et al reported two cases with cutaneous metastasis from ES.[9] As with any other cancer, metastasis to skin indicates a poor prognosis. We are reporting one case of Ewing's sarcoma which developed cutaneous metastasis, which is extremely uncommon.

About 30% of soft tissue sarcomas are associated with chromosomal translocations which involve the exchange of genetic material between two chromosomes. This leads to formation of chimeric fusion genes that alter cellular function, often dysregulating proliferation, and resulting in clinically and genetically distinct neoplasms. A variety of genes can be involved, but the reasons for specific gene recombinations are unclear. However, secondary genetic and epigenetic events probably contribute to oncogenesis because gene expression patterns can vary significantly between different tumours with the same fusion. The Ewing's sarcoma breakpoint region 1 gene (EWSR1, known previously as EWS) which maps on chromosome 22q12 has been identified as a translocation partner in a wide range of clinically and pathologically diverse tumours. These include the Ewing family of tumours, desmoplastic small round cell tumour, myxoid liposarcomas, extraskeletal myxoidchondrosarcoma, angiomatoid fibrous histiocytoma, clear cell sarcoma of soft tissue and clear cell sarcoma-like tumours of the gastrointestinal tract, primary pulmonary myxoid sarcoma, myoepithelialtumours of skin, soft tissue and bone, and rare examples of low-grade fibromyxoid sarcoma, sclerosing epithelioid fibrosarcoma, and mesothelioma. EWSR1 rearrangements have also been found in some non-mesenchymal tumours such as hyalinising clear cell carcinoma of the salivary gland and hidradenoma of the skin.

The possibility to detect these karyotypic abnormalities by the means of chromosome analysis as well as by molecular genetics on a routine basis has greatly increased diagnostic accuracy. Although most cases of ES present as localized disease, overt metastases are capable of developing rapidly. Microscopic metastatic disease has been postulated to be present at the time of presentation. However, its spread is held in check by unidentified factors secreted by the primary tumor. When the primary tumor is removed or irradiated, the loss of the putative suppressive factors may permit the metastasis to grow. The use of chemotherapy in conjunction with surgery or radiation therapy to treat presumed metastatic disease has substantially improved survival [12]. Beyond specific clinical trials, patients with metastatic disease receive similar therapy to that administered for localized disease, with appropriate local treatment of metastasis, usually radiotherapy. Certain studies have suggested benefit from intensive chemotherapy followed by autologous stem cell rescue, but randomized trials have not yet been performed and the benefit of stem cell transplant remains unproven. Patients with recurrent disease fare poorly, with 5-year survival rates of less than 20% [11]. Current studies show that, following achieving remission in patients with non-metastatic ES, 30–40% of these patients are likely to develop recurrence of local or metastatic disease [13]. The majority of these studies report a time range of 2–10 years between commencing treatment and development of recurrence [14]. Patients relapsing later than 2 years from initial diagnosis have more favorable outcomes [11].