CSF analysis			
Cells	3500	Cu mm	0-5
Aspect	Hazy		Clear
Glucose	1	mg/dl	50-80
Protein	113	mg/dl	15-40
Gram stain	No pathogen		No pathogen
Zeil Nelsen stain	No acid fast bacilli		No acid fast bacilli

Brain CT examination : normal study.(Fig.8)

The patient admitted to the ICU received dexamethazone 0.15mg/kg /day for 4 days , vancomycin and ceftriaxone, phenytoin as anticonvulsant and iv fluids in addition to baby aspirin for thrombocytosis.

Follow up of the patient was excellent, fever settled,no more convulsions, she regained consciousness and discharged without sequlae and the parents advised for her vaccination with meningococcal, pneumococcal and hemophilus influenza vaccines.

Diagnosis : Septic meningitis as overwhelming post splenectomy infection syndrome (OPSI syndrome)

Discussion

Recurrent meningitis indicate a host defect either in local anatomy or in the antibacterial and immunologic defenses (e.g recurrent N. Meningitides infections in patients with congenital or acquired deficiencies of complement (10) S. pneumonia is by far the most frequent cause of recurrent meningitis. (2). Recurrent meningitis is estimated to affect between 2–9% of sufferers [3, 4] and can be defined as the occurrence of two or more episodes of meningitis triggered by different organisms, or as multiple episodes caused by the same pathogen after adequate treatment [5]. Recurrent meningitis happens mostly in patients with acquired or congenital anatomic defects, but also in patients with ear or sinus infections, CSF leakage, or some immunodeficiency. The main causative pathogen is *Streptococcus pneumoniae*.(6)

A history of head trauma is much more frequent in patients with recurrent meningitis .(7)

The risk of a recurrence of bacterial meningitis in patients with no anatomical defect or known immunodeficiency is estimated to be 1-3%.(8)

Susceptible patients to have pneumococcal meningitis :(9) Those with defects in the dura mater after head injury or surgery,those with chronic infections in the skull e.g chronic suppurative otitis media, sinusitis,or mastoiditis patients with large nasal polyps especially after polypectomy, Individuals who lack a spleen e.g hemolytic diseases, after abdominal trauma, or after splenectomy for lymphoma or other malignancies. Organisms may directly enter the subarachnoid space through :(1)Defect in the cribriform plate of ethmoid

(the most common site) (2) In association with empty sella syndrome

(3)Via a basilar skull fracture .

(4) Through erosive sequestrum of the mastoid .

(5) Through a congenital dermal defect along the cerebero-spinal axis (usually evident before adult life) .

(6) As a consequence of penetrating cranial trauma or neurosurgical procedures .(9)

The anatomic defect may produce a frank CSF leak (rhinorrhea or less commonly otorrhea).(12)

CSF rhinorrhea may be intermittent.(13)

Meningitis may occur months or years after head injury.

The presence of CSF rhinorrhea should be considered at admission .

Rhinorrhea may clear during active meningitis only to recur when inflammation has resolved .

Clinical clues suggesting The presence of CSF fistula through the cribriform plate , pericranial air sinuses or temporal bone include :

(1 Salty taste in the throat .

(2) positionally dependent rhinorrhea (rhinorrhea only on lateral recumbent or prone position suggests an otic or sphenoid origin).

(3) Anosmia (cribriform plate leak)(9)

High definition coronal computed tomography of the anterior skull base .

If this fails to localize the site of a defect :

A T2 weighted magnetic resonance image may help .

If the site of the leak remain unclear or suspicion that there more than one defect in the skull base . Under these circumstances (10)

A diagnostic or peroperative lumbar puncture using fluorescein dye will help to localize the area .(10)

Closure of the CSF fistula should be carried out to prevent further episodes of meningitis .(11)

The prophylactic use of antibiotics in patients with CSF leaks appears to be ineffective and can not be recommended .(12)

Persistent rhinorrhea for more than 4-6 weeks is an indication for surgical repair

Operative management is reserved for traumatic CSF leaks .(13)

Endoscopic techniques have improved the results associated with extracranial repair of CSF leaks with success rate as high as 90% .

