Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

data suggesting increased venous thromboembolism rates when D-dimer concentrations exceed 3 µg/mL.[2] Five patients with D-dimer concentrations of 3 µg/mL or higher and known (or highly suspected) thrombosis received therapeutic anticoagulation. Two of the patients received intravenous heparin, and three patients received a direct thrombin inhibitor (argatroban or bivalirudin) because of heparin resistance or concern for heparin-induced thrombocytopenia. Four patients with D-dimer concentrations below 3 µg/mL received low-dose thromboprophylaxis with low-molecular-weight heparin (LMWH) or subcutaneous heparin. Six patients with D-dimer concentrations of 3 µg/mL or more and without known thrombosis received intermediate dosing (ie, subtherapeutic) of LMWH or intravenous heparin.

The 15 patients had plasma viscosity exceeding 95% of normal, as determined by traditional capillary viscometry, ranging from 1·9–4·2 centipoise (cP; normal range 1·4–1·8). Notably, the four patients with plasma viscosity above 3·5 cP had a documented thrombotic complication: one patient had pulmonary embolism, one patient had limb ischaemia and suspected pulmonary embolism, and two patients had CRRT-related clotting. Plasma viscosity and Sequential Organ Failure Assessment scores, a measure of illness severity, were strongly correlated (Pearson's r=0·841, R^2=0·7072, p<0·001; appendix).

Hyperviscosity damages endothelium and is a known risk factor for thrombosis.[3] It can result from increases in cellular components or plasma proteins, such as fibrinogen or immunoglobulin, as seen in Waldenström macroglobulinaemia. Consistent with reports of hyperfibrinogenaemia in patients with COVID-19, our patients had substantially increased fibrinogen concentrations (median 708 mg/dL, range 459–1188; normal reference range 200–393).[4] Further study is needed to evaluate which plasma components, including acute phase proteins such as fibrinogen, contribute to COVID-19-associated hyperviscosity.

Our novel observation might provide an important link between inflammation and coagulopathy in critically ill patients with COVID-19. We are actively exploring any beneficial role of therapeutic plasma exchange, a highly effective treatment for symptomatic hyperviscosity in other conditions such as hypergammaglobulinaemia, in the clinical management of these patients.[5] Finally, any causal relationship between hyperviscosity and thrombotic complications in COVID-19 warrants immediate investigation given the potential to impact clinical care.

We declare no competing interests.

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Caution against corticosteroid-based COVID-19 treatment

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In December, 2019, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) emerged in Wuhan, Hubei province, China, and COVID-19 has become a threat to global public health.[1]

Use of corticosteroid-based therapy to reduce inflammatory-induced lung injury has been described for patients with severe COVID-19,[2,3] similar to the use of corticosteroids to treat severe acute respiratory syndrome (SARS) during the SARS outbreak in 2003.[4] However, improper use of systemic corticosteroids can increase the risk of osteonecrosis of the femoral head (ONFH).

In a retrospective study of 539 patients with SARS who received corticosteroid therapy,[5] the incidence of steroid-induced ONFH was 24%, and increased incidence of steroid-induced ONFH was associated with total corticosteroid dose and the use of more than one type of corticosteroid. Improper or delayed treatment of steroid-induced ONFH can cause hip pain, claudication, and even disability of the lower limbs.

Overall, we call for caution in the use of corticosteroids for COVID-19 and do not recommend this as a routine treatment. To prevent steroid-induced ONFH, corticosteroids should be considered only for patients undergoing septic shock, or in critical cases.[6,7] Corticosteroids should be minimised in dose and duration, and the use of multiple types should be avoided. We believe that bisphosphonates and vitamin E should be prescribed to patients who are undergoing corticosteroid treatment; anticoagulants, vasodilators, and traditional Chinese medicine could also be considered.[8–10] Close follow-up should be conducted after discharge, with MRI as the best option for early detection of ONFH. Physical therapy

See Online for appendix

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and combined pharmacotherapy have been recommended for patients with early-stage steroid-induced ONFH.[11]

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Assistance Publique–Hôpitaux de Paris' response to the COVID-19 pandemic

Assistance Publique–Hôpitaux de Paris (AP-HP) is the largest teaching hospital trust in Europe, with 39 hospitals, 20 000 beds (10% of all public hospital beds), and an association with seven universities. AP-HP also hosts 40% of the French biomedical research. As a result of its size and location, AP-HP has been a major player in France's COVID-19 response, as it was in responding to terrorist attacks.[1] AP-HP has a reputation of being an institution with a size, cumbersome operations, and internal competition that prevent any nimble response. We are a group of health-care professionals at AP-HP who are directly involved in the management of the COVID-19 response by AP-HP. Having faced the COVID-19 pandemic, and remaining humble in facing the future, we describe how size and coordinated management might actually have helped the organisation, speed, and consistency of the response to the COVID-19 crisis.

AP-HP initially estimated that no more than 1000 patients with COVID-19 would be simultaneously admitted to intensive care units (ICUs) in the Île-de-France region; 400 of these patients would be admitted to ICUs in the AP-HP, close to the maximum capacity. However, the situation became worse than expected (appendix). At the height of the COVID-19 pandemic, 2677 patients with COVID-19 were admitted into ICUs in the region, 41% of whom were admitted to AP-HP. A few points deserve discussion.

A specific medical organisation was established, led by a central crisis medical director who was appointed by the CEO and supported by medical directors in each hospital. A clear chain of medical responsibilities enabled a reactive operational decision making process throughout the whole institution.

Human resources for recruiting and training specialised staff were allocated from a single platform (appendix). We experienced shortage of various equipment and consumables made in Asia.[2] Centralised logistics made it possible to adjust quantities daily, in response to supply shortages. Universities called upon thousands of students in medicine, nursing, pharmacy, and dentistry who underwent specific training to work as paramedics, research assistants, or operators on a telemedicine platform.[3]

Management of available beds was crucial, so a central ICU bed-allocation system was organised at the regional level to optimise patient transfers across hospitals. This allocation unit was led by surgeons who were available as a result of the postponement of non-essential surgeries.

Using multidisciplinary working groups, practical guidelines were edited within 48–72 h, shared among all hospitals, and published on an AP-HP website. Examples of guidelines include the appropriate use of scarce equipment or drugs, optimal use of ventilators, and ethical issues. These guidelines were discussed during the central crisis team's twice-daily telephone meetings. Critical care response was coordinated through a daily meeting of ICU heads.

A COVID-19 research committee developed a global research strategy that was shared with the universities and INSERM. The top priorities were establishment of patient cohorts, biobanking, and clinical trials. More than 40 clinical studies enrolled more than 7000 patients. This fast initiation of research was possible because of a shared research support infrastructure and reallocation of all research personnel, supplemented by volunteers. The centralised pharmaceutical office made drugs and placebos available at each hospital. The value of this research effort will

For the **AP-HP COVID-19 website** see http://covid-documentation.aphp.fr/

See Online for appendix