Sir,

Wilson’s disease is an inherited disorder due to mutation in ATP7B gene on chromosome 13,\(^1\) characterized by defective copper metabolism and its subsequent deposition in various organs of the body with myriad of clinical presentations.\(^2,3\) Literature search revealed a little information regarding possible association of hypoparathyroidism with Wilson’s disease. However, in such metabolic disorder, involvement of other organs is quite possible and requires further data for exploring its association with other organ involvement. This case report demonstrates possible involvement of parathyroid gland in Wilson’s disease leading to hypoparathyroidism and subsequent hypocalcemic seizures.

A 25-year-old male was brought to the emergency after having had a seizure. Patient’s attendants witnessed him having a generalized tonic–clonic seizure during which he bit the side of his tongue. His first memory was waking in the ambulance. Patient complained of perioral numbness and paresthesias. He has had no previous seizures; alcohol intoxication or withdrawal, use of illicit drugs such as cocaine, and no head trauma. Temperature was 36.8°C,

Figure 1: Troussaeu’s sign

Figure 2: (a & b): X-Ray hand AP & Oblique view suggesting diffuse osteopenia
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blood pressure 108/78 mm Hg, pulse 66 beats per minute, respiratory rate 18 breaths per minute, and oxygen saturation 98% on room air. Patient’s height was 152 cm, body weight was 42 kg with no delay in pubertal development. Physical examination demonstrated features of tetany in form of carpopedal spasm as evident in Figure 1 and Chvostek’s sign. Ocular examination revealed lenticular cataract in left eye. His serum calcium levels were markedly decreased. Renal functions and routine urine analysis were normal. Other laboratory test results are shown in Table 1. Electrocardiogram (ECG) revealed prolonged corrected QT interval of 550 milliseconds. Radiographic studies revealed diffuse osteopenia with normal growth plates and no evidence of rickets as shown in Figure 2.

Magnetic resonance imaging of head revealed hyperintense signals (on T2-Weighted, fluid-attenuated inversion recovery images) in bilateral basal ganglia; and blooming on susceptibility weighted imaging (SWI) as depicted in Figure 3.

The diagnosis of Wilson’s disease with Hypoparathyroidism was established and patient was started on Zinc, intravenous calcium gluconate, intravenous magnesium sulfate and calcitriol. Later, he was continued on calcium and calcitriol and his tetany started improving by the time he was discharged with no further seizures, and Zinc was continued for primary disorder.

Various endocrine and metabolic abnormalities have been reported in Wilson’s disease.\(^3\) However, association of hypoparathyroidism with Wilson’s disease has been reported only at very few instances in literature.\(^3,4\) Hypoparathyroidism is a rare endocrine disease, characterized by absent or inappropriately low levels of parathyroid hormone (PTH), low serum calcium levels, and elevated serum phosphorus in the circulation and most frequently attributed to surgical damage to the parathyroid glands.\(^5\) Wilson’s disease is one of the rare causes of hypoparathyroidism with suggested mechanism being toxic damage and destruction of parathyroid gland by copper deposition.\(^6\) Presentation of our patient with seizures along with signs of hypocalcemia in the form of tetanic spasms lead us to make the etiological diagnosis of Wilson’s disease with hypoparathyroidism, supported by relevant investigations. So, Wilson’s disease as a cause of hypoparathyroidism should be suspected in any patient who presents with features of hypocalcemic tetany, seizures along with hepatic involvement.

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**Conflicts of interest**

There are no conflicts of interest.

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