Under direct endoscopic visualization , a leak often can be identified and a sinus into which it drains can be obliterated by packing it with fat or muscle (14).

Splenectomized patients are likely to suffer from severe infections such as sepsis and meningitis which is called overwhelming post splenectomy infection (OPSI) .(15)

It seems to be more common in children but can occur at all ages , the risk is greatest in the early months and years after operation , the course is rapid ,the clinical symptoms are serious and the prognosis is poor . (15)

splenectomized patients are susceptible to infection by the polysaccharide encapsulated bacteria which are cleared by the spleen Pneumococcal infections 50- 90% of cases reported , Haemophilus influenza type B , Neisseria meningitides and group A streptococci (25% of infections) .(16)

The occurrence of this disease is reduced thanks to the use of vaccines based on polysaccharides conjugated to proteins against the main bacteria causing meningitis: *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae*.(16) These are highly protective but are costly and it has been observed that there is limited coverage of the serotypes or serogroups present in the composition of the vaccine [17]

Pneumococcal vaccine , H,influenza (Hib) vaccine and meningococcal vaccine are required at least 2 weeks before planned splenectomy , if the operation is unplanned like this patient then these should be as soon as possible afterwards,(18) antibiotic prophylaxis and seeking medical attention at the earliest sign of minor infection , Vaccination failure can be considered in sporadic cases like our patient , Most authorities recommend antibiotic prophylaxis for asplenic children especially for the first 2 years after splenectomy . The role of aspirin in reactive thrombocytosis is controversial .(19)

Awareness of risk factors for meningitis may facilitate early recognition and treatment of the disease, and vaccinations can prevent cases of bacterial meningitis in various risk groups. Sufficient preventive measures in selected groups of individuals with increased risk for bacterial meningitis may reduce the number of cases (20)

Conclusion

Any patient with bacterial meningitis particularly if it is recurrent should be evaluated carefully for any congenital or post traumatic defect .

Closure of the CSF fistula should be carried out to prevent further episodes of meningitis

Endoscopic techniques have improved the results associated with extracranial repair of CSF leaks. Splenectomized patients are likely to suffer from severe infections such as sepsis and meningitis which is called overwhelming post splenectomy infection (OPSI) .The occurrence of this disease is reduced after the use of vaccines based on polysaccharides conjugated to proteins against the main bacteria causing meningitis: *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae*.

Abbreviations

CSF
Cerebro Spinal Fluid

GCS
Glasgow Coma Scale
TLC
Total Leucocytic Count
AST
Aspartate aminotransferase
ALT
Alanine aminotransferase
BUN
Blood urea nitrogen
CT
Computerized tomography

Declarations

The participant consented to publish the study.

Author Contribution

HI wrote the manuscript, SZ reviewed the information, AE and MA prepared figures, Allauthors took part in the realization and implentation of this work, all authors read and approved the final manuscript.

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Figures



Figure 1

1, 2: Non contrast CT Scan of the paranasal sinuses revealed: small bony defect at the right aspect of floor of the anterior cranial fossa (arrows)



Figure 2

1, 2: Non contrast CT Scan of the paranasal sinuses revealed: small bony defect at the right aspect of floor of the anterior cranial fossa (arrows)

