International Surgery Journal
*Sanjay R et al. Int Surg J. 2021 Sep;8(9):2856-2859*
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: https://dx.doi.org/10.18203/2349-2902.isj20213636

**Case Report**

# Extensive post-covid mucormycosis salvaged by old fashioned negative pressure wound therapy

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**Received:** 01 July 2021
**Revised:** 04 August 2021
**Accepted:** 07 August 2021

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## ABSTRACT

Mucormycosis represents a group of life threatenening infections caused by fungi of the order *Mucorales*. *Mucorales* are ubiquitous environmental fungi that grow well in moist and damp conditions. They can invade arteries to cause thrombosis and infarction. These fungi cause infection primarily in patients with diabetes, neutropenia, elevated levels of free iron and those who are on glucocorticoid therapy, which supports fungal growth in serum and tissues. Recently the incidence of mucormycosis was found to have increased in patients previously infected with coronavirus (COVID-19). Here we presented a 52 year old lady who was previously infected by coronavirus, was administered glucocorticoids as a part of empirical treatment for COVID-19 pnemonitis. She recovered uneventfully within a few days, but, around a month later presented with erythematous papule over the anterior chest wall. The papule developed into a large fungating ulcer over a period of 10 days. KOH mount and culture confirmed it to be mucormycosis. Cutaneous mucormycosis can be highly invasive, penetrating into muscle, fascia and even bone. These are molds that grow well in moist and damp conditions. But depriving them of moisture and oxygen can help control their spread. This could be achieved by thorough debridement and applying negative pressure to the wound. The results achieved are remarkable and satisfactory.

**Keywords:** Post-covid mucormycosis, Negative pressure wound therapy, Anterior chest wall mucormycosis

## INTRODUCTION

Mucormycosis represents a group of life threatenening infections caused by fungi of the order *Mucorales* of the subphylum *Mucormycotina*, formerly known as class *Zygomycetes*. Mucormycosis is highly invasive and relentlessly progressive, resulting in higher rates of morbidity and mortality than many other infections. The *Mucorales* are ubiquitous environmental fungi to which humans are constantly exposed. These fungi cause infection primarily in patients with diabetes, defects in phagocytic function (e.g. in neutropenia and glucocorticoid therapy) and elevated levels of free iron, which supports fungal growth in serum and tissues.[1-4]

Recently the incidence of mucormycosis is found to have increased in patients previously infected with COVID-19 to such an extent that it was declared as an epidemic in some states. The reason behind this was still unclear but it was proposed that glucocorticoid induced hyperglycemia was the main culprit. Glucocorticoids are still the main stay of treatment in COVID-19 pneumonitis.

Hyperglycemia directly contributes to the risk of mucormycosis by atleast four likely mechanisms: hyperglycation of iron-sequestering proteins, disrupting normal iron sequestration; upregulation of a mammalian cell receptor GRP78 that binds to *Mucorales*, enabling tissue penetration (due to both a direct effect of

hyperglycemia and increasing free levels of iron, which independently enhances GRP78 expression; induction of poorly characterized defects in phagocytic function; enhanced expression of Co t H, a *Mucorales*, specific protein that mediates host cell invasion by binding to GRP78 (due to hyperglycemia and resulting free iron).[1,2]

## CASE REPORT

A 52 year old lady who was previously infected by coronavirus and had mild respiratory illness, was administered glucocorticoids as a part of empirical treatment. She recovered uneventfully within a few days, but around a month later presented with erythematous papule over the anterior chest wall (Figure 1). The papule developed into a large fungating ulcer over a period of 10 days (Figure 2). KOH mount and culture confirmed it to be mucormycosis. The patient had multiple episodes of fever and had breathing difficulty. But the ulcer was painless.

