Original Article

Discontinuation Of Levothyroxine In Primary Hypothyroidism And Intracranial Hypertension – Case Series :

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Abstract

Idiopathic intracranial hypertension (IIH) is persistent increase in intracranial pressure without any intracranial lesions .IIH is associated with many conditions eg.SLE, hypothyroidism ,hyperthyroidism , hypoparathyroidism etc. Eleven cases of IIH secondary to levothyroxine initiation have been reported so far .We report a case series of 3 patients who were known cases of primary hypothyroidism and were on high dose (> 100 microgram) levothyroxine therapy ,they discontinued the drug for few weeks and after reinitiation of levothyroxine developed signs and symptoms of IIH , no other causes for intracranial hypertension were found and they were treated successfully .

Keywords : Idiopathic intracranial hypertension , Systemic lupus erythematosus

INTRODUCTION :

Idiopathic intracranial hypertension (IIH) is characterized by increased intracranial pressure of unknown etiology (not due to a secondary cause) with normal cerebrospinal fluid (CSF) composition. Its annual incidence is approximately 3/100,000 persons and usually seen in young obese women [1,2] . The major morbidities are headache and vision loss if the disease is not diagnosed or there is a delay in treatment [3]. Initiation of thyroxine in newly diagnosed hypothyroidism itself can cause IIH , proposed mechanisms include decreased ability to excrete free water and alteration in CSF dynamics .Also certain acquired/ secondary causes of intracranial hypertension need to be ruled out , which are Obstruction to venous drainage (eg. Cerebral venous sinus thrombosis ,Jugular vein thrombosis ,Superior vena cava syndrome etc) ; Medications (eg. Fluoroquinolones , Tetracycline class antibiotics ,Corticosteroid withdrawal , Danazol ,Vitamin A derivatives (including isotretinoin and alltransretinoic acid) , Levothyroxine etc); Systemic disorders (eg. Chronic kidney disease/renal failure ,Obstructive sleep apnoea syndrome ,Chronic obstructive pulmonary disease , Systemic lupus erythematosus); Endocrine (eg. Addison’s disease ,Adrenal insufficiency , Cushing’s syndrome ,Hypoparathyroidism ,Hypothyroidism) [4,5] .

MATERIALS AND METHODS :

We retrospectively analysed 3 patients of idiopathic intracranial hypertension diagnosed at Dr. D Y Patil Medical College, Pune a tertiary care hospital . We assessed detailed clinical history of the patients in terms of presenting complaints, past comorbidities, vital parameters, systemic and neurological examination . Results of routine blood investigations and any special blood tests, specific to each case, were reviewed. All patients underwent initial MRI brain venography ,fundus examination for papilledema, detailed evaluation from ophthalmologist along with OCT and perimetry,CSF manometry and CSF analysis . Management plans, and the outcomes in terms of functional recovery and neurological sequelae were noted .

RESULTS :

Case-1 :

24 year old female known case of primary hypothyroidism since 7 years on tab.thyronorm 150 microgram once a day . She Presented with holocephalic headache ,binocular diplopia , transient visual obscurations ,multiple episodes of

vomiting since 1 month. She had discontinued her medications (thyronorm) for period of 2 months and reinitiated at same dose of 150 microgram 15 days before onset of headache . Examination revealed BMI- 23.4 kg/m2 , visual acquity of 5 feet bilaterally, bilateral established papilledema on fundus examination , right abducens palsy . Investigations revealed normal CBC ,ESR , iron studies , thyroid function tests revealed slightly elevated T4 and normal TSH levels . ANA –IFA negative ,serum cortisol was normal .OCT perimetry revealed bilateral papilledema. MRI brain venography was normal . CSF manometry revealed a very high opening pressure of 41 cm thus, 30 ml CSF drained . CSF revealed normal cells,proteins and glucose .Patient was managed conservatively with acetazolamide and topiramate , therapeutic CSF tap was done once and thyroxine dose was reduced to 100 microgram .Her vision and right abducens palsy improved .On follow up after 4 months she is doing well with a normal vision and resolved disk edema

Case -2 :

