Anomalies of Corpus Callosum and Septum Pellucidum

2

Soon after closure, the cephalic end of the neural tube expands to form three primary vesicles, prosencephalon, mesencephalon and rhombencephalon. They are the progenitors of the forebrain, midbrain and hindbrain, respectively. The prosencephalon, during 4–10 weeks of gestation, develops by the process of ventral induction which includes formation, cleavage and midline development. Failure of formation results in aprosencephaly or atelencephaly. Total or partial failure of cleavage results in the spectrum of holoprosencephaly. Abnormal midline development results in agenesis of CC or septal agenesis. Holoprosencephaly is dealt with in Chap. 3. Anomalies of the CC and septum pellucidum will be described in this chapter.

The corpus callosum (CC) is the largest commissure connecting the two cerebral hemispheres. It is a broad plate made up of tightly packed axonal fibres crossing from side to side. In the midsagittal section, the corpus callosum extends from the frontal region anteriorly to overlie the tectum or quadrigeminal plate posteriorly.

The segments of the CC rostrocaudally are the rostrum, genu, body and splenium. The CC begins to develop at 12 weeks in the region of the genu and progresses posteriorly, forming the body and splenium. The rostrum is the last segment to develop. The corpus callosal development is complete by 18–20 weeks gestational age.

Total absence of corpus callosum (complete agenesis of corpus callosum — CACC) is due to primary embryologic failure. The axons which should have constituted the CC are oriented anteroposteriorly to form a tract medial to the lateral ventricle on either side. These white matter tracts, Probst bundles, medially indent the lateral ventricle.

Incomplete development of the CC results in varying degrees of corpus callosal shortening. The splenium, body or rostrum may be absent. This is termed partial agenesis of the CC (PACC). The term hypoplasia of CC refers to a CC which is normal in length but thin. Corpus callosal dysgenesis refers to a CC which is thinner or thicker than normal or partially developed. CACC or PACC can also be secondary to destructive processes such as ischaemia or infection.

Table 2.1 The relationship between the CSP and CC

CSP	CC	Condition
Normal	Present	Normal
Absent	Absent	CACC
Absent	Present	Septal agenesis
Abnormal	Abnormal	PACC, dysgenesis
Normal	Absent	Not possible

The septum pellucidum is a two-layered membrane between the anterior horns of the lateral ventricles. The two layers in the fetus are separated by fluid. Hence, it is termed cavum septum pellucidum (CSP). The development of the CSP is closely related to and dependent on the development of the CC. A normal CC is associated with a normal-sized CSP. The length of the CSP (anteroposterior dimension) is directly proportional to the length of the CC. A short CC as in PACC is associated with a CSP of short length. CSP is absent in CACC. Presence of CC with absence of CSP occurs in septal agenesis. Hence, a CSP seen (on axial sections) indirectly indicates the presence of CC (on midsagittal section). As the CC development is complete by 18–20 weeks, CSP can be identified only after this gestational age. The CSP is not visualised after 37 weeks, as the fluid in the cavum is absorbed. The CSP then becomes the septum pellucidum as seen postnatally (Table 2.1).

2.1 Complete Agenesis of Corpus Callosum

US findings in the axial, coronal or sagittal sections which help suspect CACC are the indirect signs. The indirect signs on the axial sections are important and serve as initial clues that lead to a detailed “neurosonogram”. The direct sign is the inability to visualize CC on midsagittal or coronal sections. These signs are best seen at or after 22 weeks. Although present, the signs are subtle and may be missed at 18–22 weeks.

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2.1.1 Indirect Signs in the Transventricular Axial Section

1. The CSP is absent. It must be emphasized that CACC cannot be diagnosed only on the basis of absent CSP (Figs. 2.1, 2.2 and 2.3a). One must directly demonstrate the absence of CC on midsagittal sections.
2. Teardrop-shaped lateral ventricle is a combination of colpocephaly (selective mild dilatation of the atria and posterior horns of the lateral ventricles) with pinched anterior horns (Figs. 2.1, 2.2 and 2.3a).
3. The anterior horns are laterally placed (Figs. 2.1, 2.3a and 2.4).
4. Cephalad extension of the third ventricle to extend up to the level of the lateral ventricles (Fig. 2.1).
5. The interhemispheric fissure (IHF) is wide with CSF separating the medial surfaces of the cerebral hemispheres from the falx as there is no CC to hold them together. This is called the "three line" sign (Figs. 2.1 and 2.4).

2.1.2 Indirect Signs on Transcaudate Coronal Section

1. The CSP is absent and hence the IHF is uninterrupted (Fig. 2.3a).
2. The anterior horns are laterally placed (Fig. 2.3a).
3. The anterior horns are crescent or comma shaped with an outward convexity ("steerhorn" or "Viking helmet" sign). The medial indentation of the lateral ventricles is due to the Probst bundles (Figs. 2.3a and 2.5).

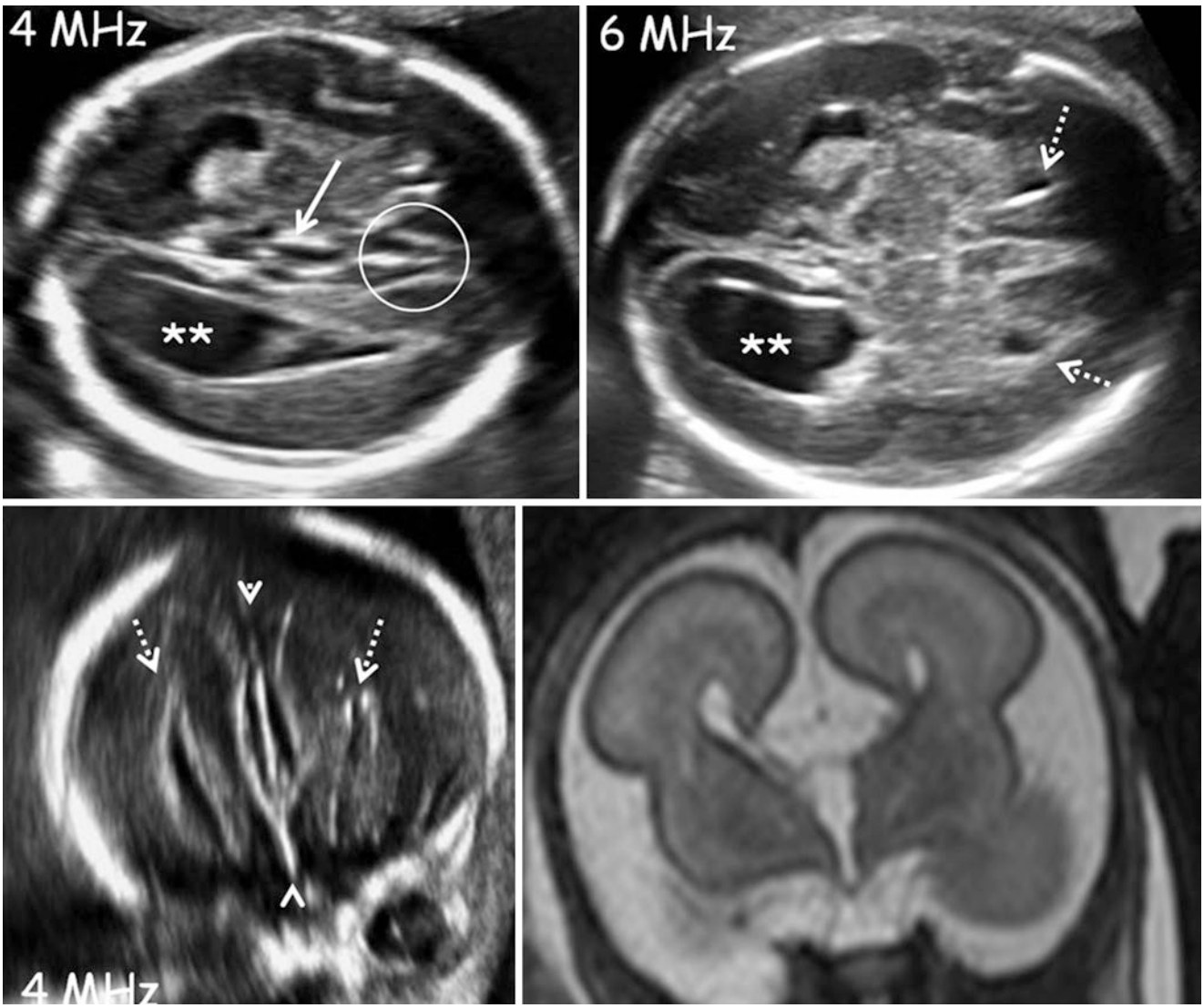


Fig. 2.1 24 weeks (TAS and MRI) *indirect signs of complete agenesis of corpus callosum*. Axial transventricular and transthalamic, coronal transfrontal section and T2W MR coronal transthalamic section – absent CSP on the axial sections, CC not seen in the coronal MR section, three-line sign seen of IHF (circled), colpocephaly/teardrop shape of lateral ventricle (**), laterally placed anterior horns which are steerhorn in shape (dotted arrows), cephalad extension of the third ventricle (solid arrow), uninterrupted IHF (arrowheads)

4. Wide IHF ("three line" sign) is seen (Figs. 2.1 and 2.3a).
5. Cephalad extension of the third ventricle is seen in the coronal transthalamic section.
6. Prominent anterior commissure may be seen (Fig. 2.6).

2.1.3 Indirect Signs on the Midsagittal Section

1. The cingulate sulcus and gyrus are absent (Figs. 2.3a, b and 2.4).
2. The presence of radiating medial hemispheric sulci and gyri is typically seen in the third trimester ("sunburst" sign) (Fig. 2.7).
3. Abnormal course of the pericallosal artery is seen on color Doppler (Fig. 2.8).

2.1.4 Direct Sign on Midsagittal, Transcaudate and Transthalamic Coronal Sections

The corpus callosum is not seen (Figs. 2.2, 2.3a, b, 2.4, 2.7 and 2.8).

2.1.5 Associated Intracranial Findings

1. The roof of third ventricle (tela choroidea) balloons out dorsally with CSF to form an interhemispheric cyst. It may extend on any one side of the falx cerebri (Fig. 2.9).
2. A nodular lipoma, when present, is seen as a focal hyperechoic lesion in the anterior midline just under the IHF (anatomical location of the genu). It may extend through the choroidal fissures on either side to reach the choroid plexuses in the lateral ventricles. Very rarely a ribbon or curvilinear lipoma may extend along the course of the missing CC (Figs. 2.10 and 2.11).
3. CACC may be associated with lissencephaly, schizencephaly, heterotopia and polymicrogyria (Fig. 2.12).

2.1.6 Associated Extracranial Findings

1. Sporadic extracranial anomalies such as genitourinary, skeletal or congenital heart defects may be present.
2. CACC may be a part of a syndrome. CACC associated with dysmorphic face (hypotelorism, small nose, micrognathia, cleft lip) and polydactyly is suggestive of Acrocallosal syndrome (autosomal recessive) (Fig. 2.13) or Orofaciodigital syndrome (X-linked dominant). Aicardi syndrome (X-linked dominant) is suspected in a female fetus with CACC, polymicrogyria, heterotopia, microphthalmia, coloboma, hemivertebra, scoliosis and brain tumor. CACC can occur in inborn errors of metabolism. Diagnosis is by testing for the specific gene mutations.
3. Karyotyping is indicated to rule out chromosomal abnormalities.

