Is the Incidence of Thyroid Cancer Increasing in Medical Staff?

The Editor,

Sir,

Thyroid cancers (TC) are malignant neoplasms originating from follicular or parafollicular thyroid cells, with five-year relative survival of approximately 85% in Europe (1). Current incidence of TC is 14.3 per 100 000 individuals in the United States of America [USA] (2).

While the incidence of many head or neck cancers in the USA is decreasing, a number of studies have indicated that the incidence of TC is increasing (3–6). Li *et al* showed that, in the past decades (1980–2008), there is an increased incidence of TC in the USA after adjustments were made to null the effect of improved diagnostic modalities (6).

To examine the incidence of TC, we reviewed the records of 4009 medical staff including doctors and nurses (2236 female *vs* 1773 males; aged 30–60 years old), who had undergone routine physical examination in our physical examination centre from December 2010 to November 2013. All of these medical staff members were subjected to ultrasound screening and those with suspicious findings were finally confirmed with fine needle aspiration biopsy. Among the medical staff, 1.10% (44/4009) were finally confirmed with TCs at the first time. Thyroid cancers were significantly more prevalent in females (1.34%, 30/2236) than males (0.79%, 14/1773) in the medical staff. Incidence in the 40–50-year old group was 7.23% (29/4009); higher than in the 50–60-year old group (0.03%, 12/4009) and the 30–40-year old group (0.07%, 3/4009). In thyroid cancers, 20.5% (9/44) of cases had a history of benign thyroid nodules and 4.55% (2/44) had non-toxic goitre. Pathology results showed that 0.95% (38/4009) of the cases was papillary TC while 0.15% (6/4009) was follicular TC.

The best-established cause of TC is exposure to ionizing radiation, particularly in childhood and young women (7). Chronic iodine deficiency and consequent rise in thyroid-stimulating hormone (TSH) secretion were firmly established as risk factors for goitre and follicular TC (8), while some studies have suggested that iodine supplementations could increase the incidence of papillary TC (9).

For the medical staff, there was no evidence that they had received excess ionizing radiation, but very low-dose radiation may be unavoidable in the working environment of a hospital. Besides, working stress is also a risk that cannot be ignored in China. Factors such as stress, environment and individual well-being could impact the pathogenesis. Currently, whether the high incidence of TC is a small probability coincidence requires research. For scientific researchers, this phenomenon should be taken seriously to give an evidence-based explanation.

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Keywords: Incidence, medical staff, thyroid neoplasms

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Dural Venous Sinus Thrombosis in Sickle Cell Disease in a West Indian

The Editor,

Sir,

Intracranial neurovascular manifestations in sickle cell disease include cerebral infarction, parenchymal and epidural haemorrhage, subarachnoid haemorrhage with single or multiple aneurysms, Moyamoya syndrome and posterior reversible encephalopathy syndrome.

Dural venous sinus thrombosis (DVST) in sickle cell patients has been reported rarely in patients who carry the trait and the sickle cell disease (1–3). We report the first case of DVST in sickle cell disease from the West Indies, a region where this disease is endemic.

A 14-year old Afro-Trinidadian male with confirmed haemoglobin SS (HbSS) disease presented with fever, generalized body pains and right orbital swelling for 10 hours. His past medical history was significant for recurrent painful crises and a right orbital cellulitis at the age of seven years. He was not on hydroxyurea treatment. Both parents had the trait and a brother was HbSS.

On examination, he was febrile with temperature 39.5 °C, oxygen saturation (SpO2) 90% on room air with a respiratory rate of 32/minute. He was ill-looking and dehydrated. Neurological and other system examination was normal. His Hb was 7.4 g/dL and white blood cell count was 39 200 cells/uL. Treatment included oxygen, intravenous fluids, paracetamol for fever and parenteral antibiotics. Chest X-ray revealed minimal scattered infiltrates in both lung fields. Computed tomography (CT) scan of the brain showed a left cerebellar acute ischaemic infarct. Computed tomography venography or magnetic resonance imaging (MRI) with venous MR of the brain and thrombophilia screens were not obtained due to unavailability at the time. The patient was intubated, ventilated and died sixteen hours after presentation. Postmortem showed cerebral oedema (brain 1240 g) and DVST which also involved the superior sagittal region (Figure). Other postmortem findings were bilateral pulmonary thromboembolism involving medium sized and small vessels with pulmonary infarction. There was also cardiomegaly 430 g, with biven-tricular dilatation. The liver weighed 1660 g and there were features of right heart failure and iron overload. Splenic atrophy (10 g) was also noted. Death was likely due to a combination of pulmonary arterial thrombosis and dural DVST.

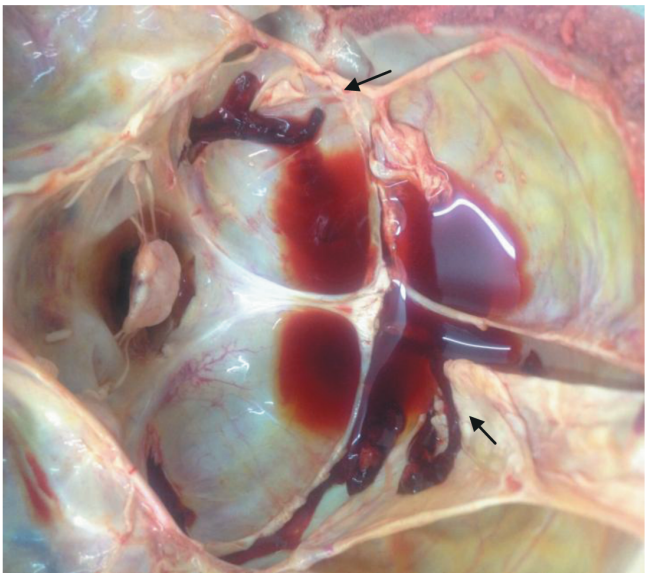

Figure: Extensive dural venous sinus thrombosis in anterior and middle cranial fossa (arrows).

Symptoms in DVST vary from only headaches, motor deficits, aphasia and seizures (4). Diagnosis is confirmed by detection of thrombus in the cerebral venous sinus system and in the brain parenchyma. Though CT scan with contrast can aid with diagnosis, the noninvasive investigation of choice is CT venography or magnetic resonance venography (MRV) which was unavailable to us at the time. Three signs seen on unenhanced CT scan are: (i) dense clot (triangle) sign, signifying acute thrombosis, present only in 55% of cases; (ii) empty delta sign, secondary to a partial recanalization of the thrombus on a contrast scan, seen in 20% of cases; (iii) cord sign: curvilinear or linear hyperdensity over the cerebral cortex caused by a thrombosed cortical vein. Computed tomography scan with contrast can miss the diagnosis in up to 40% of patients.

The initial treatment should be intravenous heparin/low molecular weight heparin with thrombolysis or thrombectomy reserved for cases undergoing secondary deterioration (5). Oral anti-coagulation with warfarin is given for three months if DVST was secondary to a transient risk factor or six to 12 months in patients with idiopathic DVST. Early recognition by appropriate imaging studies – CT venography or MRV – is necessary to begin treatment and thus prevent death.

Keywords: Dural venous sinus thrombus, haemoglobin, sickle cell disease

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