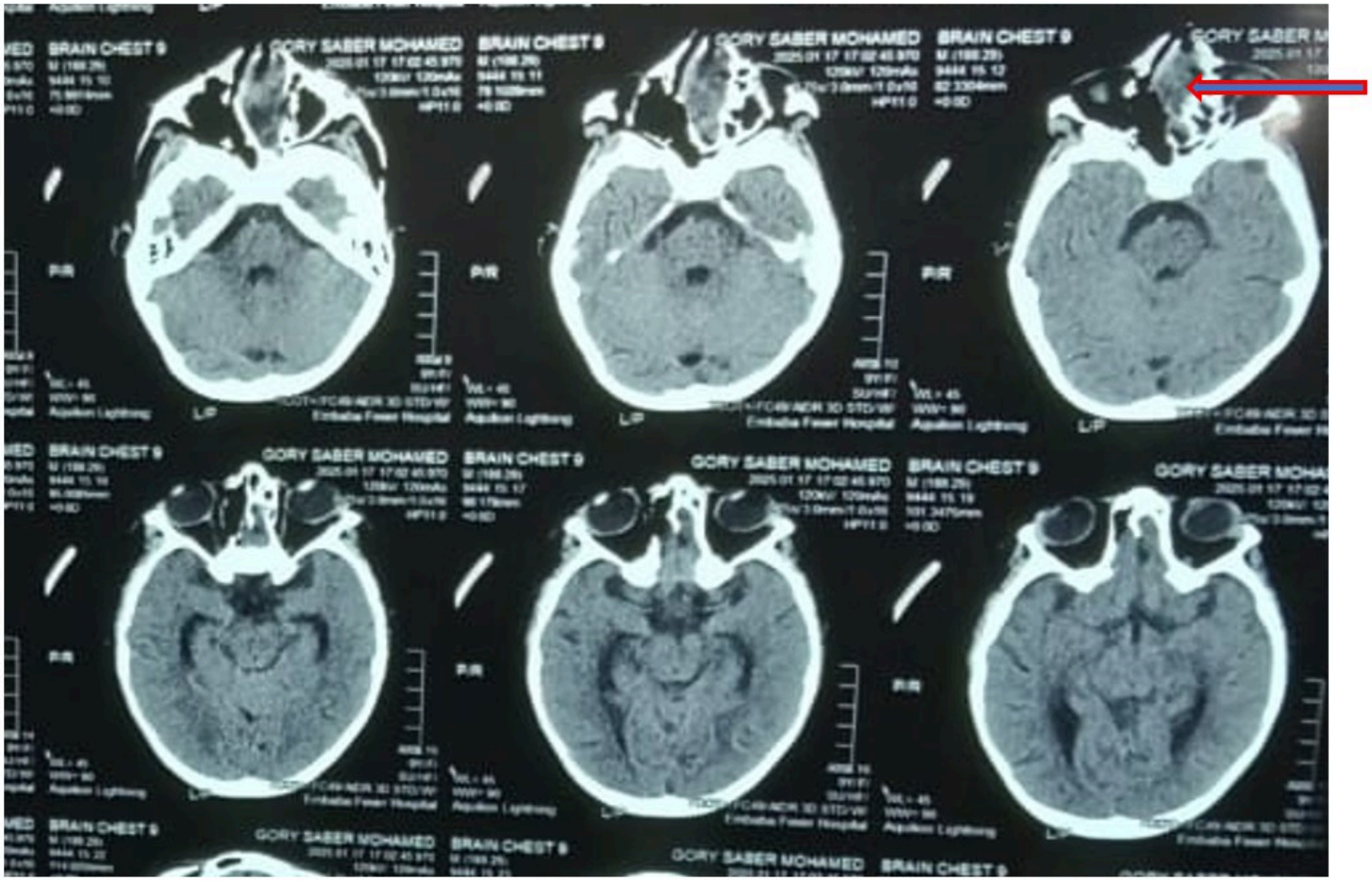


Figure 3

Multislice- non contrast CT examination revealed soft tissue mass 2.2x 4.4 cm (arrow) oblitrating the left nasal cavity with related bone defect at the left side of cribriform plate, the mass is rarefying the bony nasal septum which is seen deviated to the right side, features of left cribriform bony defect with fronto ethmoidal encephalocele.