2.1.7 Differential Diagnosis

Occasionally, a SP without fluid between its layers may be seen. Most often, the CC is normal in such cases (Fig. 2.14).

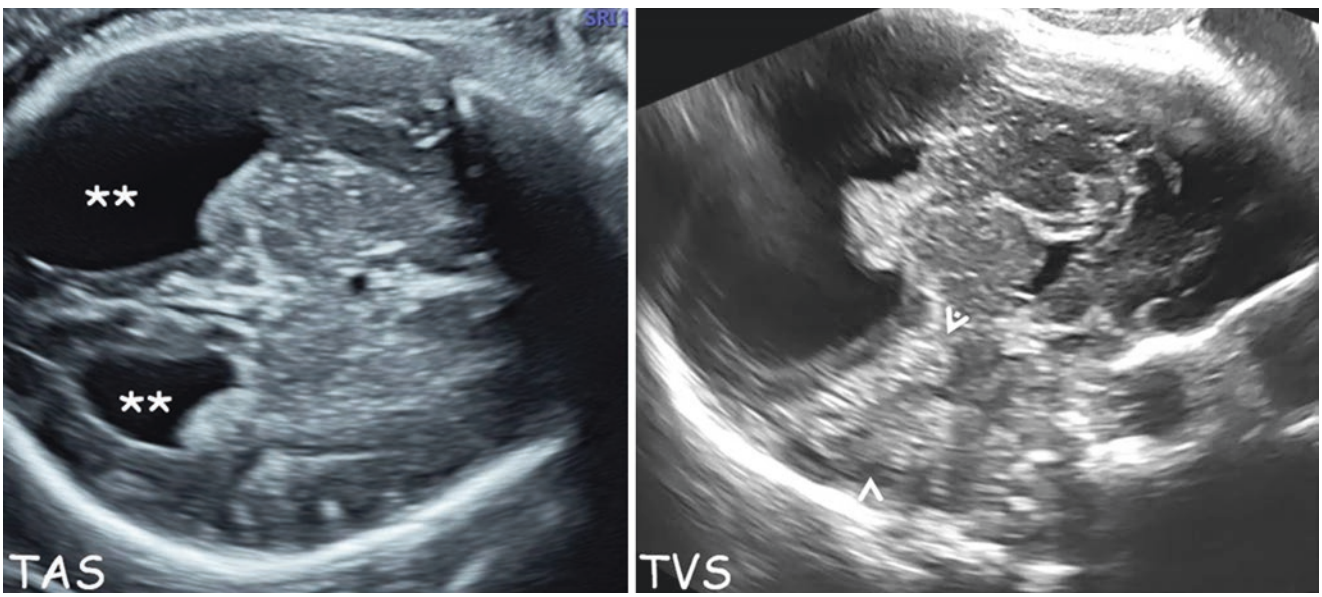


Fig. 2.2 33 weeks (TAS and TVS) *complete agenesis of corpus callosum.* Axial transthalamic and midsagittal sections – absent CSP, colpocephaly (**), vermis (arrowheads) confirms section to be midsagittal. The CC is absent

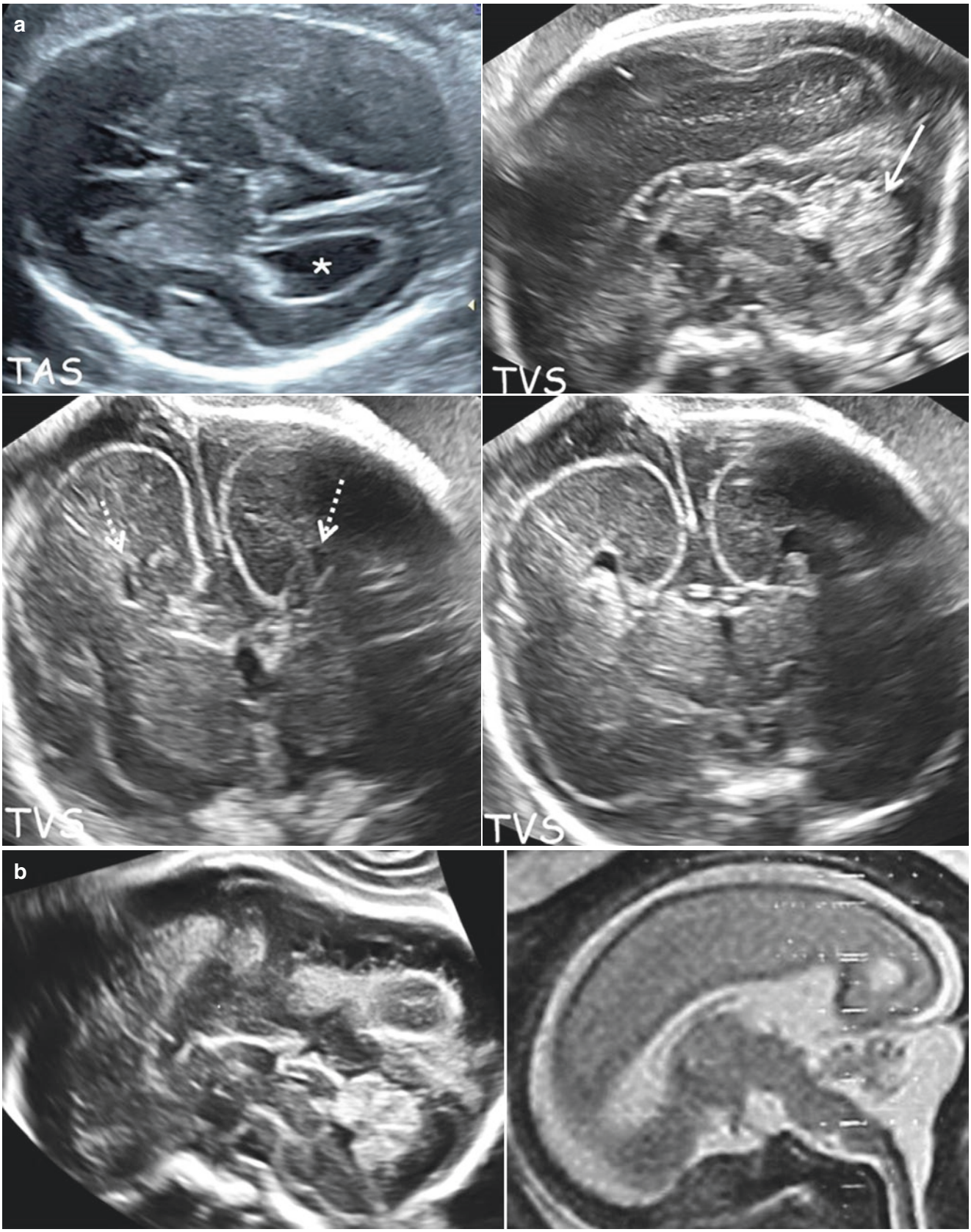


Fig. 2.3 (**a**) 27 weeks (TAS and TVS) *complete agenesis of corpus callosum* (*direct and indirect signs*). Axial transventricular, midsagittal and coronal transthalamic sections – CSP is absent on axial section; colpocephaly (*), corpus callosum and cingulate sulcus are absent on midsagittal and coronal sections; vermis (solid arrow); laterally placed, steerhorn-shaped lateral ventricles (dotted arrows). (**b**). 27 weeks (TVS 3D US and MRI) *complete agenesis of corpus callosum* (*direct sign*). 3D Multiplanar midsagittal section with volume contrast imaging (VCI) and T2W MR midsagittal section – the entire CC is not seen

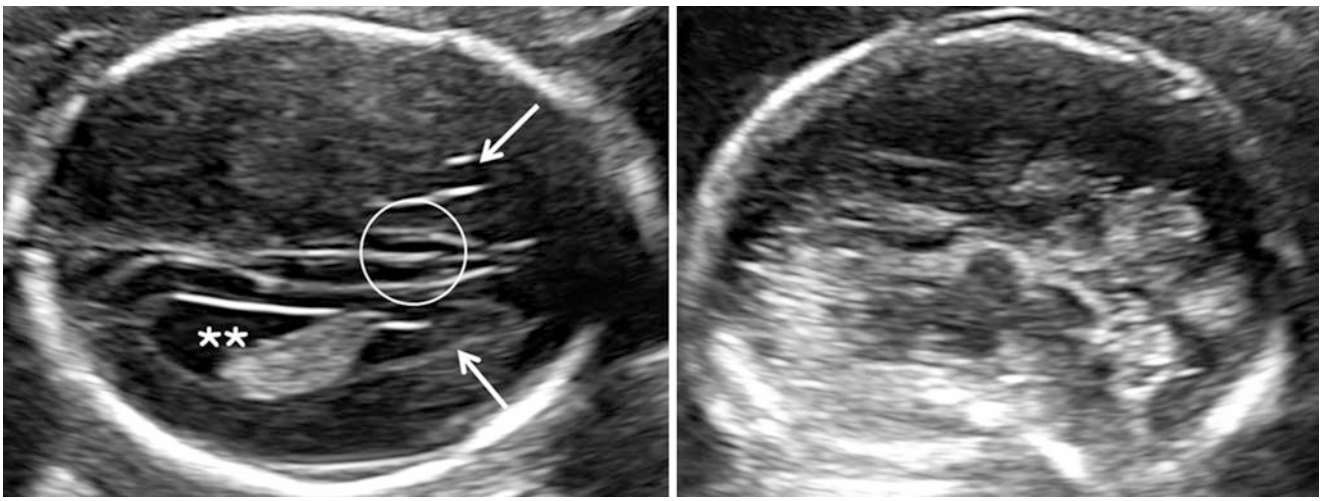


Fig. 2.4 19 weeks (TAS) *complete agenesis of corpus callosum* (*direct and indirect signs*). Axial transventricular and midsagittal sections – colpocephaly (**), pinched and laterally placed anterior horns (solid arrows), absent CSP and three line sign (circled), entire CC is absent on midsagittal section

