

8.01 Intrinsic coagulation in early Covid-19; A central role for the Contact-Kinin System

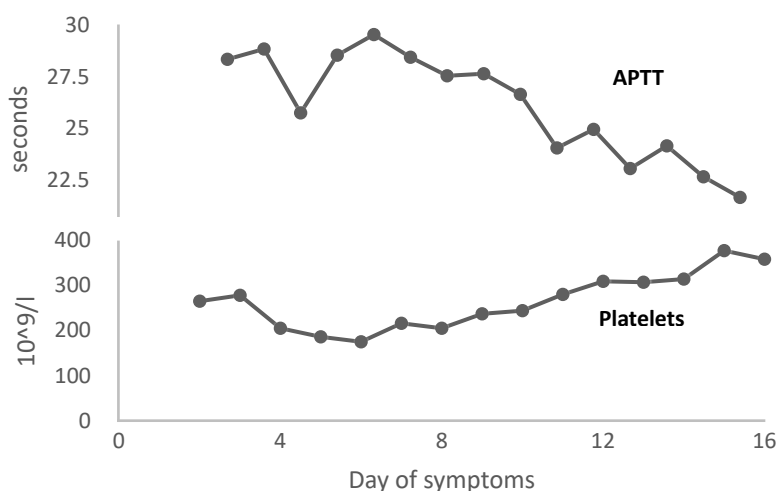
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Covid-19 causes venous thromboembolism in 17% of hospitalised cases. It also causes ground glass opacities and increased IL-6. Our hypothesis is that the contact-kinin system is canonical in the initiation of Covid-19 inflammation; Factor XII is activated to FXIIa, simultaneously triggering the intrinsic coagulation pathway and the kallikrein-kinin-system (KKS), whereby pre-kallikrein is converted to kallikrein, cleaving high molecular weight kininogen. The released bradykinin causes increased IL-6 and oedema. Ground glass radiological changes suggestive of oedema are seen in early Covid-19. We wished to determine the coagulation response in Covid-19. Blood results from 98 hospitalised patients were recorded by day of symptoms. The intrinsic pathway activation, measured by mean APTT, peaked at day 7 and returned to normal by day 10. Simultaneously, mean platelets dropped until day 6 before rising.

The increased APTT and decreased platelets demonstrates a consumptive coagulopathy. This suggests activation of the contact-kinin system which occurs in parallel with radiological GGO seen in early Covid-19. The KKS releases bradykinin which causes oedema and increased IL-6. A study is underway to inhibit bradykinin in early hypoxic Covid-19 patients.

(8.1)



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