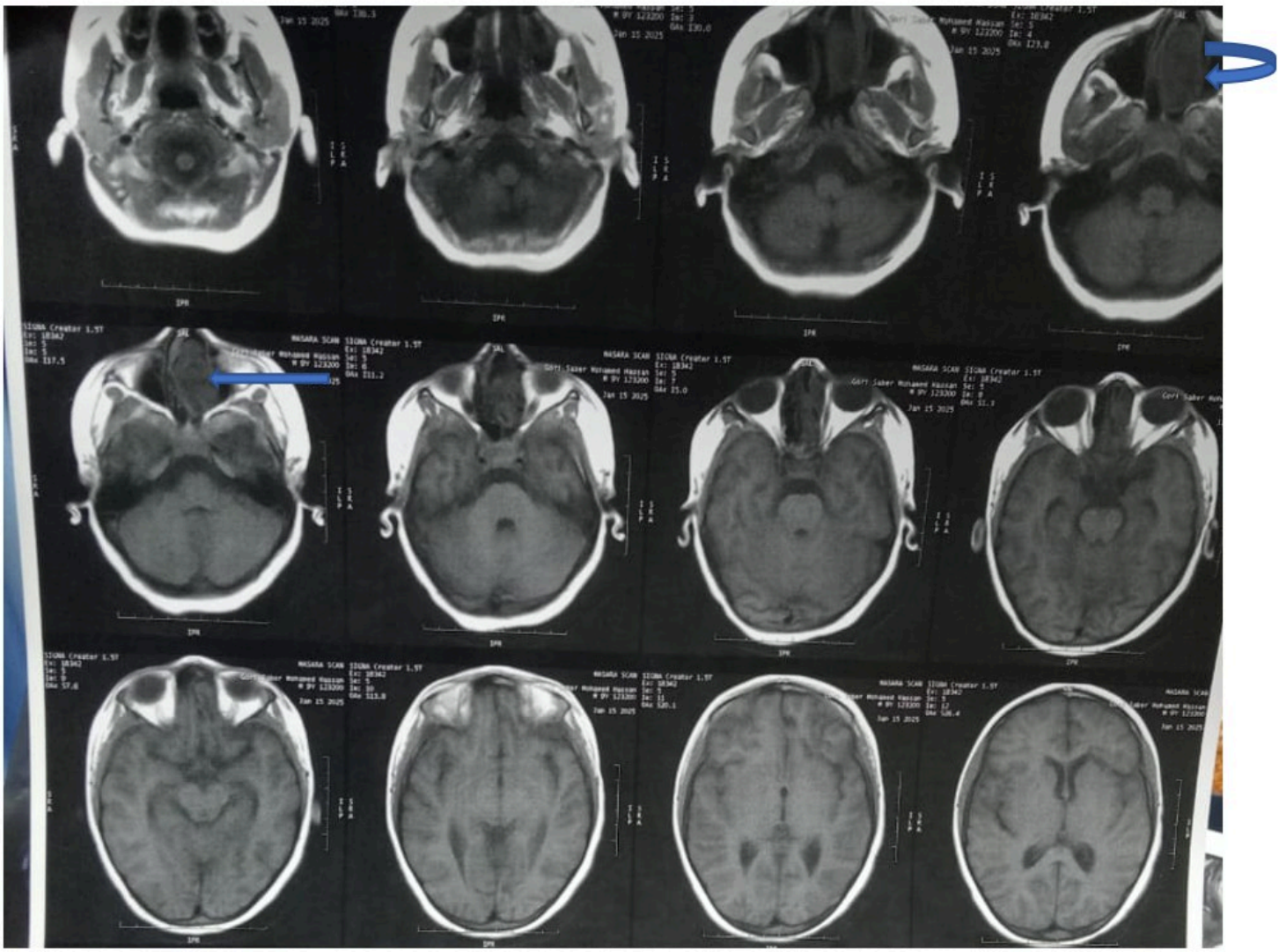


Figure 4

MRI brain Axial T1 without contrast showing Ill defined defect noted at the left cribriform plate with herniation of the meninges and left frontal lobe inferior aspect into the ethmoidal air cells and the left nasal cavity (arrows)