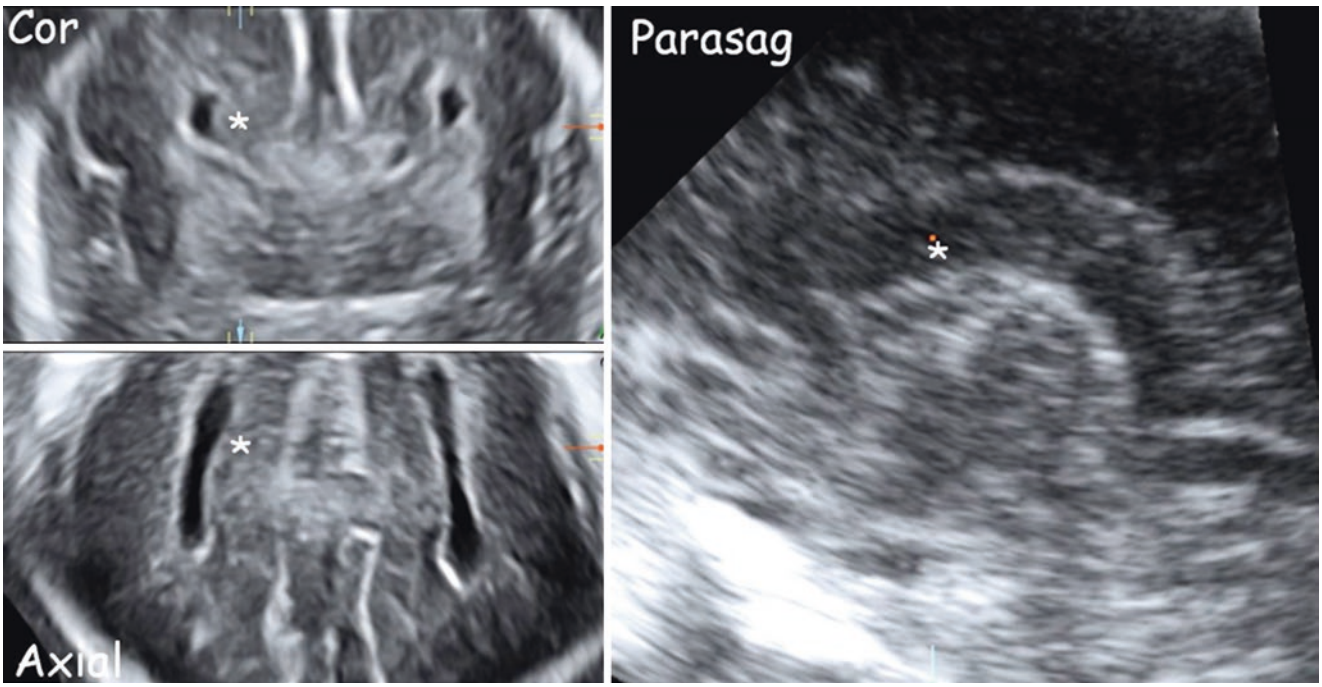


Fig. 2.5 35 weeks (TVS 3D US) *complete agenesis of corpus callosum* – 3D multiplanar coronal, axial and parasagittal sections – navigation dot (*) is placed on the Probst bundle

Absence of CSP is not unique to CACC. It is absent in holoprosencephaly (HPE), septal agenesis, schizencephaly and hydrocephaly. Absence of IHF and presence of monoventricle and dorsal sac confirm alobar HPE. Failure of cleavage of anterior horns results in an abnormal shape and is seen in semilobar and lobar HPE. The CC is not normal in these disorders. In septal agenesis, the shape of the anterior horns is normal in the coronal transcaudate section. They are, however, continuous with each other across the midline. The CC is normal in septal agenesis. The presence of full thickness cerebral parenchymal cleft distinguishes schizencephaly.

Mild lateral ventriculomegaly is a common finding seen in chromosomal abnormalities, malformation of cortical development, infections and other conditions. Colpocephaly in CACC has to be differentiated from mild lateral ventriculomegaly. Pinched appearance of the anterior horns (teardrop shape of the lateral ventricle) and the increased distance from the midline are the features of colpocephaly.

In the absence of associated intracranial, extracranial and karyotype abnormalities, CACC is considered to be isolated. MRI in addition to confirming the absence of CC will also confirm or rule out associated CNS anomalies. Prognostication depends on whether CACC is isolated or not.

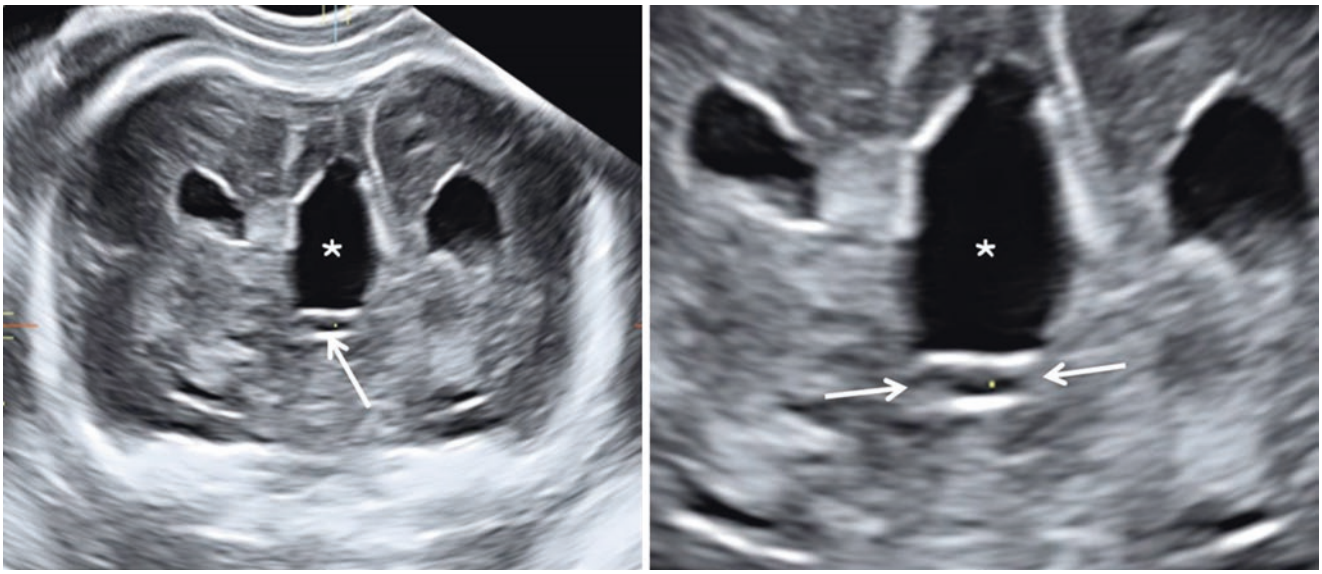


Fig. 2.6 24 weeks (TVS) *complete agenesis of corpus callosum with hypertrophy of the anterior commissure* – coronal transcaudate section and transcaudate section magnified – interhemispheric cyst (*), prominent anterior commissure inferior to the cyst anteriorly (solid arrows)

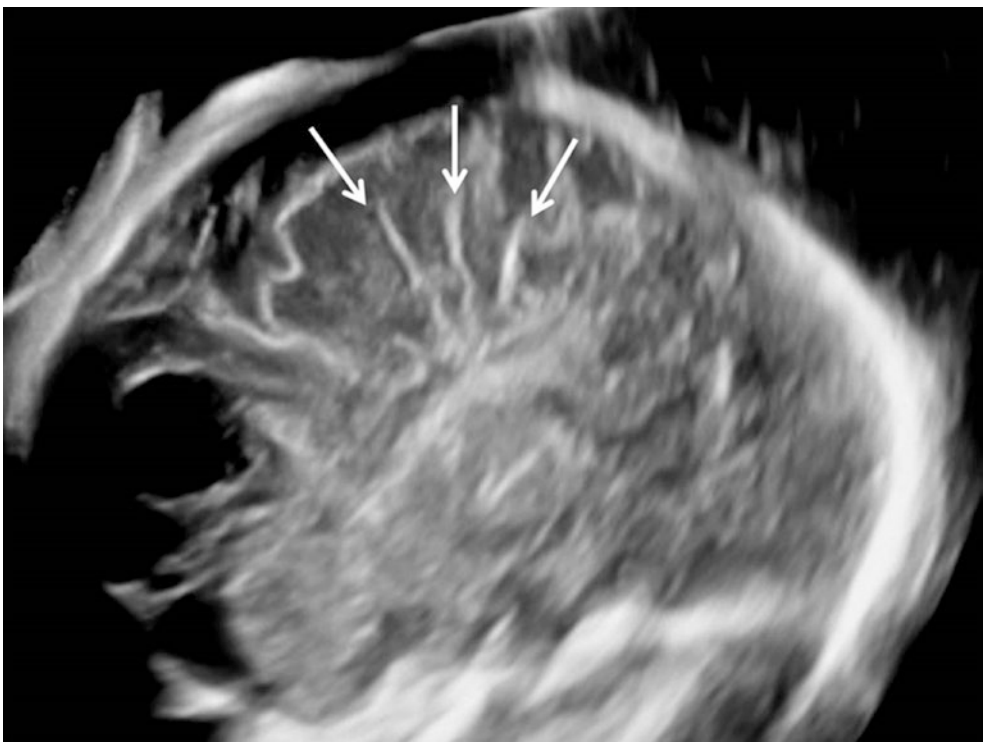


Fig. 2.7 31 weeks (TAS) *complete agenesis of corpus callosum* – midsagittal section – absent corpus callosum and cingulate sulcus with radially arranged medial hemispheric sulci (arrows)

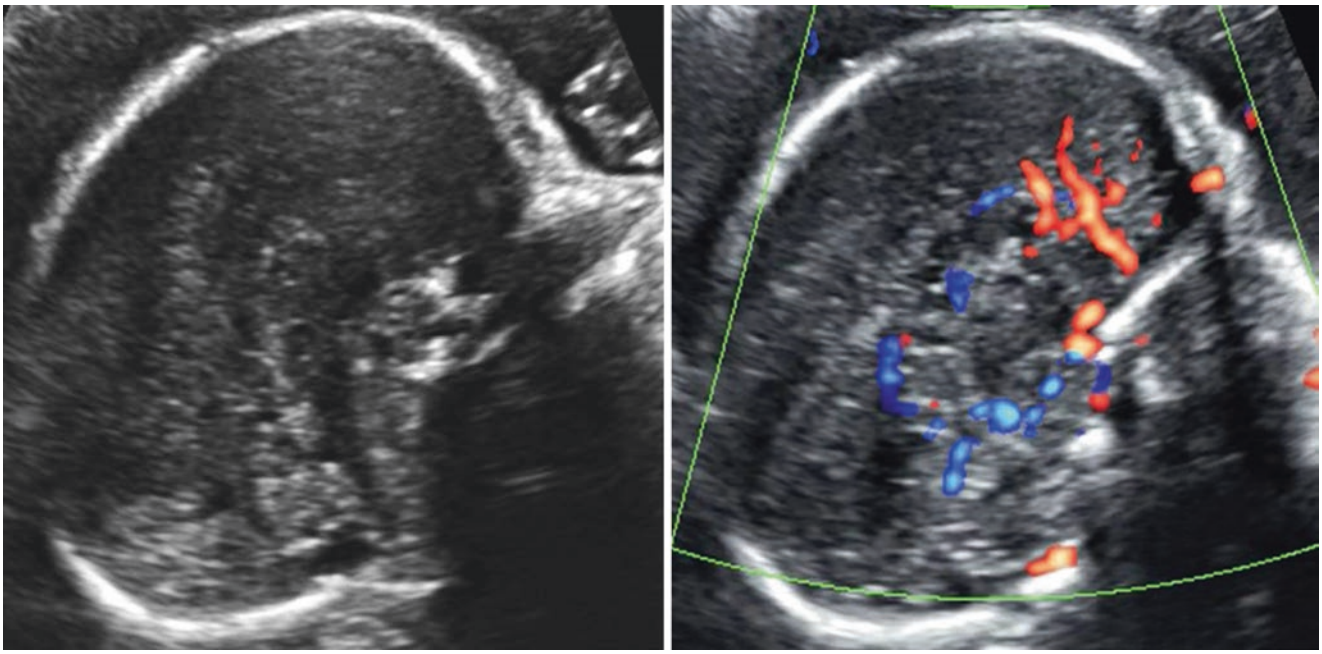


Fig. 2.8 25 weeks (TAS) *complete agenesis of corpus callosum* – midsagittal sections B mode and color Doppler – abnormal course of pericallosal artery

