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Short Communication

Does Exposure to Air Pollution Fine Particles and COVID-19 Contribute to the Risk of Ischemic Stroke?

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Introduction

A stroke in the brain is caused by ischemia that led to sensorimotor disorders that could potentially brain damage or cause dementia. The stroke can be classified according to the cause of ischemia. Ischemic stroke results from atherothrombotic blockage of blood vessels inside the brain or vessels that lead to the brain (such as carotid arteries), or bleeding from the brain due to rupture of cerebral arteries and cerebral hemorrhage. In any case, oxidative stress is one of the major features of ischemia, and the ischemia caused damage by sudden tissue reperfusion and pathophysiological changes in cells of affected area. Was suggested that after inhaling vehicle exhaust gases, oxidative stress plays an important role in disrupting the Blood Brain Barrier (BBB) [1-4]. Indeed, the photochemical induction of the thrombosis in cerebral arteries was intensified after Diesel Exhaust Particles (DEPs) exposure [2]. COVID-19, which first appeared in Chinaand has rapidly spread worldwide, is a highly transmissible viral infectious disease caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) [3]. SARS-CoV-2 is seventh known Coronavirus in humans. While four types of Coronaviruses namely (OCw43, HKU1, 229E and NL63) and contribute to a significant proportion of mild lower and upper respiratory tract infections, MERS-CoV, SARS-CoV, and SARS-CoV-2 may cause severe disease [1]. Several studies reported that air pollution exposure exacerbates the intensity of various respiratory diseases [4], for example influenza infection [5] and Severe Acute Respiratory Syndrome (SARS) or another Coronavirus [2]. One study in the US indicates that PM2.5 exposure and ozone was dangerous and increased the risk of SARS among older adults [6]. Based on this presupposition, it is possible that air pollution exposure will alter the intense of the COVID-19 symptoms or help explain differential-spatial patterns of disease prevalence. Recent surveys have reported that people with severe COVID-19 may already have respiratory disease [7]. Recent studies on viral respiratory disease (such as influenza) have shown that aviable virus can be emitted from infected peoples by speaking even breathing, without sneezing or coughing [8,9]. Normal and ordinary speech converts significant amounts of respiratory particles into airborne aerosols. Experimental research has shown that vocalization emits up more aerosols than breathing [10], also, a recent study indicated the louder one speech, and the more aerosols are produced [11]. COVID-19 is a severe respiratory infection, and recent studies clearly identified the SARS-CoV-2 presence in a tract of the respiratory system [12]. Therefore, particles derived from breath and speech may contain viruses. These particles may be due in part to the mechanism of "liquid film bursting" in alveoli in the pulmonary, and or through the vibration of the vocal cords during a speech [13]. The findings suggest that particles and aerosols in the air reach the brain and affect CNS health, with changes in the Blood-Brain Barrier (BBB) or leakage and transmission along the olfactory nerve to the Olfactory Bulb (OB) and active Microglia are the main components [14,15].

In the first 221 patients retrospective study, 1 (0.5%) patient developed cerebral hemorrhage, 11 (5%) patients acute ischemic stroke, and 1 (0.5%) developed Cerebral Venous Sinus Thrombosis (CVST) [16]. Of the 12 reports that up to May 1, 2020, published, 4 were case studies (between 2 to 6 cases reporting), 5 were case reports, and 2 were observational surveys describing the CVA incidence in the COVID-19 patients. Whilst a meta-analysis study of four surveys reported that cases with stroke history were 2.5 times more likely to develop the severe COVID- 19 than those without stroke history [17]. In the second 288 patients' prospective study, 9 (2.5%) patients were diagnosed with ischemic stroke [18]. These 9-case series and case reports detail a total of 21 patients (5 females and 16 males) together [19]. Age averaged was 59.8 years (female 49.8, male 63.1), although it may be decreased due to the young strokes case series [20].

Our recent studies support the increase of oxidative stress biomarker and inflammatory effects in blood and the brain tissue in pathological samples of cognitive and emotional impairment following exposure to air pollution DEPs. For example, anxiety and depression in male mice [21], hippocampal inflammatory cytokine response, and altered morphology [14,15], and disorders in memory and learning [22,23]. Interestingly, in 14 cases a significant increase [24] in D-Dimer levels (≥ 1000 μg/L) was reported, of which 9 had the anticardiolipin antibodies and the variable results in the lupus anticoagulant test. In a group of 6 cases from UK, one cases had a medium titre for the anti-cardiolipin IgM antibody and anticoagulant of lupus was positive in the five patients out of the six [24]. In a group of 3 cases from Beijing, anti-cardiolipin IgA antibodies also IgG antibodies and anti-β2-glycoprotein I IgA, were positive in all patients but no lupus anticoagulant was observed in any patients [25].

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Exposure to PM10, PM2.5 and UFPs showed acute side effects of ischemic stroke. Due to the occurrence of temperature inversion in the cold seasons of the year, exposure to UFPs will increase and will have more severe effects. Implementing effective air pollution control policies can be helpful in reducing the stroke burden. Further researches are needed to discover the UFPs effects on human health. Also, advanced technology and tools for PM monitoring and control need to provide a little more information about the sources of PM pollution. Considering the additional risk that some communities may face with COVID-19, the interplay between COVID-19 prevention measures and coping strategies against the epidemic are essential.

Keywords: COVID-19; SARS-CoV-2; Blood brain barrier; Blood vessels

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