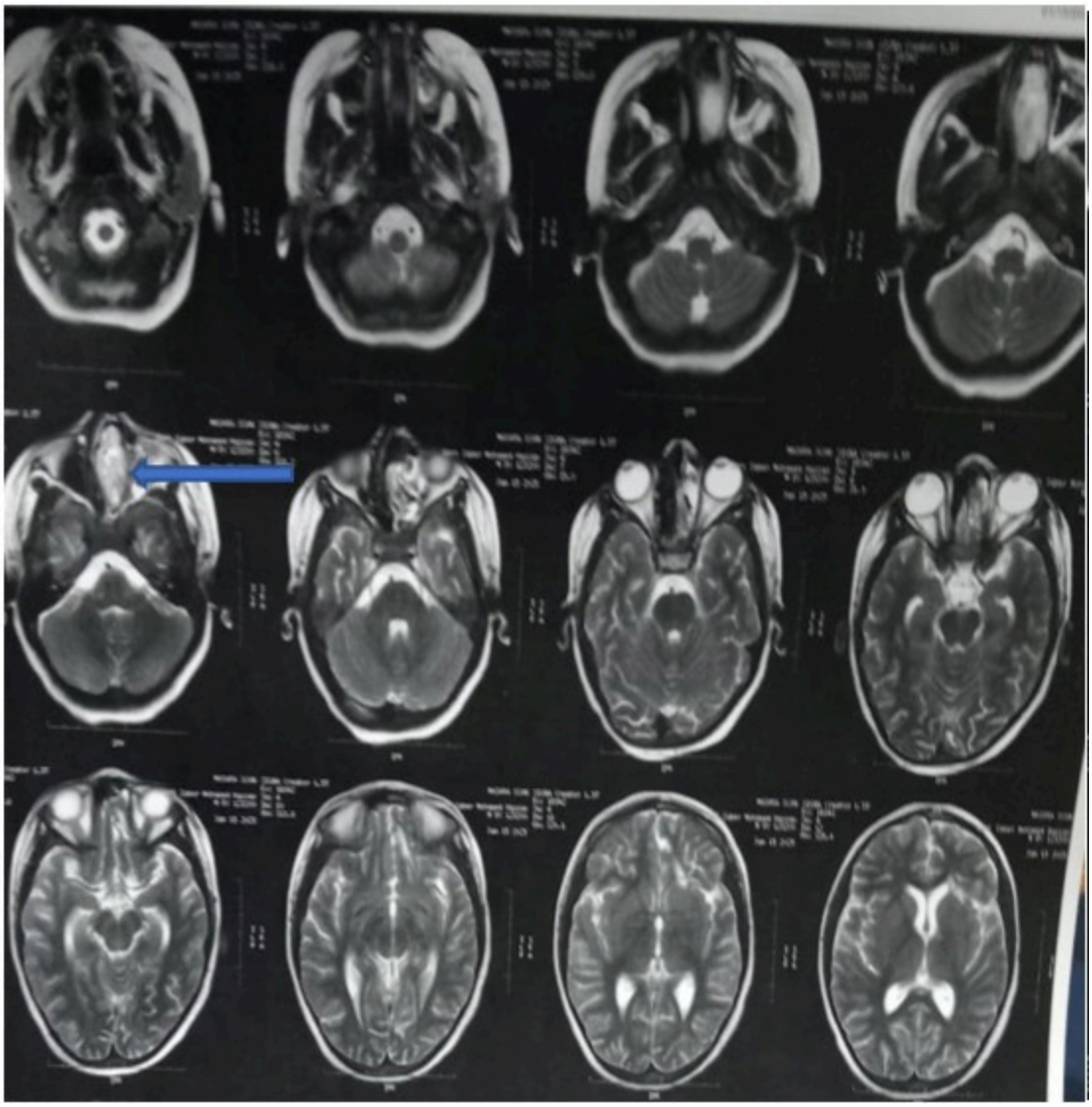


Figure 5

5,6: MRI Brain Axial T2 and FLAIR showing Ill defined defect noted at the left cribriform plate (arrows,)

