diagnosis of schizophrenia. Neurological examination was normal. Cranial computed tomography (CT) revealed calcification in the bilateral CP and pineal gland and cerebellar atrophy. CPC was located in the atrium of both ventricles, and its size was 12.5 mm and 13.5 mm in the central anteroposterior and right ventricle, and in the left ventricle, respectively (Figure 1). Electroencephalography was normal. The amount of calcium, phosphate, magnesium, levels of vitamin D, parathyroid and thyroid hormones were found within the normal ranges. No organic cause was found for the CPC. She was discharged with risperidone therapy after a one-month follow-up.

Discussion

This paper presents an adolescent patient having an association between very early onset schizophrenia and bilateral macro CPC. Age-related physiological, congenital, infectious, endocrine, metabolic, vascular and neoplastic causes are known to be implicated in the etiopathogenesis of CPCs. This reason is not well understood although it assumes that physiological calcifications are associated with aging or degenerative changes.

CPC incidence increases with increasing age. Modic et al. [3] examined about 1,000 consecutive CT scans for calcification of the choroid plexus, and found that the incidence of calcification ranged from 0.5% in the first decade of life to 86% in the eighth decade. Relevant literature overlaps with the CPC seen in our patient and with the patient's clinical picture. Sandy et al. [4] suggest that CPC size positively correlated with intensity of hallucination, and was unrelated to delusions, conceptual disorganization, grandiosity, excitement and global Brief Psychiatric Rating Scale psychopathology score, but might be a major marker for hallucinations seen in schizophrenia. The same study found that CPC was not related to patients' age, sex, age at onset of psychiatric illness, duration of psychiatric illness, duration of patients' education, and present IQ [4,5]. There was a significant relationship between CPC and depressive symptoms seen in patients with schizophrenia, this coincided with a change in the 5-HT functions of CPC. CPC might be useful as a marker of suicidality in patients with schizophrenia and bipolar disorder [5]. It is also reported that pineal calcification may serve as a marker of hallucinations in schizophrenia [6]. In our case, we also observed hallucinations, delusions and severe negative symptoms with bilateral CPC and pineal calcification.

It has been noted that CPC was associated with the atrophy in brain, and with important cognitive dysfunction, persistence of negative symptoms, decline in social functioning, incomplete remissions and multiple hospitalizations in a recent study, which suggests that cerebellar atrophy is especially related to severe cognitive impairment and resistance to treatment [2]. Cerebellar atrophy was present in our case, in which severe negative symptoms, marked cognitive impairment and partial response to treatment were observed.

The CP contains a high density of 5-HT (5-HT 1C and 5-HT 2C) receptors. 5-HT increases blood flow in the CP, thereby increasing the permeability and secretory activity of the epithelium of the CP. 5-HT elevates intracellular levels of calcium in CP epithelium by acting 5-HT receptors and increasing calcium [Ca^{2+}] influx. A decrease in 5-HT functions in the CP could lead to a reduction in metabolic activity, which results in a fall of CSF production, and this reduction in metabolic activity of CP damage, and may be a predisposition to calcification. So, altered serotonergic innervation patterns can facilitate the process of calcification [7].

The blood-brain barrier is permeable to (Ca^{2+}) ions, which control the neurotransmitters and function of NMDA receptors. Reduction of the NMDA receptors is responsible for the etiopathogenesis of schizophrenia [8]. The excessive (Ca^{2+}) entry into neurons produces excitotoxicity and destruction of neurons whilst reduced extracellular (Ca^{2+}) decreases the stimulation of NMDA receptors. CPC could be an important marker, which suggests that such a mechanism leads to brain damage.

In conclusion, CPC may particularly be a neuroimaging marker for very early onset schizophrenia; a finding that needs further research.

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