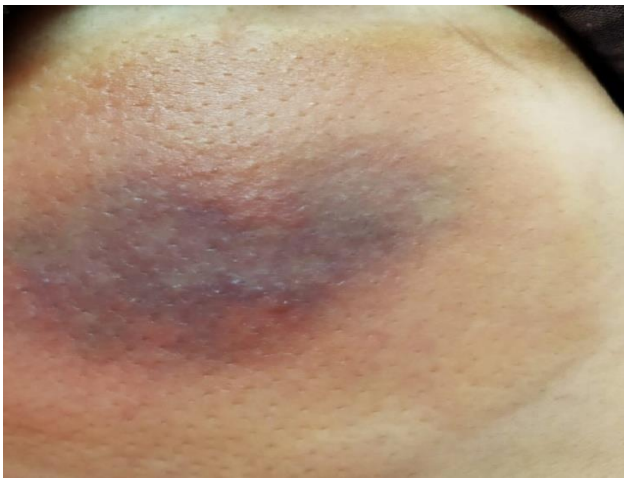

**Figure 1: Initial presentation of mucormycosis as an erythematous papule.**

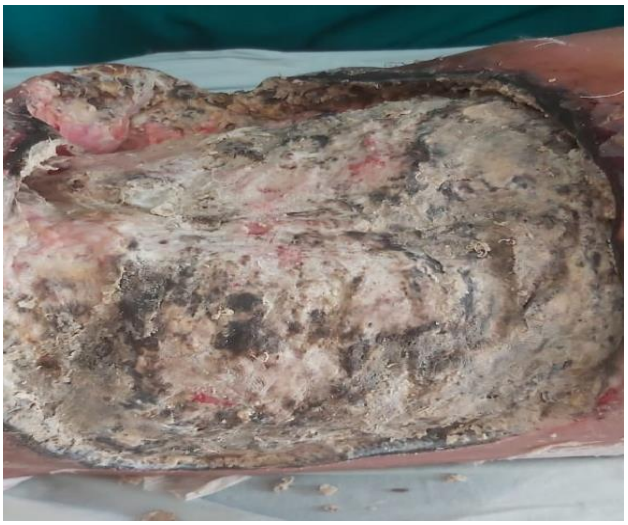

**Figure 2: Developed into a large fungating ulcer within 10 days.**

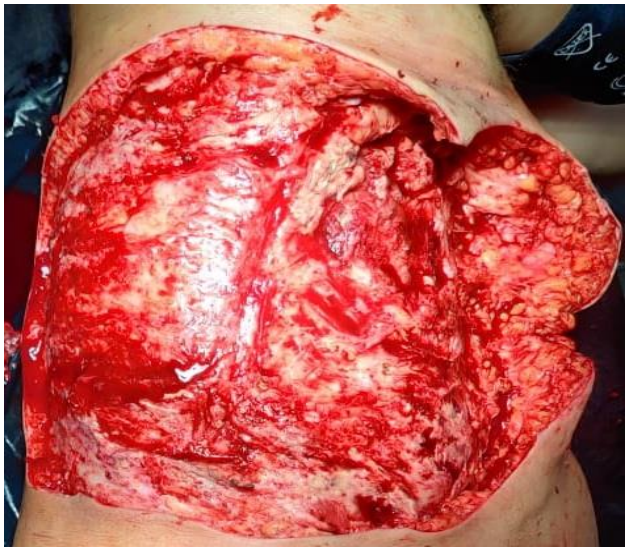

**Figure 3: After initial debridement.**

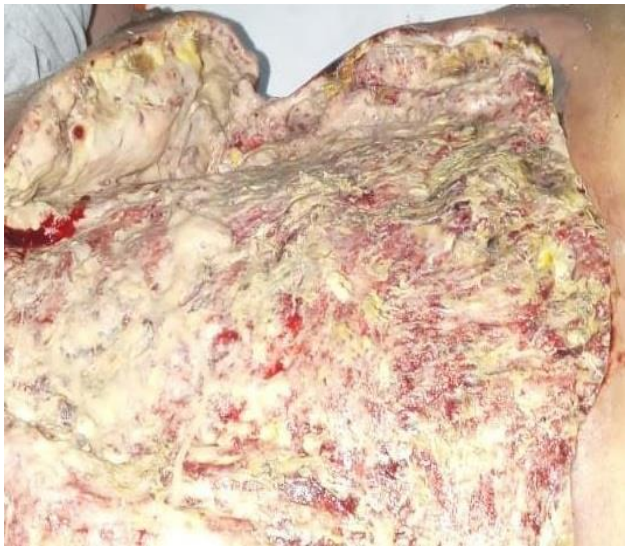

**Figure 4: Two days later initial debridement.**

Patient was debrided under general anesthesia but the fungal infection was so extensive that it grew again within 2 days (Figure 3 and 4).

## DISCUSSION

Cutaneous mucormycosis can be highly invasive, penetrating into muscle, fascia and even bone. Necrotizing fasciitis caused by mucormycosis carries a mortality rate approaching 80%.[3,6] Necrotic cutaneous lesions in the setting of hematogenous dissemination also were associated with an extremely high mortality rate.[1-4] *Mucorales* are ubiquitous molds that grow well in moist and damp conditions. They can invade arteries to cause thrombosis and infarction.[3,4,6] Depriving the fungus of moist and damp conditions can help control its spread. This could be achieved by thorough debridement and creating a negative pressure over the wound. This

negative pressure helps in creating a vacuum and hence prevents spread of fungal hyphae.