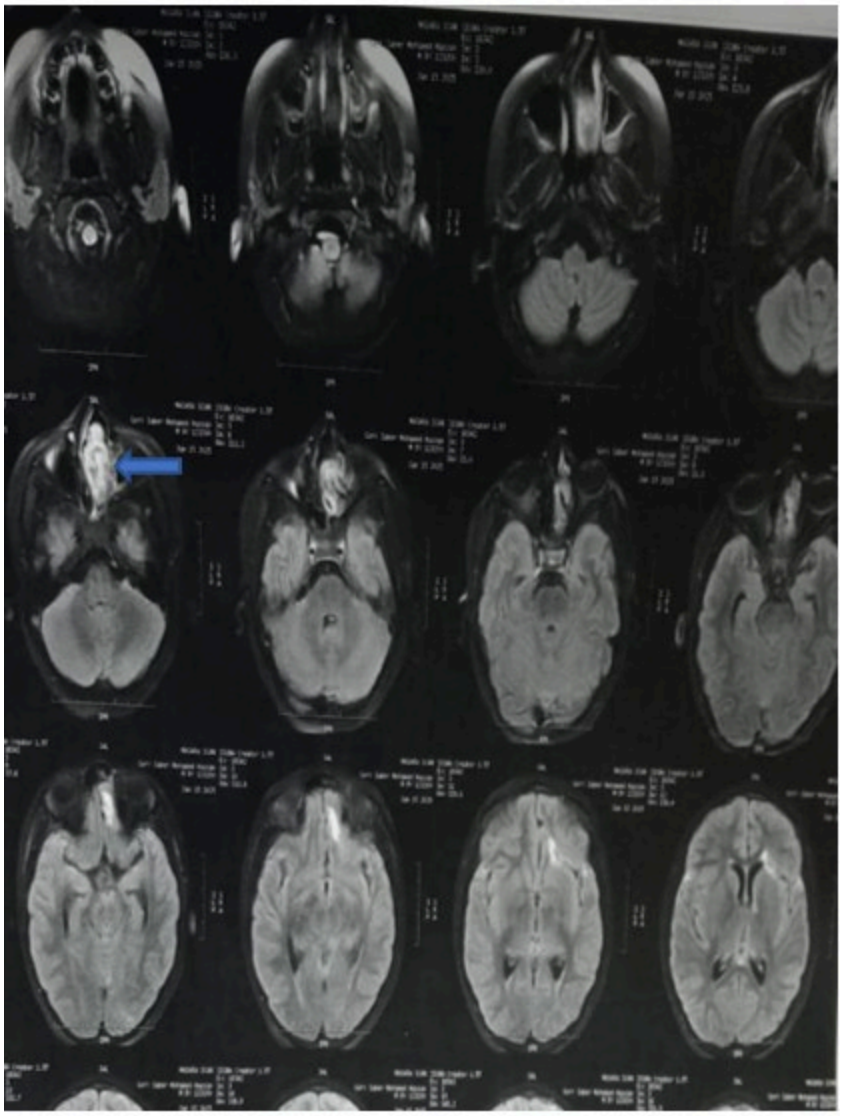


Figure 6

5,6: MRI Brain Axial T2 and FLAIR showing Ill defined defect noted at the left cribriform plate (arrows,)



