Heat shock proteins (HSPs), molecular chaperones, are crucial for the cancer cells to facilitate proper functioning of various oncoproteins involved in cell survival, proliferation, migration, and tumour angiogenesis. HSPs are overexpressed in many cancers due to upregulation of transcription factor Heat-shock factor 1 (HSF-1), a multifaceted master regulator of heat shock response. Therefore, pharmacological targeting of HSPs via HSF-1 is an effective strategy to treat malignant cancers like triple negative breast cancer (TNBC). In the current study, we evaluated the efficacy of a pyrrole derivative [bis(2-ethylhexyl)1H-pyrrole-3,4-dicarboxylate], TCCP, purified from leaves of Tinospora cordifolia for its ability to suppress heat shock response, angiogenesis and increase apoptosis, using MDA-MB-231 cells and the murine mammary carcinoma: Ehrlich ascites tumour (EAT) model. HSP90 was downregulated by TCCP by inactivation of HSF-1 resulting in inhibition of tumour cell proliferation, VEGF-induced cell migration, and concomitant decrease in tumour burden and neo-angiogenesis in vivo. The mechanism of suppression of HSPs involved inactivation of PI3K/Akt and phosphorylation on serine 307 of HSF-1 by the activation of ERK1. HSF-1 and HSP90 and 70 localization and expression were ascertained by immunolocalization, immunoblotting, and qPCR experiments. Further, the pro-apoptotic nature of TCCP on MDA-MB-231 was determined by assessing apoptotic markers. Treatment of MDA-MB-231 cells with TCCP caused generation of endogenous reactive oxygen species, increase in intracellular calcium and activation of phospho-p53 in a dose dependent manner. This led to the downstream altered expression of BCL-2 and BAX proteins, mitochondrial membrane depolarization, MPTP, and cardiolipin peroxidation. TCCP caused cytosolic cytochrome c release in to cytosol, caspase activation and ultimately resulted in DNA fragmentation. In addition, induction of apoptosis and morphological alterations were evident from the phosphatidylserine externalization and increase in sub G1 population. The anti-angiogenic effect of TCCP was studied in vivo in tumour-bearing mice and ex vivo using rat corneal micro-pocket assay. All the results thus corroborate the logic behind inactivating HSF-1 using TCCP as an alternative approach for cancer therapy. TCCP was shown to be capable of inducing cytotoxicity, cell death in MDA-MB-231 cells and decreased tumorigenesis. Furthermore, TCCP effectively inhibited DNP-induced oxidative stress on blood components, which was evidenced by the levels of endogenously generated reactive oxygen species and hydrogen peroxide along with decreased expression of oxidative stress markers such as lipid peroxidase, catalase, lactate dehydrogenase (LDH), and phosphatases. In addition, the depletion of reduced glutathione (GSH) content in DNP-treated cells was prevented by TCCP. Therefore, these studies suggest the use of TCCP as an antioxidant agent.