The drug that blocks inflammation: "First improvements in 48 hours"

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A **experimental drug** could inhibit the new coronavirus. This is what was observed at the San Raffaele hospital in Milan, where a 71-year-old man improved after 10 days of treatment.

The patient was among the most at risk people with various diseases, including hypertension, renal failure and hypercholesterolemia. The man, hospitalized for an operation, had been diagnosed with pneumonia, which revealed the consequence of **COVID-19**. The doctors, who told the case in one study, they gave him the experimental drug: after **48 hours** from the start of treatment, "*the patient showed a remarkable **improvement** of all abnormal baseline parameters, resulting in rapid resolution of the extensive inflammatory response associated with COVID-19*". The improvement of laboratory parameters was also associated with that of" p*respiratory rests, with a gradual reduction of oxygen requirements*", which led him to breathe autonomously after about ten days.

The experimental drug administered to "patient 1" is called

amy-101 and belongs to the "complement inhibitors". The complement system is a set of proteins used by **immune system** to fight viruses and bacteria. A study on Sars-CoV-2 has shown that activation of the C3 component of the complement system can aggravate the disease. "*This suggests that the**inhibition** of C3 can also alleviate lung inflammatory complications of Sars-CoV-2 infection "*, explained the researchers in an article on *Nature*. Amy-101 targets the C3 protein, trying to avoid the onset of the "cytokine storm" at the origin of the aggravation of the disease. The drug acts upstream of the "storm", unlike two other experimental drugs (tocilizumab and anakinra), which block interleukins 6 and 1.

The results of the study on the first patient who was given the drug are **positive** is "*indicate that C3 inhibition has potential as a new anti-inflammatory therapy in Covid-19 and paves the way for systematic prospective studies*". A challenge carried out by Fabio Ciceri, deputy scientific director for clinical research at San Raffaele in Milan:"*We decided to point to the switch upstream of everything, namely the activation of the complement (C3), which is the first event of the inflammatory cascade*- explains the expert, according to what reported in the *Sole24ore- If I act on interleukin 1 (IL1) I only switch off the mediation of inflammation that is orchestrated by IL1, the same if I intervene only on interleukin 6. With the complement inhibitor, however, I block*

*everything, because **I speak at the root** of inflammation, consequently all that is downstream goes out*".

And now, according to Sole24ore, there would be a second 58-year-old patient who improved after following the same treatment. For the time being, there are only two patients, but the results are encouraging.