35 year old female known case of primary hypothyroidism, polycystic ovarian disease , obesity on tab.thyronorm (Thyroxine) 100 microgram once a day ,presented with headache ,tinnitus and transient visual obscurations since 1 ½ months not relieved by medications . She was not taking thyroxine regularly since past 2 months before onset of headache ,but taking regularly at dose of 100 microgram since last 1 month . Examination revealed BMI – 31.5 kg/m2 , visual acquity of 4 feet bilaterally ,fundus examination revealed bilateral early papilledema .Thyroid function tests revealed raised serum TSH – 11.25 IU .MRI brain venography was was within normal limits .Hemogram, ANA-IFA was normal . CSF routine studies were normal and manometry revealed a high opening pressure of 35 cm thus , 25 ml CSF was drained following which her headache symptoms were relieved and vision improved .She was managed conservatively with acetazolamide and topiramate and dose of thyroxine was reduced to 75 microgram, her vision gradually improved over 2 months and she has a normal vision on follow up over past 3 months

Case -3 :

36 year old female , known case of type-2 diabetes mellitus , hypothyroidism was initially on L-thyroxine 125 microgram once a day presented with throbbing holocephalic headache, blurring of vision since 2 months .And had no any past history of headache .She was not taking her L-thyroxine supplements since 25 days and she reinitiated her L-thyroxine at 150 microgram dose . 1 month after reinititation of L-thyroxine,her headache started . Examination revealed BMI- 25.9 kg/m2 ,fundus examination showed bilateral early papilledema .CSF routine microscopy was normal and manometry revealed high opening pressure of 36 cm, thus 20 ml CSF was drained and closing pressure was 21 cm. Serum TSH was normal .She improved symptomatically and was managed with topiramate , acetazolamide and dose of thyronorm was reduced by 25 microgram.

DISCUSSION :

IIH developing after starting levothyroxine therapy has been reported by several authors [6]. All our patients were on high dose thyroxine ,had history of discontinuation of thyroxine therapy and all symptoms of IIH developed after reinitiation of thyroxine therapy . Thus female gender ,high dose thyroxine and sudden discontinuation followed by reinitiation at high dose can contribute for development of IIH .How thyroxine supplements cause IIH mechanism is unclear , presumed mechanism include decreased ability to excrete free water in newly diagnosed hypothyroidism ,but our patients were known cases of hypothyroidism .Thus reinitiation of thyroxine at a lower dose after discontinuation ,monitoring for headache needs to be done to diagnose IIH at an early stage , to prevent visual loss .

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Images :

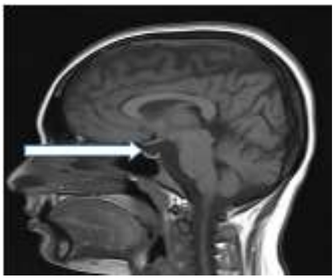

Figure -1

Figure -1 : Saggital T1 Section Of First Patient Showing Partial Empty Sella

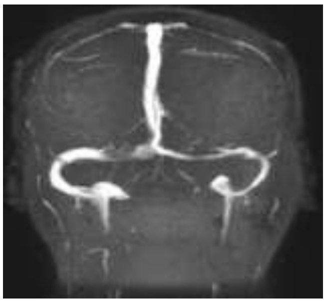

Figure-2

Figure 2 Showing A Normal MR Venography Of First Patient

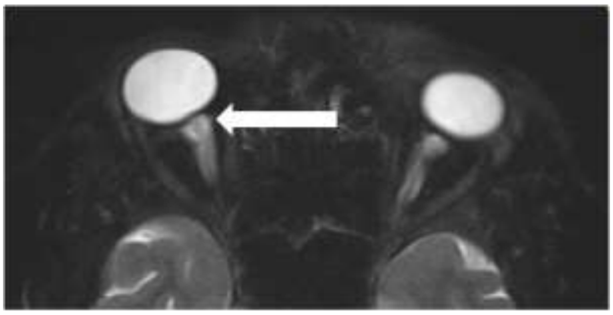

Figure-3 : Axial T2 Section Showing Flattening Of Optic Disc Of Third Patient

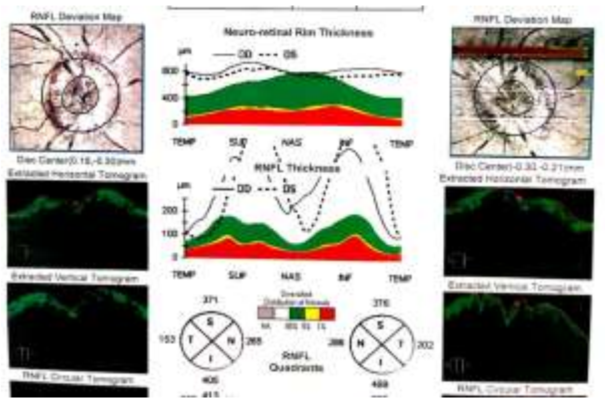

Figure-4 :OCT showing bilateral papilledema of second patient