An unmarried 21 year old female was brought to a tertiary care hospital due to some mental illness since last 2 years with complaints of gradual onset of withdrawn behavior, poor communication, maintaining one posture for hours and mutism. History of suspiciousness towards parents and referential ideas was present during initial 1 year. Then she stopped going to the school and decreased her social activities. Gradually she developed irritability, started beating family members. She used to throw utensils, shout loudly, and beat her sister without reason. Relatives were afraid of her and started keeping her in a close room where she used to remain asleep almost whole day, not taking self-care. Mother also noticed that sometimes she used to laugh without any reason. She started avoiding any communication with family members. She did not take food prepared by mother and had suspiciousness. Once she tried to burn herself by kerosene but fortunately mother saved her. This behavior lasted for 2 years. Finally, she was taken to psychiatry department. On detailed evaluation it was found that patient had been detected as having hypothyroidism 5 years back but did not receive any treatment for that. She was relatively well before 6 years, studied up to high school with good performance in school and had good pre morbid functioning. She was taking interest in day to day activities and social functions. She developed edema over legs and facial puffiness 5 years before but was not investigated. She was investigated after 1 year and found to have raised serum TSH levels 200microgram. She was given 50 microgram of thyroid for 2 months but there was no improvement in her clinical profile. It was decided by experts to add antidepressant and antipsychotic medicines. Patient had marked improvement. She started talking with family, her aggression subsided. Only persecutory delusion and memory impairment persisted so doses of antipsychotic medicine was gradually built up. She showed marked improvement like she started taking self-care, and her affect improved. She was given discharge on request by relatives with marked improvement in psychosis though her thyroid profile was still not normal.

**Abstract**

Hypothyroidism is one of the most important causes of organic psychosis and sometimes can be missed. There are some cases showing extreme degree of hypothyroidism leading to psychiatric illnesses like mania, depression and psychosis.

In present case, a 21year old well-functioning female developed hypothyroidism initially with symptoms of Myxedema which was undiagnosed for 2 years. She had psychotic features after 2 years in form of hallucinatory behavior, poor self-care, persecutory and referential delusions, episodes of violent behavior and suicidal attempts. Thyroid supplementation was given in therapeutic dose but there was no improvement in her clinical profile. It was decided by experts to add antidepressant and antipsychotic medicines. Patient had marked improvement. She started talking with family, her aggression subsided. Only persecutory delusion and memory impairment persisted so doses of antipsychotic medicine was gradually built up. She showed marked improvement like she started taking self-care, and her affect improved. She was given discharge on request by relatives with marked improvement in psychosis though her thyroid profile was still not normal.

**Keywords:** Hypothyroidism; Myxedema Madness; Psychosis

**Case report**

An unmarried 21 year old female was brought to a tertiary care hospital due to some mental illness since last 2 years with complaints of gradual onset of withdrawn behavior, poor communication, maintaining one posture for hours and mutism. History of suspiciousness towards parents and referential ideas was present during initial 1 year. Then she stopped going to the school and decreased her social activities. Gradually she developed irritability, started beating family members. She used to throw utensils, shout loudly, and beat her sister without reason. Relatives were afraid of her and started keeping her in a close room where she used to remain asleep almost whole day, not taking self-care. Mother also noticed that sometimes she used to laugh without any reason. She started avoiding any communication with family members. She did not take food prepared by mother and had suspiciousness. Once she tried to burn herself by kerosene but fortunately mother saved her. This behavior lasted for 2 years. Finally, she was taken to psychiatry department. On detailed evaluation it was found that patient had been detected as having hypothyroidism 5 years back but did not receive any treatment for that. She was relatively well before 6 years, studied up to high school with good performance in school and had good pre morbid functioning. She was taking interest in day to day activities and social functions. She developed edema over legs and facial puffiness 5 years before but was not investigated. She was investigated after 1 year and found to have raised serum TSH levels 200microgram. She was given 50 microgram of thyroid for 2 months but there was no improvement in edema, so she stopped taking medicines. Since last 2 years she started behaving abnormally like not taking self-care, inappropriate laughing, and episodes of aggressive behavior. On admission she was completely mute with no speech at all, marked psychomotor retardation, blunt affect. She remained withdrawn, rarely initiate eye to eye contact, occasionally became aggressive, screamed and had sleep disturbance. Features of severe hypothyroidism like facial puffiness and non pitting leg edema were present in her investigations (Table 1).

<table>
<thead>
<tr>
<th>Name of investigation</th>
<th>Report(on admission)</th>
<th>Normal range</th>
<th>Report(on discharge)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>9.4gm%(low)</td>
<td>11-15</td>
<td>11gm%</td>
</tr>
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<td>Serum TSH</td>
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<td>50microunit/ml</td>
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<td>Total T3</td>
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<td>1.30-3.10</td>
<td>1.2nmol/L</td>
</tr>
<tr>
<td>Total T4</td>
<td>28.39nmol/L(low)</td>
<td>66-181</td>
<td>60nmol/L</td>
</tr>
</tbody>
</table>

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Patient was given 100 microgram thyroxin/day but there was not much improvement till 20 days in her psychotic symptoms. Although TSH level came to 100 microunit/ml.

