IVERMECTIN and FENBENDAZOLE Testimonial 70s year old patient with Diffuse Large B-Cell Lymphoma - dramatic impact in first two wee

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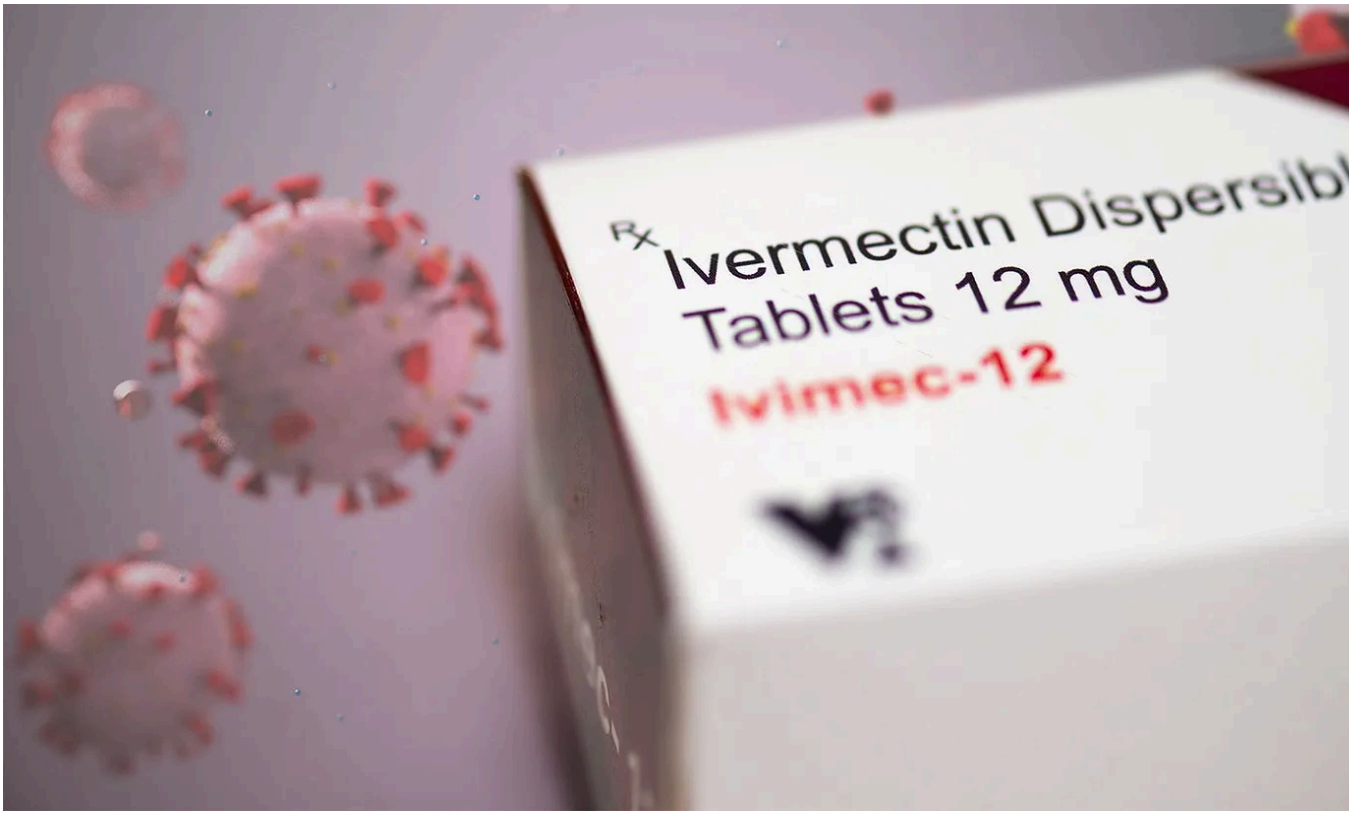
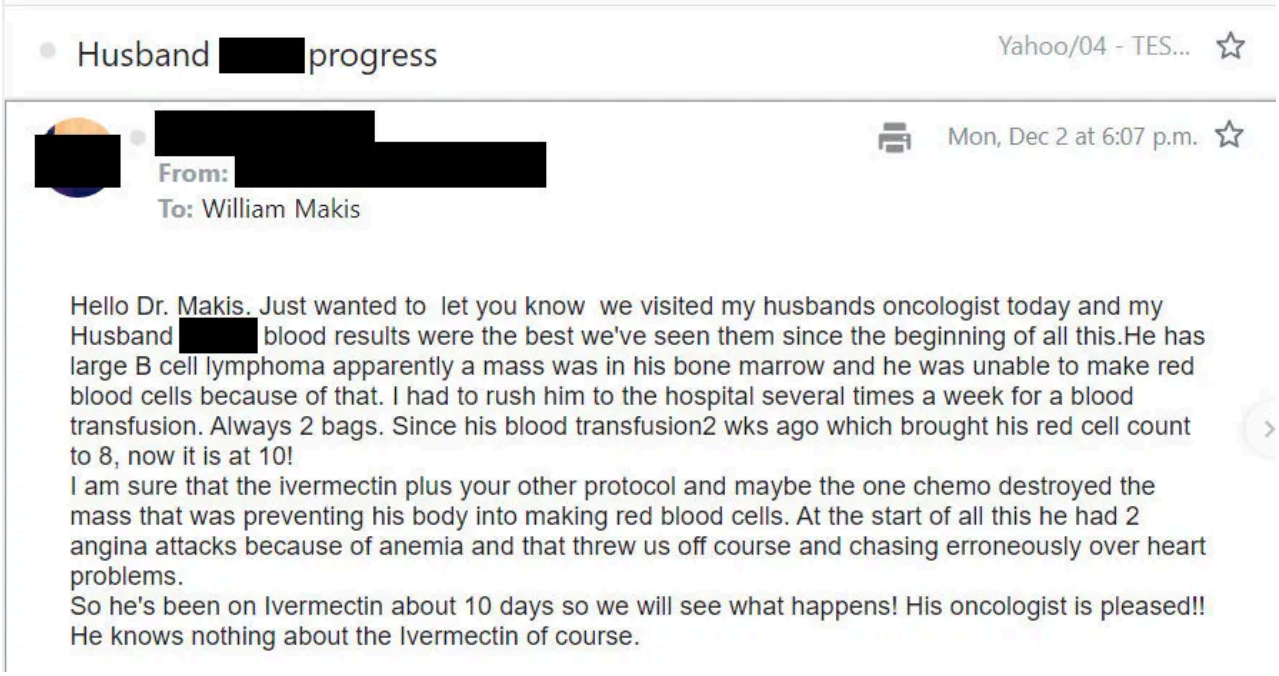
DEC 08, 2024 · PAID