This negative pressure wound closure or vacuum-assisted closure was an old fashioned cost effective technique which could be done easily in wards. Applying intermittent negative pressure of approximately 125 mmHg appears to hasten debridement and the formation of granulation tissue in chronic wounds and ulcers.[5,7,8] After thorough debridement and applying amphotericin ointments to the wound, foam sheets (foam dressing) which were autoclaved beforehand were cut in the size of the wound. Romovac drain tubings (perforated drains) were stitched to the foam sheets. Opsite incise sheet was tightly applied to the wound followed by two layers of ioban sheets making the wound airtight. Edges were sealed with tape to prevent air leak. Negative pressure was obtained by connecting the romovac tube to suction device in wards (Figure 5 and 6). This created a vacuum inside the wound depriving the fungus of its optimal requirements to grow, that is, aerobic, moist and damp conditions. Negative pressure may act by increasing blood flow. As a result, bacterial counts decreased and cell proliferation increased, thereby creating a suitable bed for graft or flap cover.[5]

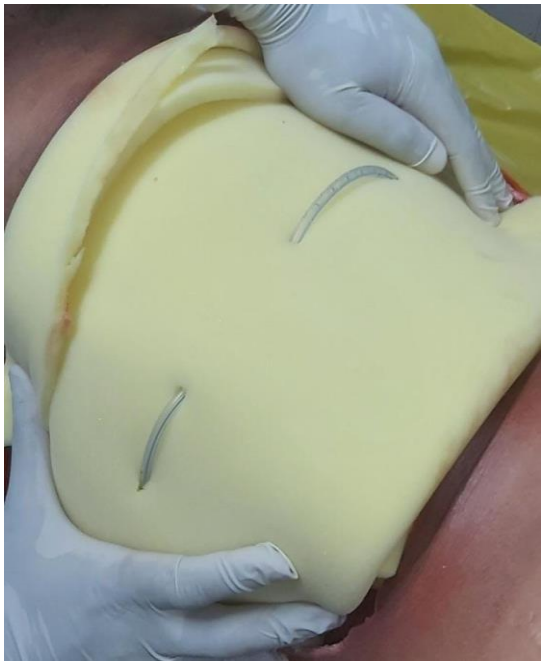

**Figure 5: Foam sheets cut in the shape of the wound.**

There was marked decline in the spread of mucor following application of negative pressure and healthy granulation tissue began to appear. This dressing was changed everyday after almost 24 hours and continous suction was given. After 4 days of this negative pressure wound therapy, the ulcer now had healing margins and healthy pink granulation tissue.

The effect of negative pressure wound therapy over extensive mucormycosis ulcer was given in Figure 7-10.