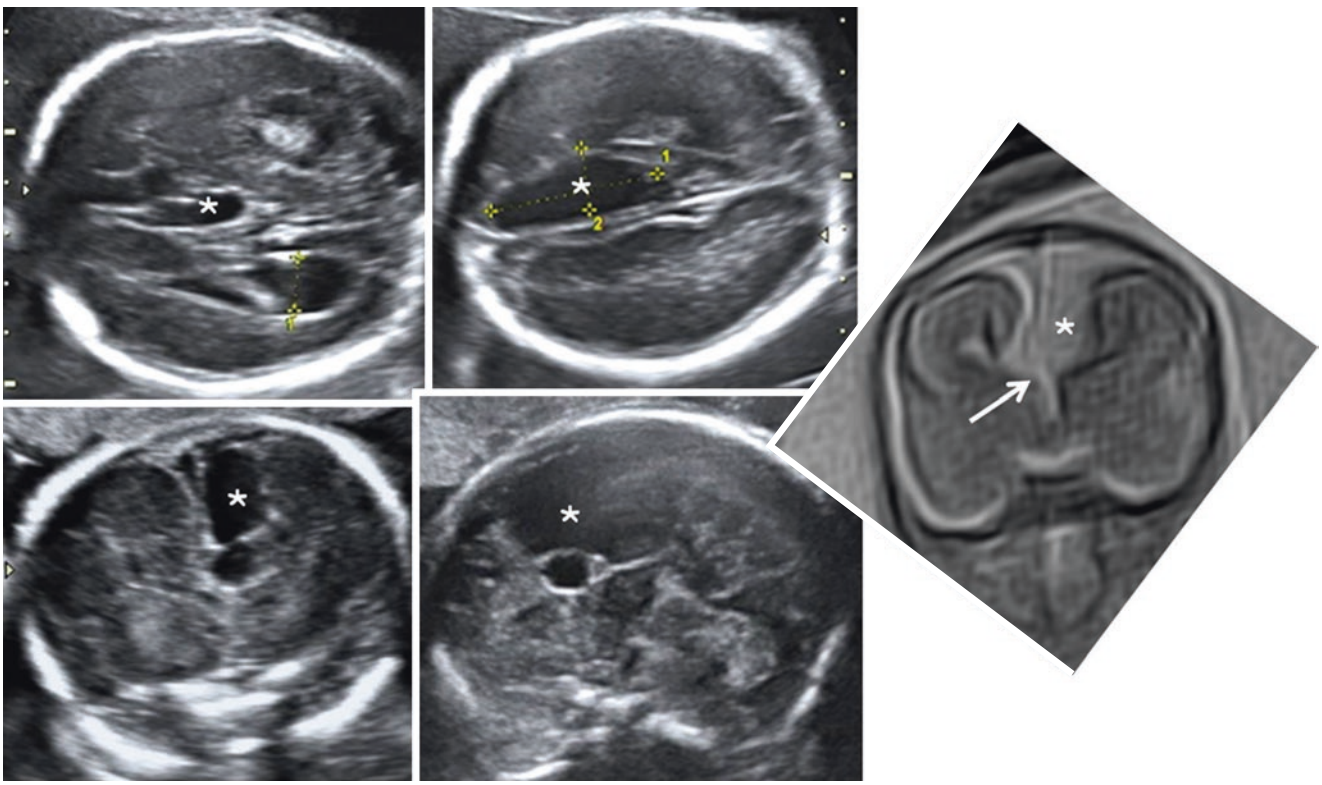


Fig. 2.9 26 weeks (TAS and MRI) *complete agenesis of corpus callosum with left interhemispheric cyst* – axial transventricular, axial section at a slightly cephalic plane, coronal transthalamic and midsagittal sections and T2W MR coronal transthalamic section – interhemispheric cyst extending to left of the falx cerebri (*), the cyst inferiorly is communicating with the third ventricle (solid arrow). The other indirect and direct signs of CACC are seen

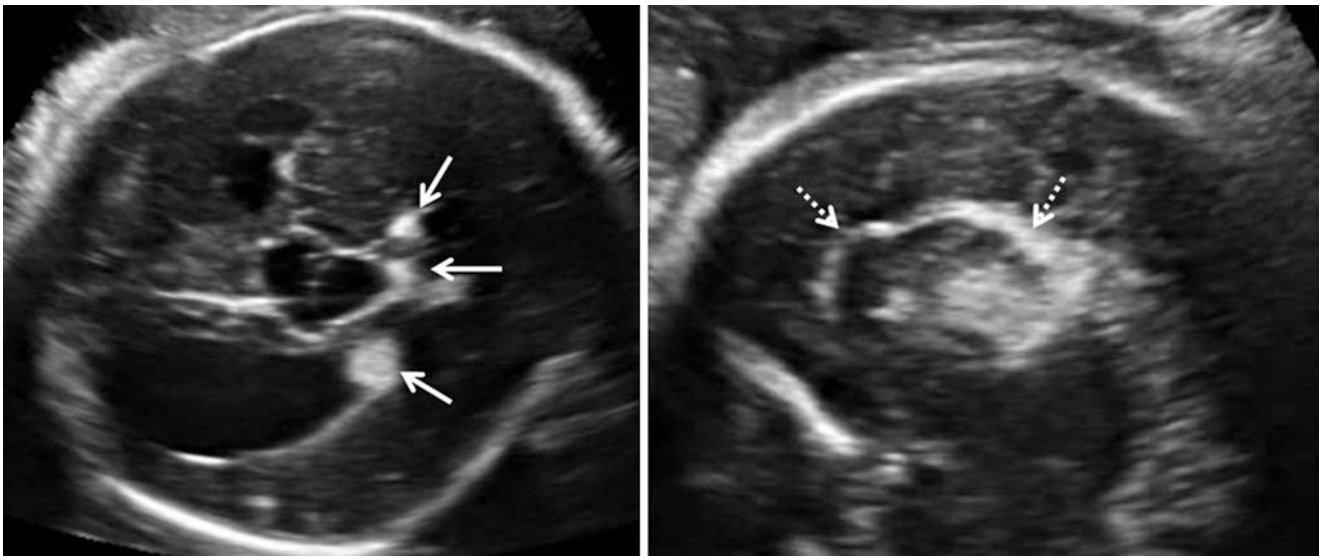


Fig. 2.10 36 weeks (TAS) *complete agenesis of corpus callosum with lipomas* – axial transventricular and midsagittal sections – midline and bilateral intraventricular lipomas (solid arrows), ribbon lipoma (dotted arrow) replaces CC

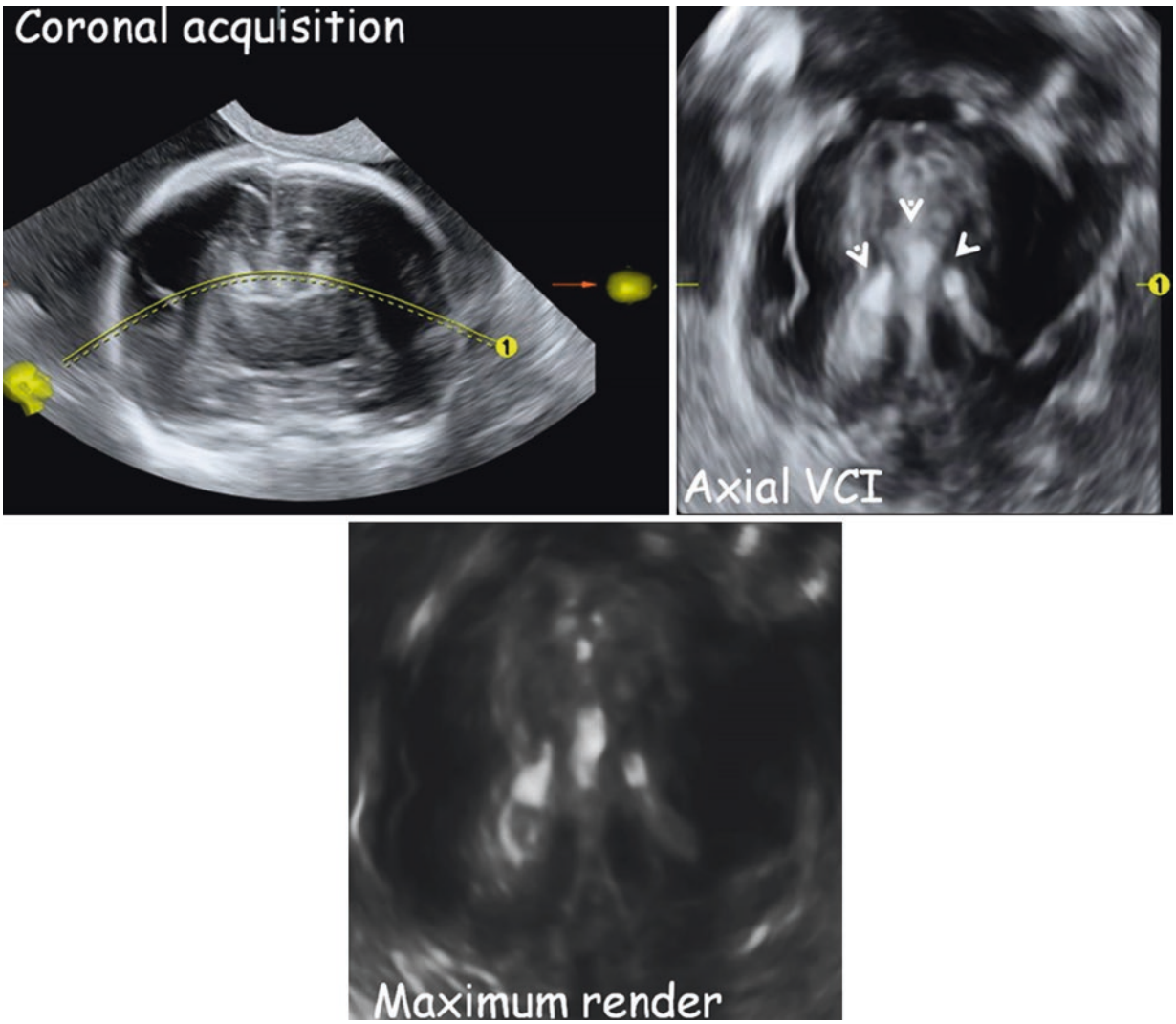


Fig. 2.11 25 weeks (TAS 3D US) *complete agenesis of corpus callosum with lipomas* – axial transventricular section obtained with 3D omniview and volume contrast imaging (VCI) and 3D rendered axial transventricular section – midline and bilateral intraventricular lipomas (arrowheads). The bilateral lipomas are seen tipping the choroid plexuses anteriorly