STORY:

- 70s year old USA patient recently diagnosed with LYMPHOMA.
- We started a high dose Ivermectin and Fenbendazole Protocol



My Take...

I've had many people ask me about Ivermectin and lymphomas.

I have dozens of lymphoma and leukemia patients and we are already starting to bloodwork changes.

2020 Li et al - Progress in Understanding the Molecular Mechanisms Underlying the Antitumour Effects of Ivermectin

"Studies have demonstrated that some cancers, including leukaemia, breast canc and lymphoma, are more metabolically active and dependent on mitochondria th normal cells and therefore more responsive to Ivermectin than their normal counterparts"

"Ivermectin Inhibits Mitochondrial Respiration"

Ivermectin Inhibits Mitochondrial Respiration

Mitochondrial dysfunction is a major effect of reactive oxygen species (ROS), and superoxide, a precursor of many other forms of ROS, is formed mainly by mitochondrial electron leakage caused by mitochondrial dysfunction.[25] Liu Yingying et al demonstrated the dose-dependent inhibitory effect of ivermectin on the basal oxygen consumption rate (OCR) and maximum OCR in U87, T98G and human microvascular endothelial cells (HBMEC) cells, which indicates that ivermectin inhibits mitochondrial respiration by decreasing the activity of respiratory complex I enzyme.[24] These researchers also found that the exposure of these cells to ivermectin decreased the mitochondrial membrane potential, an electrochemical proton gradient generated by the mitochondrial respiratory chain.[24] Consistently, obviously increased levels of ROS and mitochondrial superoxide as well as decreased ATP levels were also found in glioblastoma, HBMECs and chronic myeloid leukaemia (CML) cells treated with ivermectin.[24,25] The anti-proliferative and apoptotic effects of ivermectin were abrogated through communication with the antioxidants α-tocopherol or mannitol, and the levels of phosphorylated Akt, mTOR and ribosomal S6 protein (rS6), which are downstream of mTOR, were reduced in U87, HBMEC, T98G and K562 cells exposed to ivermectin, reflecting the negative effect of ivermectin on mitochondrial function through its suppression of the Akt/mTOR pathway, which leads to oxidative stress.[24,25] Similarly, acetyl-l-carnitine (ALCAR, a mitochondrial fuel) and N-acetyl-l-cysteine (NAC, an antioxidant) reversed the inhibitory effects of ivermectin in renal cell carcinoma (RCC) cells, which indicates that mitochondria are the target of ivermectin.[52] Furthermore, consistent with ROS induction, ivermectin appears to dysregulate genes, including STAT1, which has been connected with increased ROS production, and IFIT3, OAS1, and TRIM22, which are downstream targets of STAT1.[11] Some recent data demonstrate that ivermectin exhibits selective toxicity in inducing mitochondrial dysfunction and oxidative stress and enhances the role of BCR-ABL TKIs in CD34 CML cells.[25] Studies have demonstrated that some cancers, including leukaemia, breast cancer and lymphoma, are more metabolically active and dependent on mitochondria than normal cells and therefore more responsive to ivermectin than their normal counterparts.[52]

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