The biological treatment [Di Bella Method] has improved survival, objective response and performance status in 122 cases of mammary carcinoma

Di Bella Giuseppe – Di Bella Foundation
posta@giuseppedibella.it
+39-051-239662 / +39-339-3830224
via Marconi 51 - 40122 Bologna

Aims

Study of objective response, survival rate and performance status following DBM biological treatment. Increase the efficacy and reduce the toxicity of cancer therapy.

Methods

A 5-year retrospective observational clinical study was conducted in 92 cases of mammary carcinoma treated with the biological therapy [Di Bella Method (DBM)].
We observed remission/stability/progression performance status and 5-year survival rate for each stage in accordance with the criteria of the American Joint Committee on Cancer Staging (7th edition).
For each stage we observed better results in comparison with the data found in the literature.
30 other cases were also examined by experts and certified by the Court of Lecce (Italy).

Results

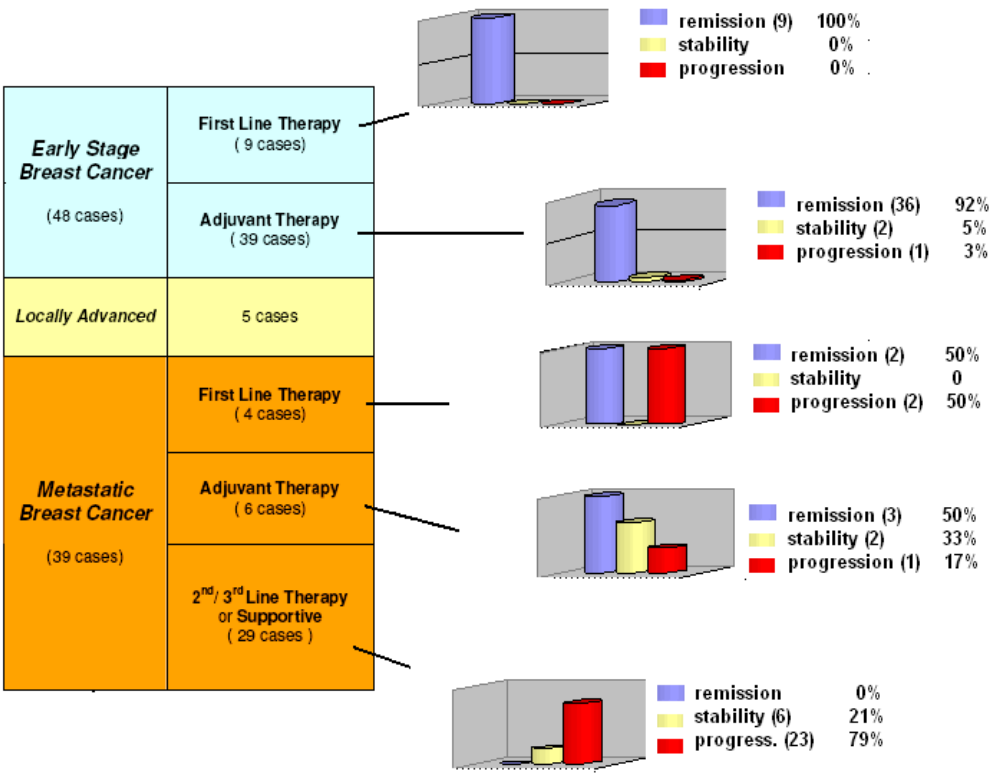
All of the 9 cases at stages I – II and III/a who had not undergone any previous drug or surgical treatment showed a favourable response, with 4 complete stable objective responses and 5 progressive appreciable reductions in tumor mass.
Following the "DBM" treatment, results are considerably better if compared with those of the statistics shown in the literature regarding survival rate, objective response and performance status for the same stages, histotypes and gradings. We found a 50% 5-year survival rate for stage IV patients treated with the "DBM" vs 19.9% as divulged by the SEER Project of the National Cancer Institute for the period 1999-2006.

Drugs

DBM employs such antiproliferating and antiangiogenic molecules as prolactin inhibitors and somatostatin; differentiating, apoptotic and antiangiogenic agents as retinoids, vitamin E, melatonin, vitamin D3, vitamin C; calcium, chondroitin sulfate, calcium levofolinate with purpose of differentiation; estrogen inhibitors and minimal, apoptotic doses of cyclophosphamide or oncocarbide

Conclusions

Following the "DBM" treatment, results are considerably better if compared with those of the statistics shown in the literature.



References

1. Ben-Jonathan N, et al. Prolactin as an autocrine/paracrine growth factor in human cancer. Trends Endocrinol Metab. 2002; 13(6):245-250.
2. Cucina A. Evidence for a biphasic apoptotic pathway induced by melatonin in MCF-7 breast cancer cells. J Pineal Res. 2009 Mar;46(2):172-80.
3. De Souza I. et al. Growth-hormone dependence among human breast cancers. Lancet 1974; 2(7874):182-184.
4. Di Bella G. The Di Bella Method (DBM). Neuro Endocrinol Lett. 2010;31 Suppl 1:1-42. Review.
5. Gruszka A, Pawlikowski M, Kunert-Radek J. Anti-tumoral action of octreotide and bromocriptine on the experimental rat prolactinoma: anti-proliferative and pro-apoptotic effects. Neuro Endocrinol Lett. 2001; 22(5): 343-348.
6. Hsieh TC. - Anticancer Res. 2010 Oct;30(10):4169-76. Differential suppression of proliferation in MCF-7 and MDA- MB-231 breast cancer cells exposed to alpha-, gamma- and delta-tocotrienols is accompanied by altered expression of oxidative stress modulatory enzymes.
7. Kleinberg DL. Pasireotide, an IGF-I action inhibitor, prevents growth hormone and estradiol-induced mammary hyperplasia. Pituitary. 2011 Mar;14(1):44-52.
8. Manni A. et al. Endocrine effects of combined somatostatin analog and bromocriptine therapy in women with advanced breast cancer. Breast Cancer Res Treat. 1989; 14(3): 289-298.
9. Pollak M. The potential role of somatostatin analogues in breast cancer treatment. Yale J Biol Med. 1997; 70(5-6): 535-539.
10. Wennbo. The role of prolactin and growth hormone in breast cancer. Oncogene. 2000 Feb 21;19(8):1072-6.
11. Xu. Growth hormone signaling in human T47D breast cancer cells: potential role for a growth hormone receptor-prolactin receptor complex. Mol Endocrinol. 2011 Apr;25(4):597-610