Figure 7

visible scar of splenectomy

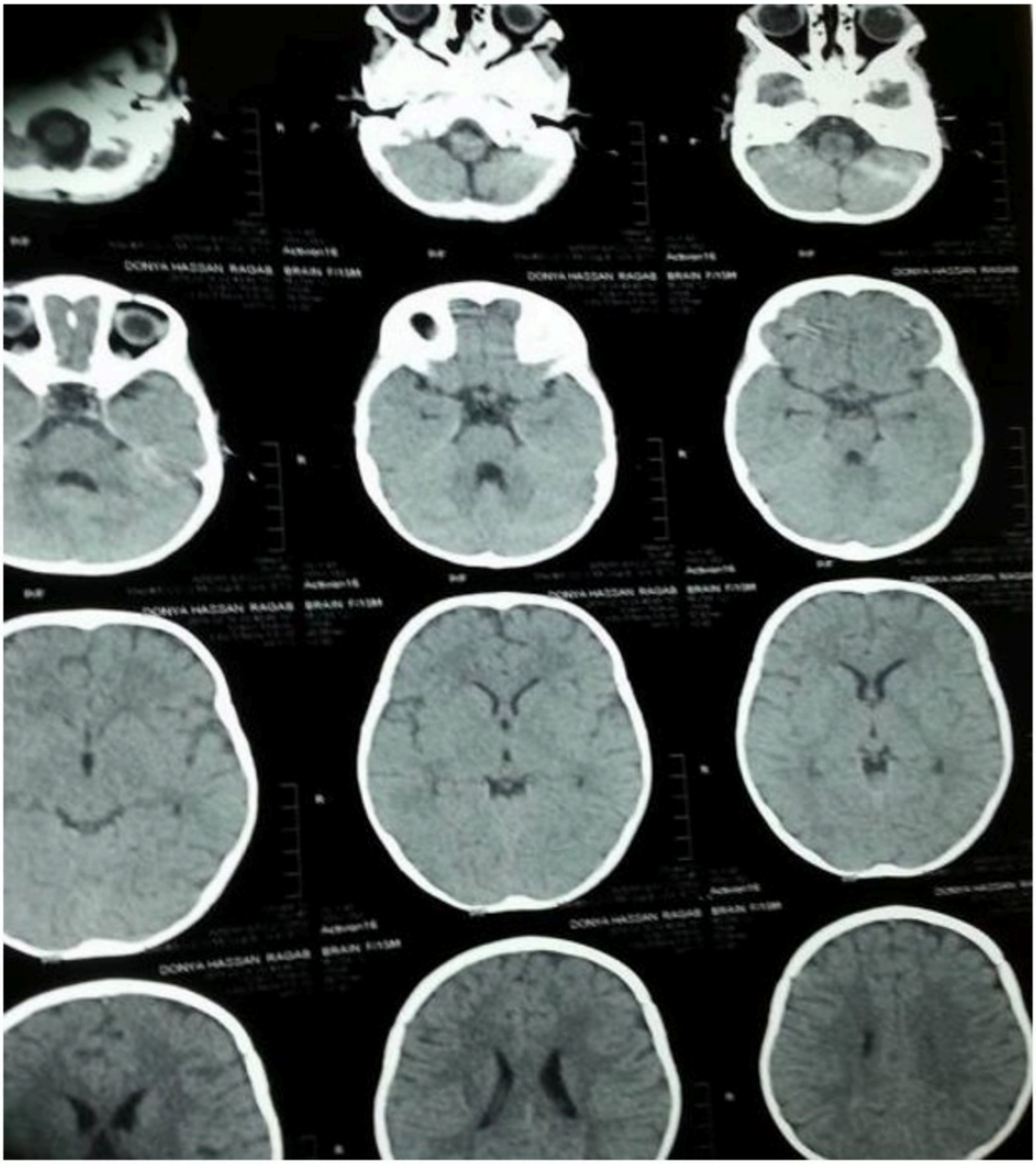


Figure 8

normal CT brain examination