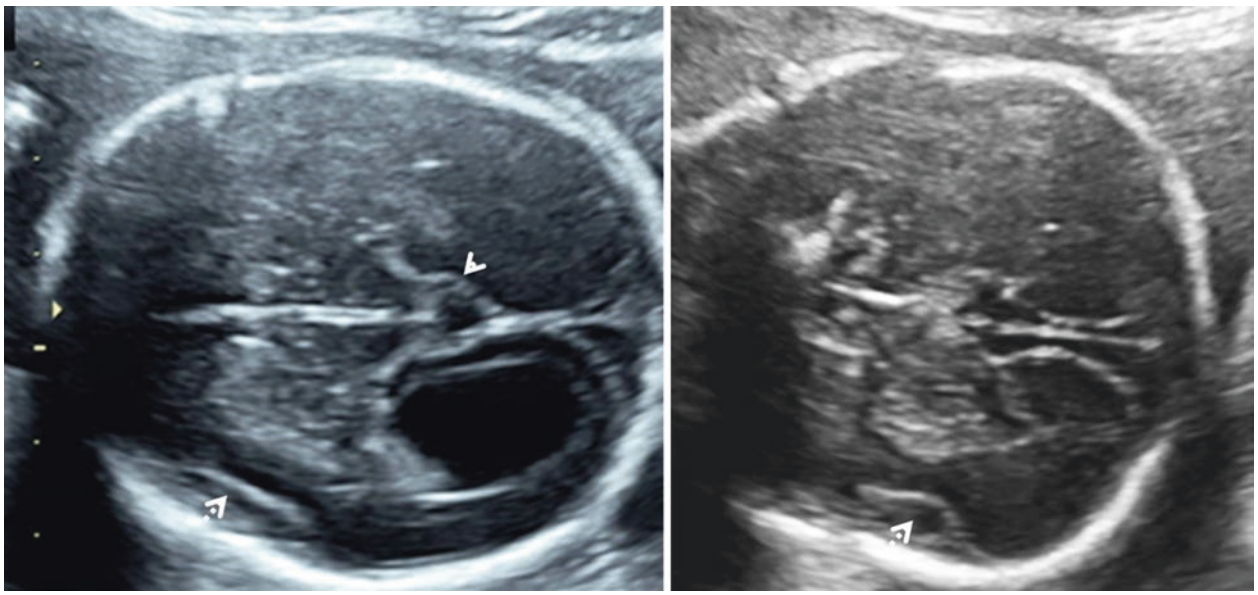


Fig. 2.12 24 weeks (TAS) *complete agenesis of corpus callosum with lissencephaly* – axial transventricular and coronal transcaudate sections – shallow parieto-occipital sulcus (arrowhead) and shallow lateral fissure (dotted arrows). Other direct and indirect signs of CACC are seen

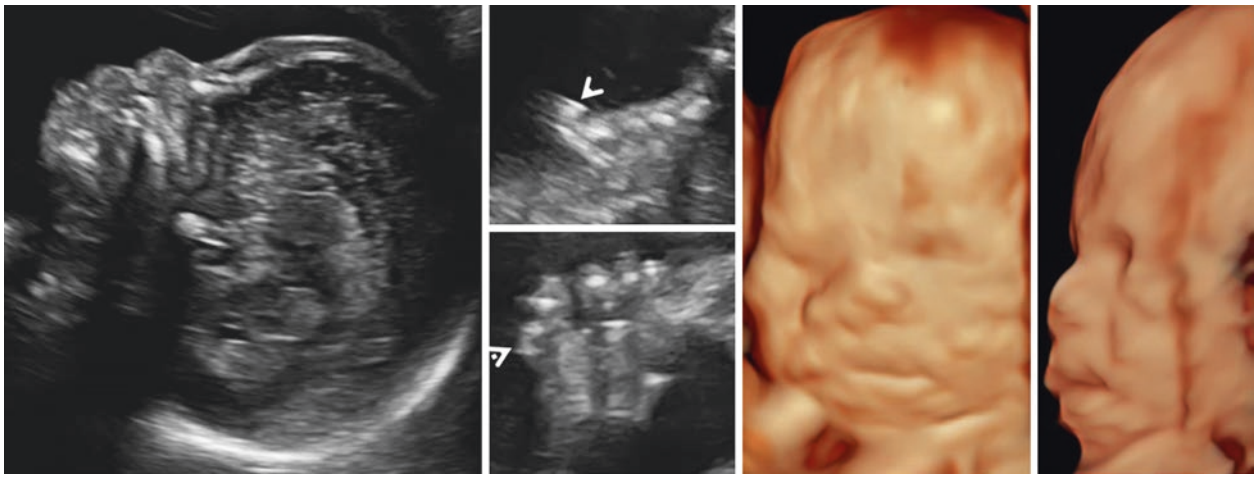


Fig. 2.13 23 weeks pregnancy in a second-degree consanguineous couple (TAS). *Acrocallosal syndrome* – midsagittal section of the cranium, coronal sections of both hands and 3D surface rendered images of the face – complete absence of CC in the midsagittal section, bilateral postaxial polydactyly (arrowheads) and facial dysmorphism

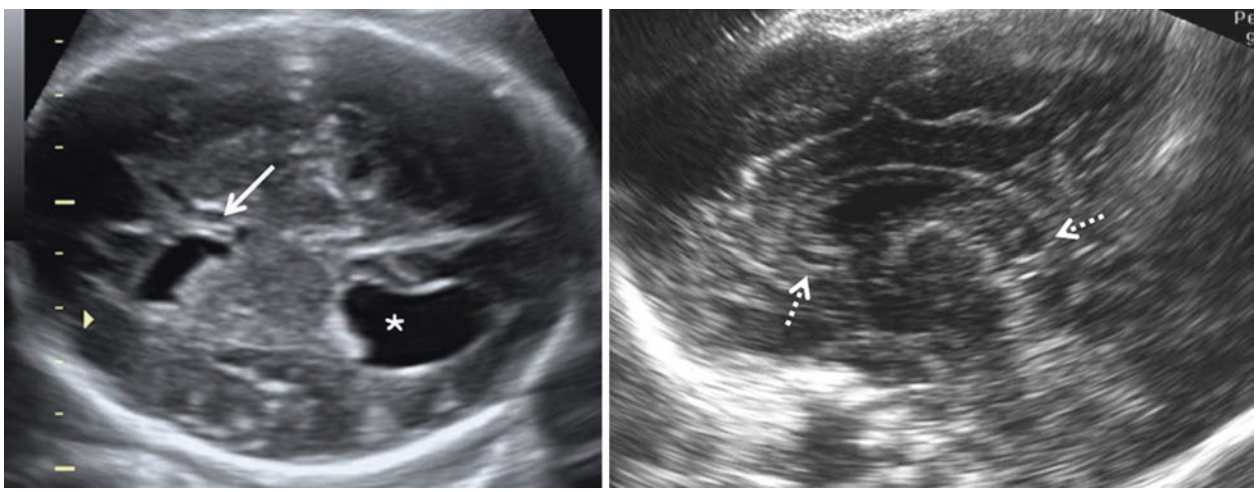


Fig. 2.14 32 weeks (TAS) *no fluid in the CSP with a normal CC* – axial transventricular and midsagittal sections – mild left lateral ventriculomegaly (*), no fluid in the CSP (solid arrow), normal CC (dotted arrows)

2.2 Partial Agenesis of Corpus Callosum

US findings in the axial, coronal or sagittal sections which help suspect PACC are the indirect signs. The indirect signs on the axial sections are important and serve as initial clues that lead to a detailed "neurosonogram". A CC of short length and inability to visualize all segments are direct signs seen on the midsagittal section.

2.2.1 Indirect Signs on Axial Sections

1. The length of the CSP is directly related to the length of the CC. Hence, the CSP is short in PACC. This serves as an indirect sign on axial sections. In severe PACC (only a small length of the CC is present), the CSP is very small and could even verge on absence. In less severe PACC (variable lengths of CC present), the CSP is short and wide. The normally rectangular CSP tends to be a square (Fig. 2.15).
2. The length of the CSP is measured from the callosal sulcus anteriorly to the fornices posteriorly. The width of the CSP is measured in its midportion ("on to on"). The length-to-width ratio is termed the CSP ratio. In a short and wide CSP (as in PACC), the ratio is less than 1.5 (Figs. 2.16, 2.17, 2.18a and 2.19).
3. Other indirect signs on axial view include colpocephaly (Fig. 2.18a) and teardrop shape of the lateral ventricle. These signs may be subtle.
4. Associated intracranial findings such as lissencephaly, polymicrogyria, heterotopias, interhemispheric cyst (Fig. 2.20a, b), midline lipoma (nodular or curvilinear types) and Dandy-Walker malformation may be present.

2.2.2 Direct Signs on Midsagittal Section

1. The length of the CC is lesser than the fifth percentile or −2SD (Fig. 2.18a, b).
2. In the absence of the splenium and posterior body, the CC does not extend posteriorly to overlie the quadrigeminal (tectal) plate of the midbrain (Figs. 2.16, 2.17, 2.18b, 2.19 and 2.20b).
3. In some cases the splenium and rostrum may be absent (Fig. 2.16). Absence of bulbosity of the splenium is a subtle sign of PACC.
4. The cingulate sulcus is seen parallel to the extant CC (Fig. 2.17). It is not seen in the regions where the CC is absent.
5. The medial hemispheric sulci are radially arranged in the regions where the CC (and hence the cingulate sulcus) is absent (Fig. 2.17).
6. The pericallosal artery sweep is present only over the segment of the CC that is present. The artery subsequently has an abnormal course (Fig. 2.19).