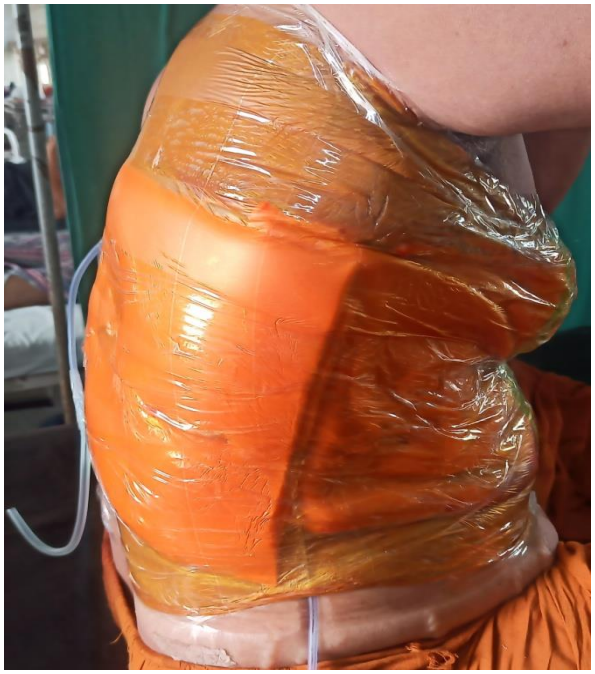

**Figure 6: Negative pressure wound therapy.**

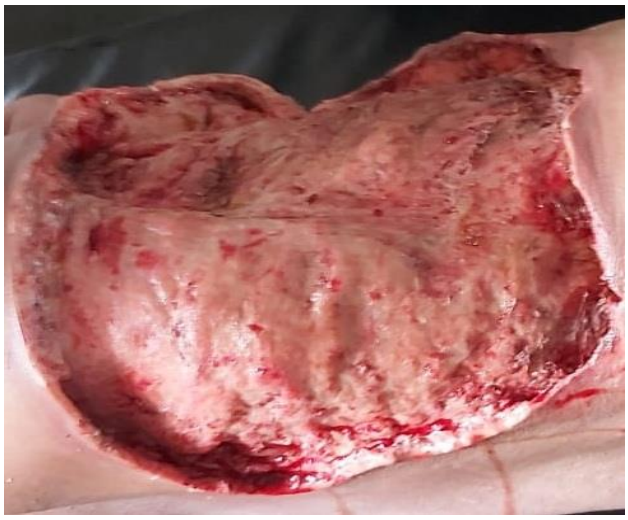

**Figure 7: Day 1 of negative pressure wound therapy.**

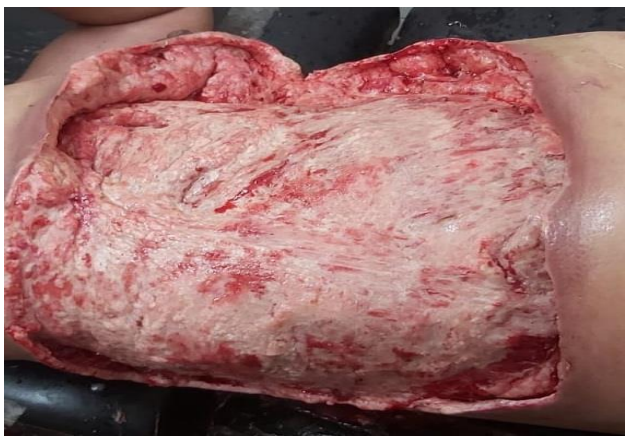

**Figure 8: Day 2 of negative pressure wound therapy.**

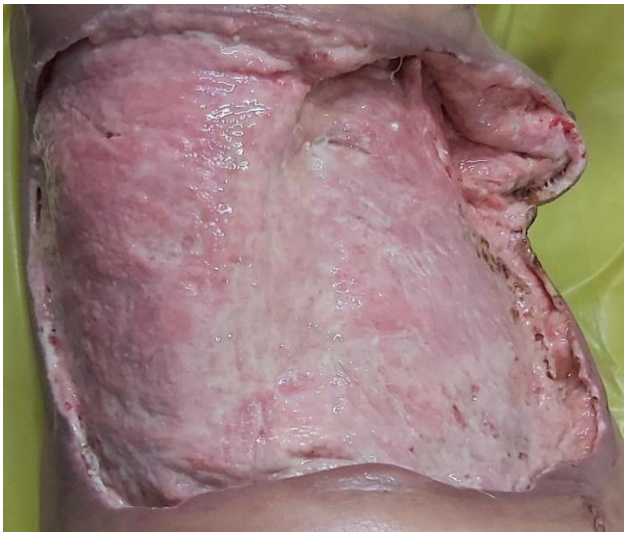

**Figure 9: Day 3 of negative pressure wound therapy.**

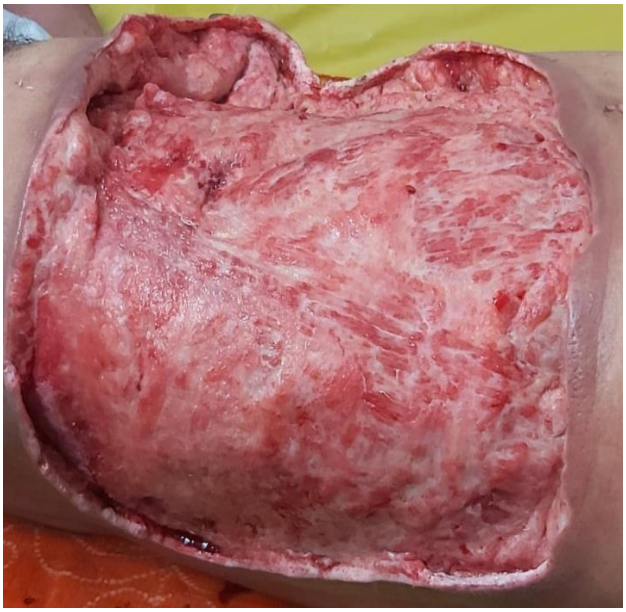

**Figure 10: Day 4 of negative pressure wound therapy.**

## CONCLUSION

In recent times the incidence of post-covid mucormycosis has increased mainly due to the use of glucocorticoids which is the mainstay of treatment of COVID pneumonitis. The *Mucorales* are ubiquitous environmental fungi to which humans are constantly exposed. These fungi cause infection primarily in patients with diabetes, defects in phagocytic function and elevated levels of free iron, which supports fungal growth in serum and tissues. These are molds that grow well in moist and damp conditions. But depriving them of moisture and oxygen can help control their spread. This could be achieved by thorough debridement and applying negative pressure to the wound. This is an old fashioned cheaper but very effective idea to control the spread of mucormycosis. The results achieved are remarkable and satisfactory.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

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**Cite this article as:** Sanjay R, Khan F, Gupta S, Chikkarangappa R, Choudhary K. Extensive post-covid mucormycosis salvaged by old fashioned negative pressure wound therapy. Int Surg J 2021;8:2856-9.