Her behavior was full of suspiciousness towards parents, irritable and withdrawn. She was put on Escitalopram 10 mg and Risperidone 2 mg which was gradually built up to 10 mg and 8 mg respectively along with Tablet Trihexyphenidyle 6 mg. Patient started showing improvement. she started talking, taking self care, Her edema and puffiness also decreased with weight loss of 3 kg in 1 month Though there was impairment in form of impaired attention and concentration, and poor recent memory, she was well and given discharge on request by relatives with serum TSH level 50 microunit/ml

### Review of literature

Hypothyroidism can present a wide range of psychiatric manifestations, including personality disturbance, neurotic traits and psychotic features. Diagnostic treatment techniques without recognition and correction of the endocrine root of the mental disturbance will result in a failure of treatment. Studies have shown, however, that 5% to 15% of Myxedematous patients have some form of psychosis [1]. Another study described the classic manifestations of hypothyroid-induced psychosis [2]. Although study of 14 patients and resulting description of Myxedema madness has been often cited as a typical example of psychosis secondary to hypothyroidism, subsequent case reports have revealed considerable variation in clinical psychotic presentation [3]. The association between thyroid deficiency and psychiatric presentation is not infrequent and is commonly overlooked as an etiology for behavioral, affective, and cognitive changes.

Many symptoms of psychological dysfunction have been described with hypothyroidism. Those symptoms most commonly related to thyroid deficiency include forgetfulness, fatigue, mental slowness, inattention, and emotional lability. The predominant affective disorder experienced is depression. Perceptual changes may develop with alterations of taste, hearing, and vision. Delusions and hallucinations may also occur as the disease progresses. No correlation, however, appears to exist between the degree of thyroid dysfunction and psychiatric symptoms that subsequently develop [4]. The prevalence of neuropsychiatric sequel of thyroid deficiency is related to the fact that most hormones present in the human body are represented in the central nervous system. The brain appears to have a unique sensitivity to thyroid hormone and to utilize thyroid hormone differently than other organ systems [5]. As many as 15% of patients can exhibit psychosis with hypothyroidism. The clinical presentation of psychosis is not uniform, and no specific group of findings is typical. Hallucinations have frequently been reported. While most studies show a slow reversal of psychosis (within weeks or months), rapid improvement (within one week) has rarely been described [2,6]. The addition of antipsychotic medications may lead to earlier remission of psychotic symptoms than thyroid replacement alone. Case reports indicate that atypical antipsychotics initiated at a low dose appear to be well tolerated. Discontinuation of thyroid supplementation may lead to the return of symptoms [7]. A study has been made of the thyrotrophic hormone (T.S.H.) blood concentration, using a modified McKenzie method of assay, in normal subjects and in patients in various psychiatric states. The conclusion is drawn that the emotional stress associated with a variety of psychiatric disorders may result in an increase in the serum level of T.S.H [8].

### Discussion

Hypothyroidism can present with a variety of physical signs and symptoms, mostly related to slowing of the metabolic process secondary to lack of effects of thyroid hormone. Fatigue, cold intolerance, slow speech, weight gain, delayed deep tendon reflexes, and bradycardia are all symptoms that result from a slowing of metabolic processes. In severe disease, non-pitting edema (myxedema) can occur from infiltration of the skin with glycosaminoglycans with associated water retention Mental status examination of a hypothyroid patient may reveal a broad spectrum of dysfunction, ranging from mild attentional impairment to significant agitated delirium or psychosis. One of the consequences of chronic hypothyroidism is what is now termed ‘Myxedematous psychosis’ and it includes progressive dementia, delirium, hallucinations and delusions [2].

It was observed that delay in effective hypothyroid treatment may result in symptoms that fail to remit completely. Patients with hypothyroidism and affective disturbance should be treated first with thyroid hormone replacement. If, after euthyroid state is established, the patient continues to display a mood disturbance, the appropriate antidepressant or mood stabilizer should be started. If psychiatric medications are utilized in patients with hypothyroidism, low starting doses and gradual titration are recommended by the authors. If treatment is initiated carefully, most physical and mental symptoms resolve over a brief period of time. But in our patient, even after giving thyroid supplementation, there was no improvement. So we started psychotropic medications and patient showed marked improvement. Psychiatric symptoms can range from subclinical to full Psychotic or Manic symptoms. No symptom cluster is indicative of Myxedema madness. Most cases are a result of severe hypothyroidism [9].

- Case reports in literature describe four possible scenarios

  1. Improvement with an antipsychotic prior to thyroid Replacement therapy

<table>
<thead>
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<tbody>
<tr>
<td>Serum Ferritin</td>
<td>28.72 mg/dl (low)</td>
<td>68-434</td>
<td>70 mg/dl</td>
</tr>
<tr>
<td>Serum TIBC</td>
<td>560 mcg/dl (high)</td>
<td>250-450</td>
<td>350 mcg/dl</td>
</tr>
</tbody>
</table>

*Table 1: Investigation results of Patient*
2. Worsening with antipsychotics alone prior to thyroid Replacement therapy
3. Improvement with thyroid replacement therapy alone
4. Improvement with thyroid replacement therapy AND Concomitant antipsychotic treatment.

In present case, patient showed improvement with thyroid replacement therapy and concomitant antipsychotic treatment

References