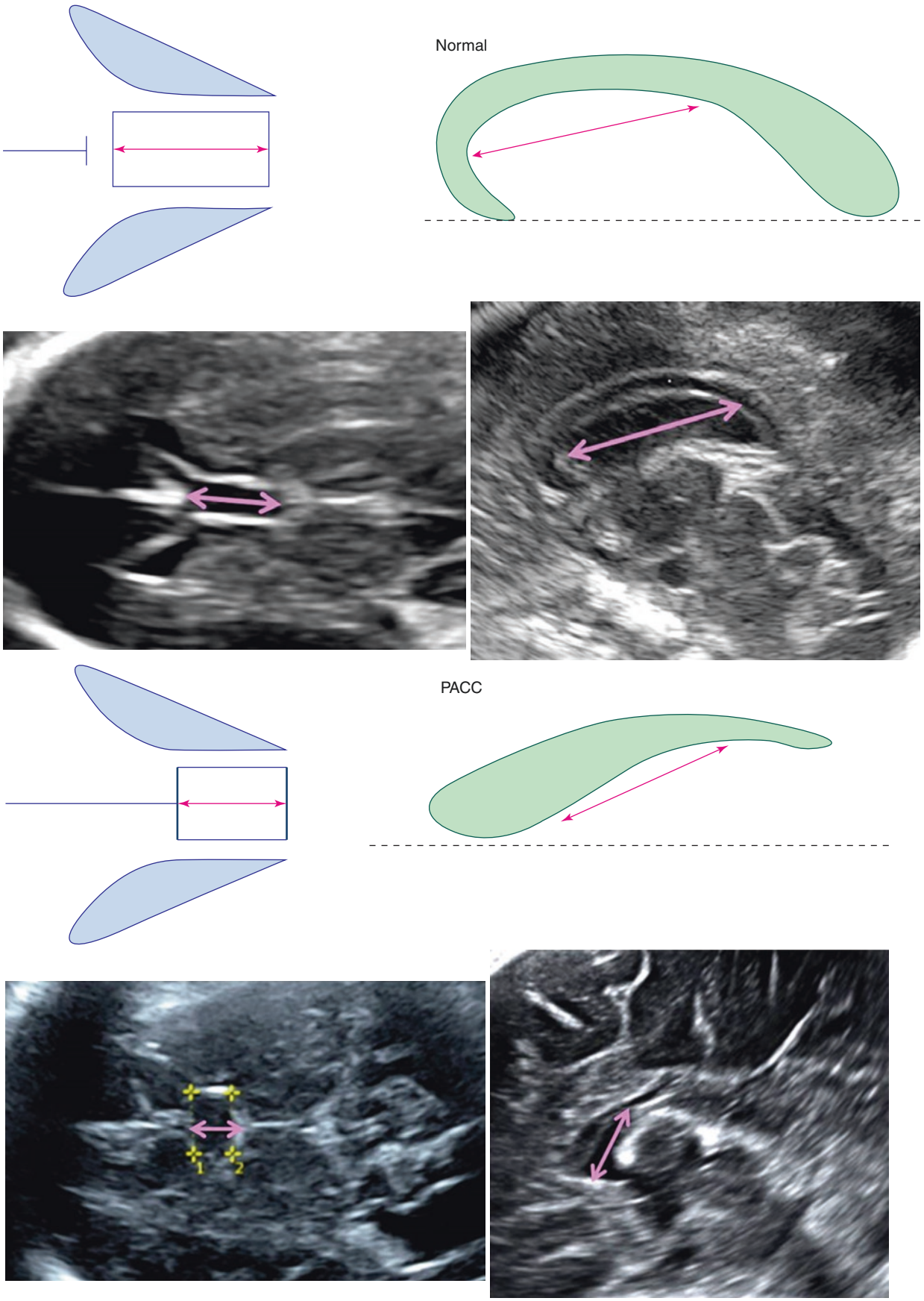


Fig. 2.15 Schematic diagram showing the relation of the length of the CSP (axial section) to the length of the CC (midsagittal section). The normally rectangular CSP reflects a CC of normal length. A short CSP reflects a CC of short length, as in partial agenesis of CC. This relationship is understandable given that the CSP is immediately subcallosal in location

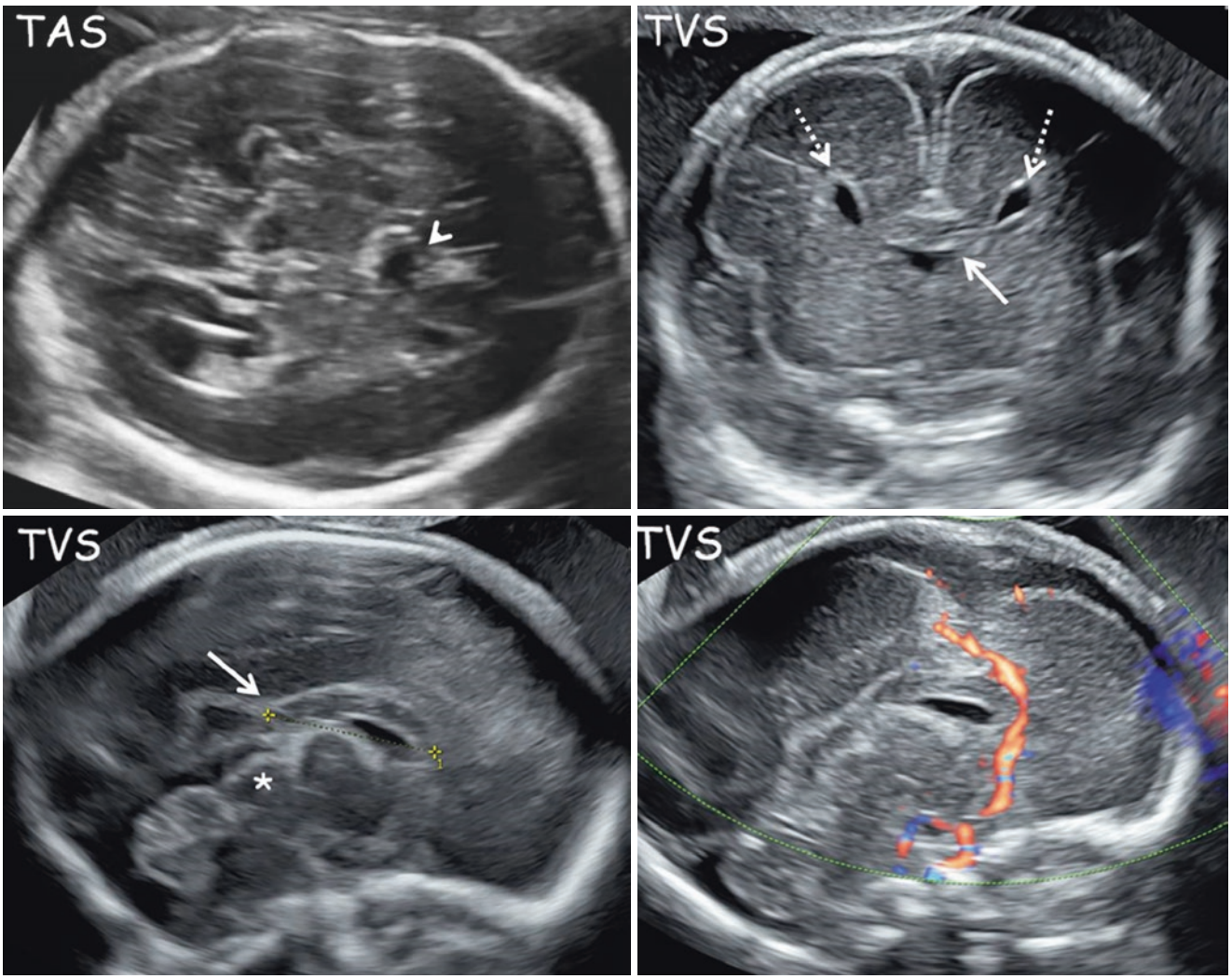


Fig. 2.16 24 weeks (TAS and TVS) *partial agenesis of corpus callosum* – axial transventricular, coronal transthalamic, midsagittal (without and with color Doppler) sections – short and wide CSP (arrowhead), short thick CC, splenium and rostrum are absent, body of CC is present, posterior limit of CC (solid arrow) does not overlie the quadrigeminal plate (*), dysmorphic lateral ventricles (dotted arrows), abnormal course of pericallosal artery

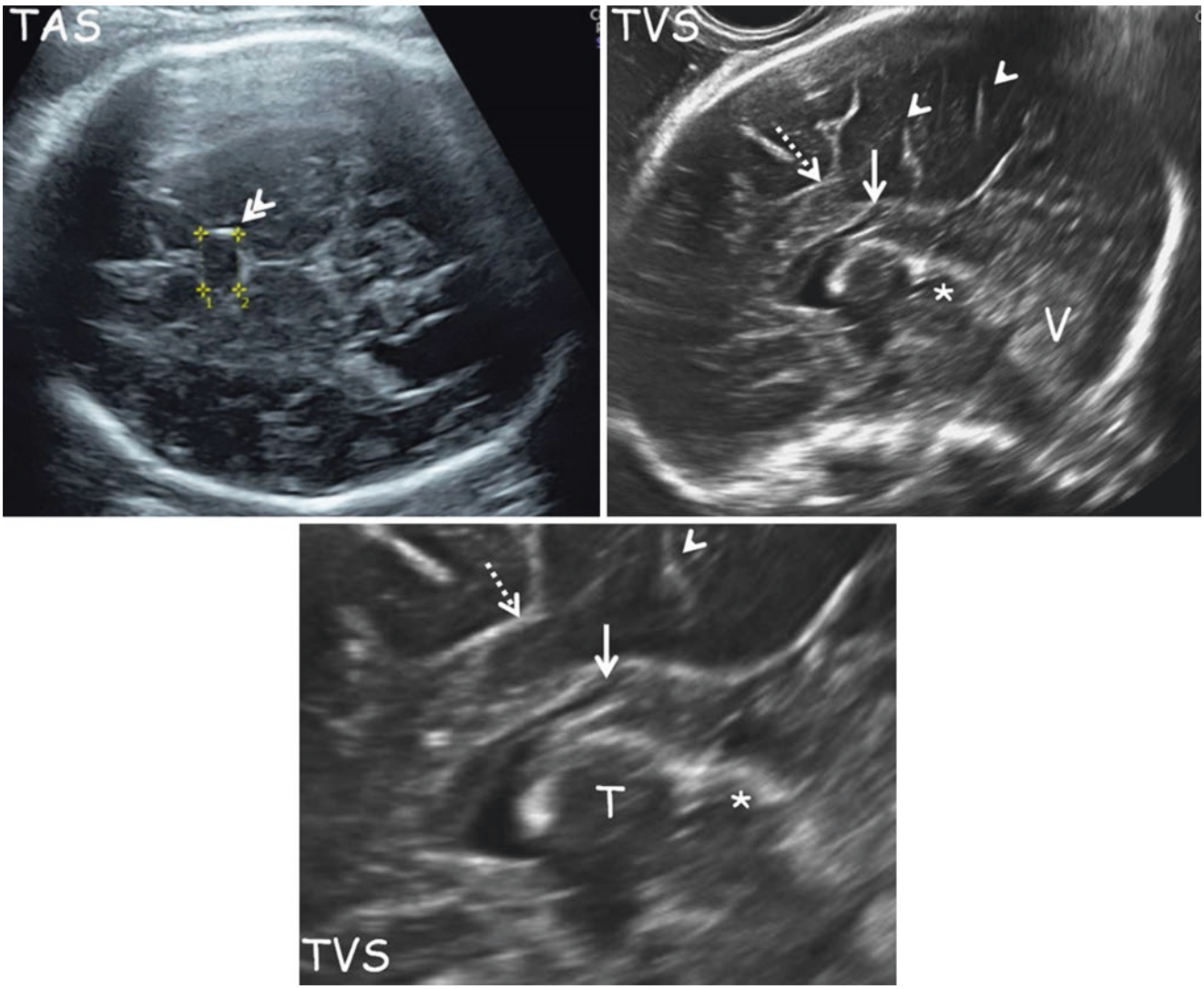
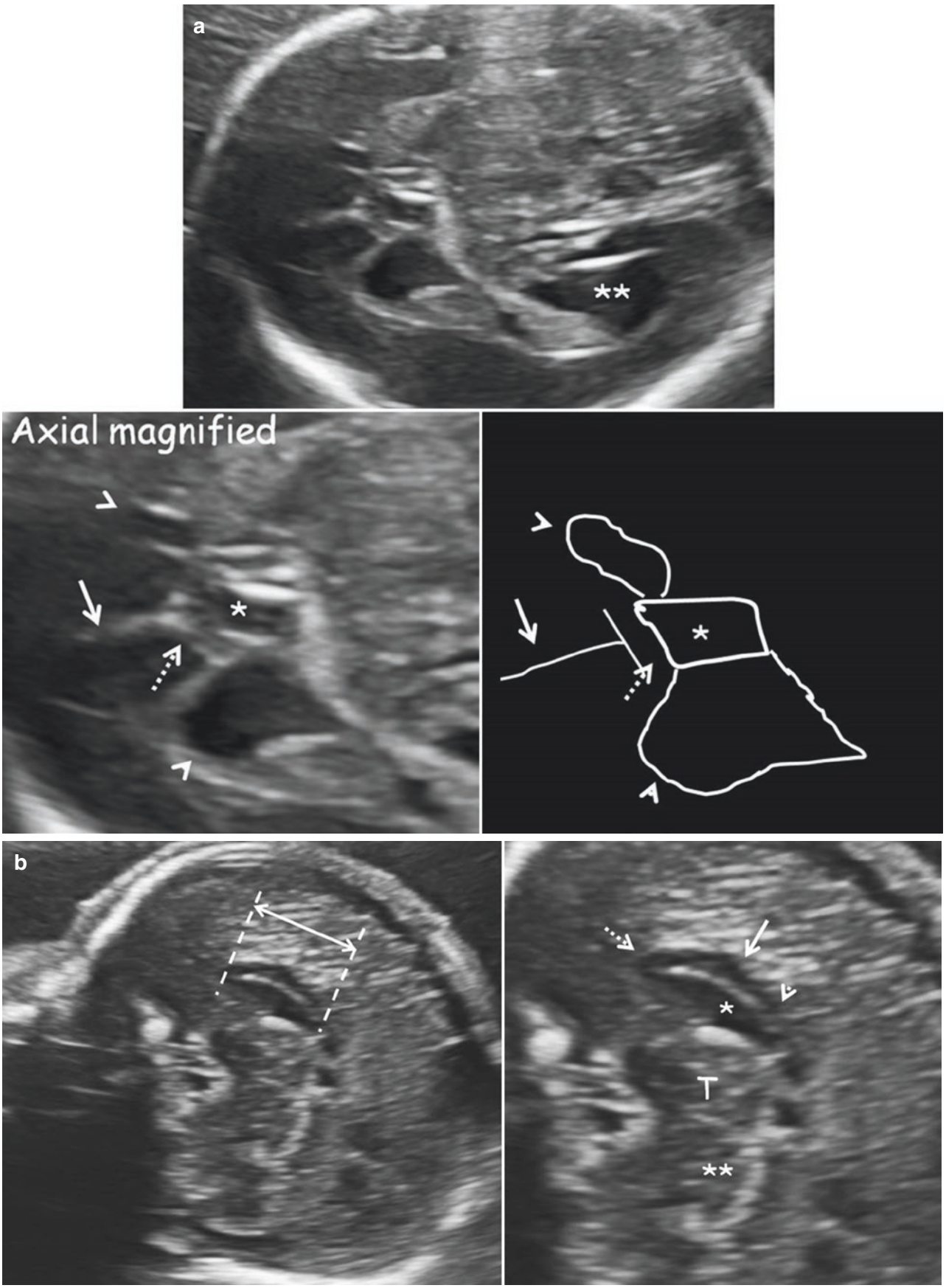


