

Introduction Spontaneous abortion is a common complication in human reproduction. There is a paucity of epidemiological evidence pertaining to the risk factors for embryonic chromosomal aberrations in cases of spontaneous abortion. Most clinical miscarriages occur during the first trimester, and the leading cause of miscarriage is embryonal chromosomal abnormalities. Although parental chromosomal structure abnormalities are the main genetic factor leading to recurrent miscarriages, the prevalence of chromosomal abnormalities in the affected couples is relatively low (2.78–4.1%). Apart from inheritable factors, a variety of maternal factors have been found to be related to spontaneous abortion or embryonic chromosomal aberrations, including age, reproductive history, and immune or endocrine dysfunction [1–3, 9]. An estimated 60% of spontaneous abortions occur before or after implantation (termed preclinical losses), while 10–15% are confirmed by ultrasound or histological evidence (termed clinical miscarriage). Polymorphisms in folate metabolizing genes are associated with chromosome breaks and fetal chromosomal aneuploidy [13, 14]. The aim of this study was to assess the risk factors for spontaneous abortions with and without embryonic chromosomal aberrations by investigating the clinical and demographic characteristics of these cases. Therefore, exploring the etiopathogenesis of spontaneous abortion may help inform interventions to protect the developing embryo and prevent miscarriage. Apart from maternal factors, embryonic chromosomal abnormalities may be attributable to abnormal gametogenesis in father. Other non-hereditary factors implicated in embryonic chromosomal aberrations may include environmental factors. Supplementation of folic acid can decrease the concentration of homocysteine and reduce the risk of pregnancy loss [11, 12]. Therefore, advanced paternal age may be associated with miscarriage, infertility, and birth defects [18]. Prenatal exposure to environmental factors, such as drugs and pesticides, may increase the risk of birth defects or embryonic DNA damage [19, 20]. Few studies have investigated the parental karyotype of sporadic miscarriage [3]. In addition, elevated maternal serum level of homocysteine was shown to increase the risk of fetal loss and stillbirth [10]. Sperm DNA fragmentation index tends to increase with paternal age [15, 16]. While several causes of teratogenesis have been identified, the etiopathogenetic mechanisms are not well characterized [20–22]. The frequency of balanced rearrangements in the general population is very low (0.4%) [1, 2, 8]. Patients with high levels of sperm DNA damage showed a significantly higher miscarriage rate [17].