Fig. 2.17 32 weeks (TAS and TVS) *partial agenesis of corpus callosum* – axial transventricular, midsagittal and midsagittal magnified sections – short and wide CSP (double arrowhead), short CC, body of CC is thin posteriorly, splenium, rostrum and genu absent, posterior limit of the CC (solid arrow) does not extend to overlie the quadrigeminal plate (*), cingulate sulcus stops exactly where the CC ends (dotted arrow) beyond which a few radial sulci are seen (arrowheads), thalamus (T), vermis (V)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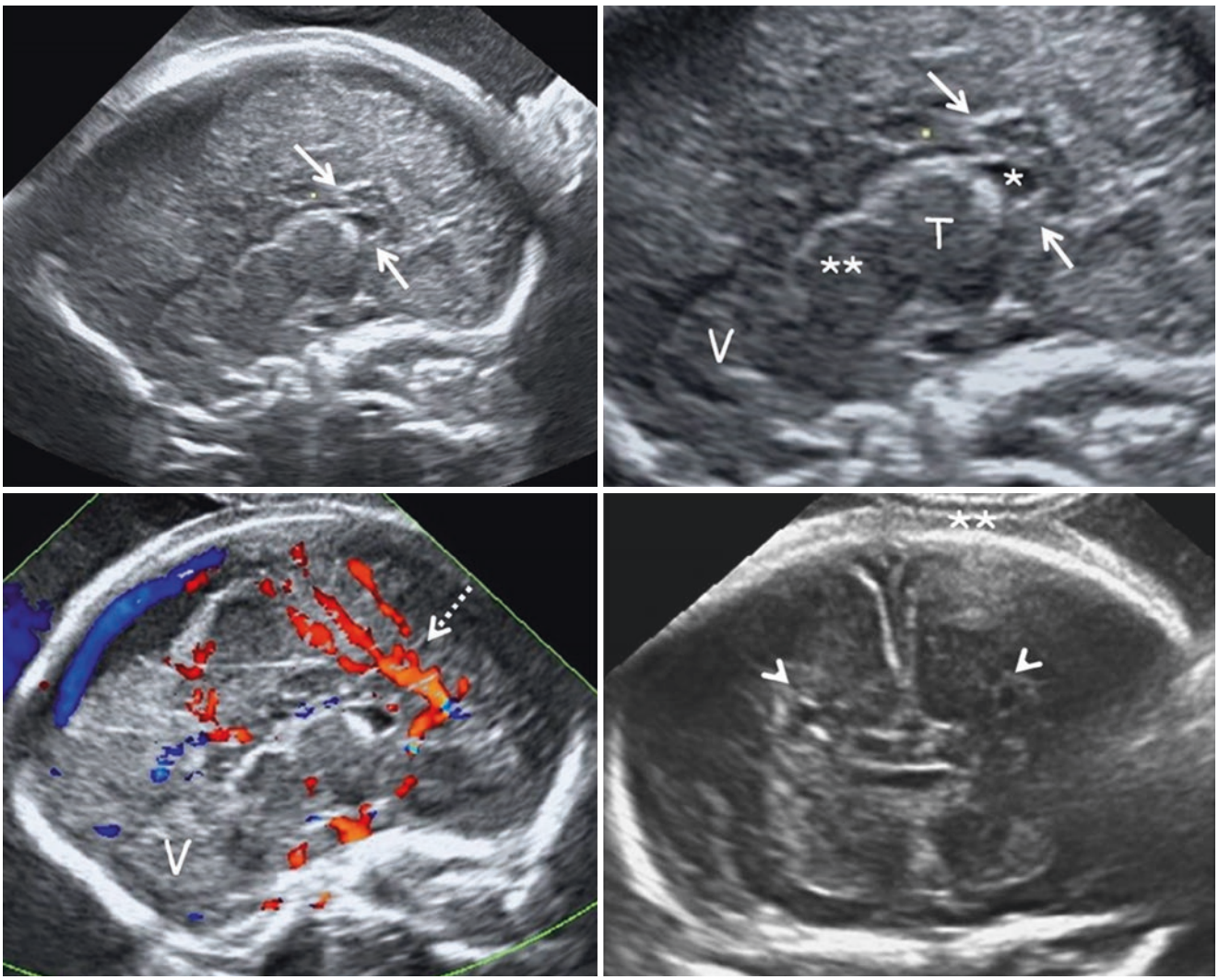

Fig. 2.19 25 weeks (TVS) *partial agenesis of corpus callosum* – midsagittal, midsagittal magnified, midsagittal with color Doppler and coronal transcaudate sections – only the rostrum and genu are present, most of the body and splenium are absent (solid arrows), CC does not extend posteriorly to overlie the quadrigeminal plate (**), CSP is small (*), pericallosal artery sweep stops at posterior limit of CC and takes a radial course (dotted arrow), anterior horns are laterally placed with a hint of steerhorn configuration (arrowheads), thalamus (T), vermis (V). The CSP on axial section was almost absent (US image not included)

Fig. 2.18 (**a**) 23 weeks (TAS) *partial agenesis of corpus callosum* – axial transventricular, axial transventricular magnified section and corresponding line diagram – unilateral mild lateral ventriculomegaly (**) short and narrow CSP (*), asymmetric size and shape of anterior horns (arrowheads), anterior IHF (solid arrow) not collinear with CSP, the genu of CC (dotted arrow). (**b**) 23 weeks (TAS) *partial agenesis of corpus callosum* – midsagittal and midsagittal section magnified – CC is short (CC length of 2.0 cm is less than fifth percentile or −2SD) (double-headed solid arrow), the genu (dotted arrow), body of CC (solid arrow) are present, rostrum and splenium are absent, posterior limit of CC (arrowhead) does not overlie the quadrigeminal plate (**), CSP (*), thalamus (T)

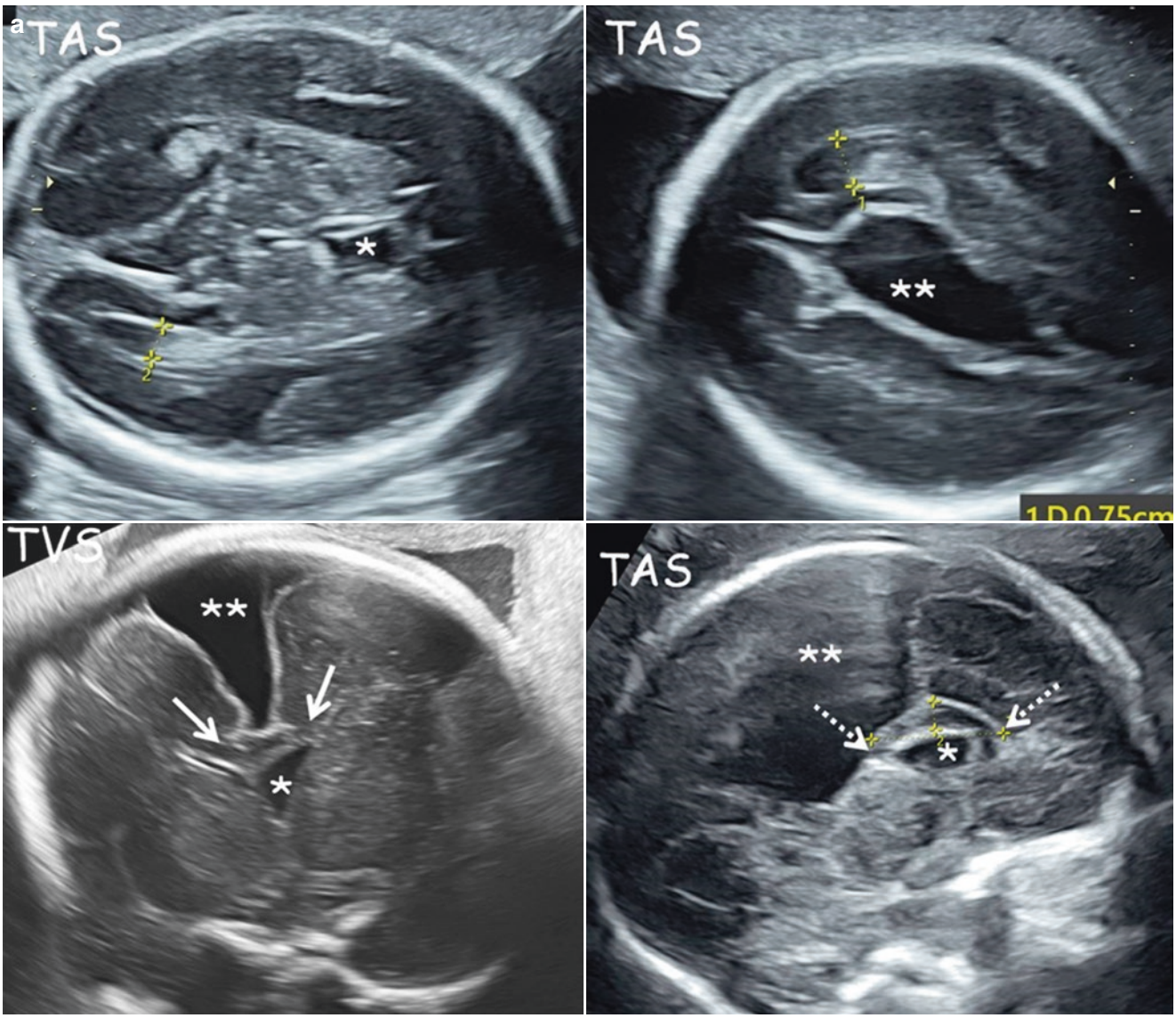

Fig. 2.20 (**a**) 26 weeks (TAS, TVS and MRI) *partial agenesis of corpus callosum with interhemispheric cyst* (*right parafalcine*) – axial transventricular, cephalad to transventricular plane, coronal transcaudate and midsagittal sections – CSP is normal (*), lateral ventricles are normal, interhemispheric cyst to the right of the falx (**), side-to-side asymmetry of CC, right side of the CC is thicker compared to the left side (solid arrows), CC is short, and the splenium is absent (dotted arrows). Differential diagnosis is interhemispheric arachnoid cyst with compression of the splenium. (**b**) 26 weeks (TAS, TVS and MRI) *partial agenesis of corpus callosum with interhemispheric cyst* (*right parafalcine*) – Coronal transthalamic and midsagittal US and T2W MRI sections – interhemispheric cyst (**) is inferiorly communicating (solid arrow) with third ventricle (fluid space between the thalami), CC is short with absent splenium (dotted arrows), thalamus (T). As the cyst is in communication with the third ventricle, it is likely to be an interhemispheric cyst associated with PACC rather than an arachnoid cyst

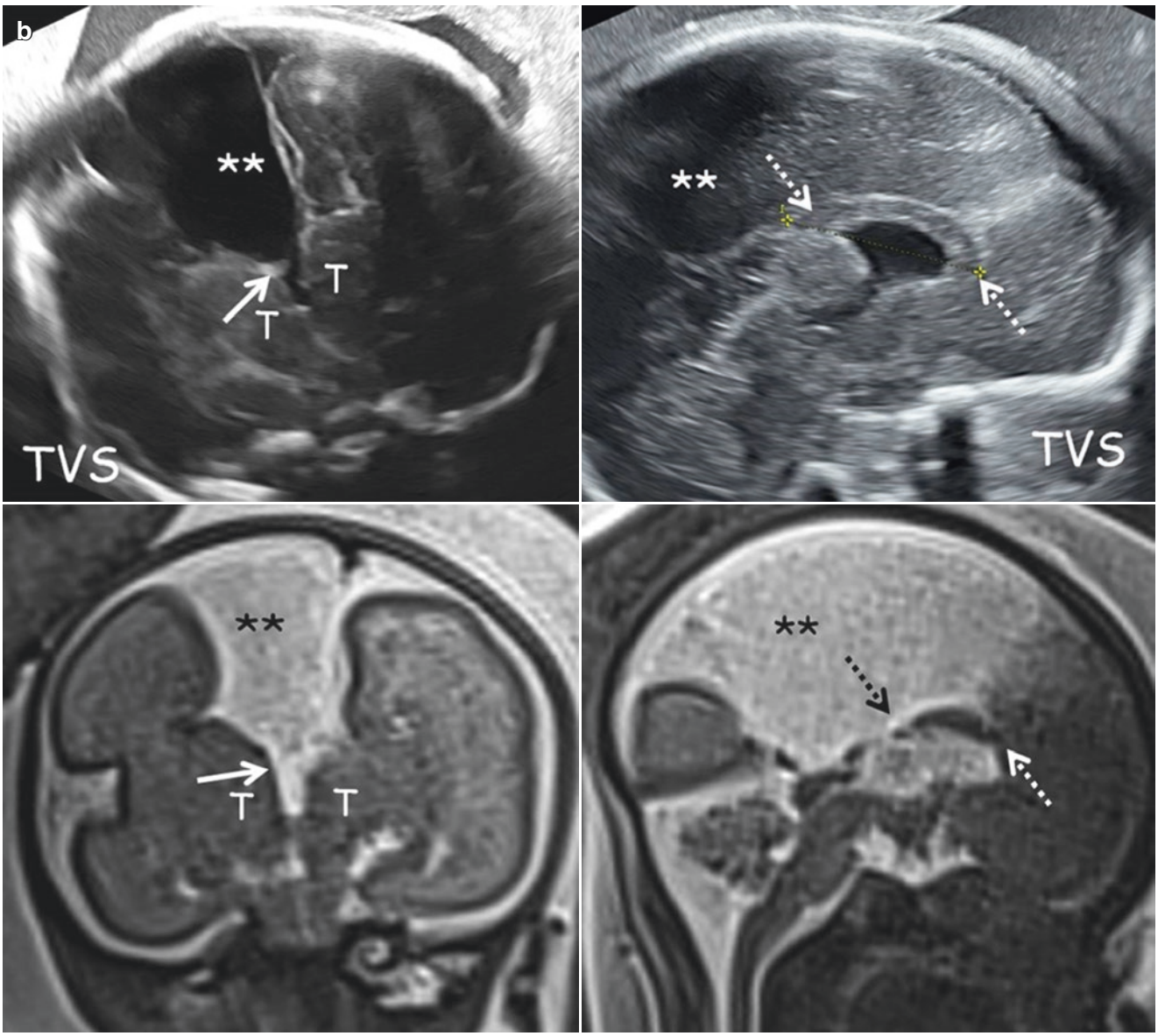

Fig. 2.20 (continued)

2.3 Other Callosal Abnormalities

1. In CC hypoplasia, the entire length of CC is present; however, the thickness is reduced (lesser than −2SD) (Fig. 2.21a, b). This is due to fewer axons crossing from side to side and is associated with other brain anomalies such as lissencephaly. It can also occur due to teratogenic effects of radiation or alcohol or due to compression in hydrocephalus.
2. Subtle changes in the harmonious contour of the CC may be seen in callosal hypoplasia (Fig. 2.21b).
3. The CC is termed abnormally thick CC when the thickness is more than the +2SD (Figs. 2.22 and 2.23a–c). This is always associated with other intracranial findings such as PACC, abnormalities of sulcation, ventriculomegaly, encephalocele, macrocephaly and vermian abnormalities.

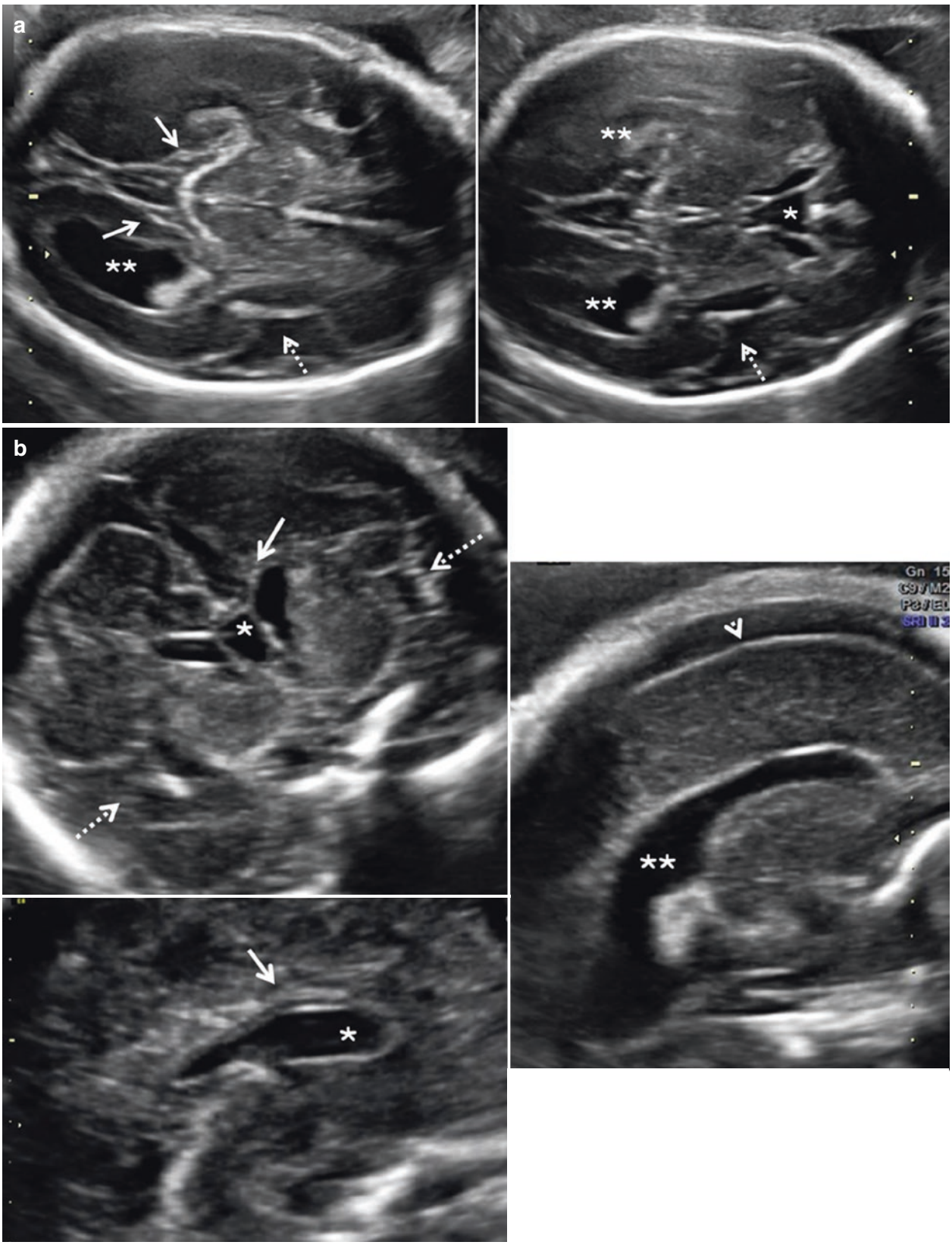

Fig. 2.21 (**a**) 29 weeks (TAS) *corpus callosal hypoplasia and lissencephaly*. Axial transthalamic and transventricular sections – bilateral mild lateral ventriculomegaly (**), normal CSP (*), lateral fissure is "open" without operculation (dotted arrows), shallow parieto-occipital sulcus (solid arrows). (**b**) 29 weeks (TAS) *corpus callosal hypoplasia with lissencephaly* – coronal transcaudate, midsagittal and parasagittal sections – mild lateral ventriculomegaly (**), normal CSP (*), lateral fissure is "open" without operculation (dotted arrows), CC is thin (entire length seen) with midcallosal subtle angulation (solid arrow), convexity sulci are not seen (arrowhead)

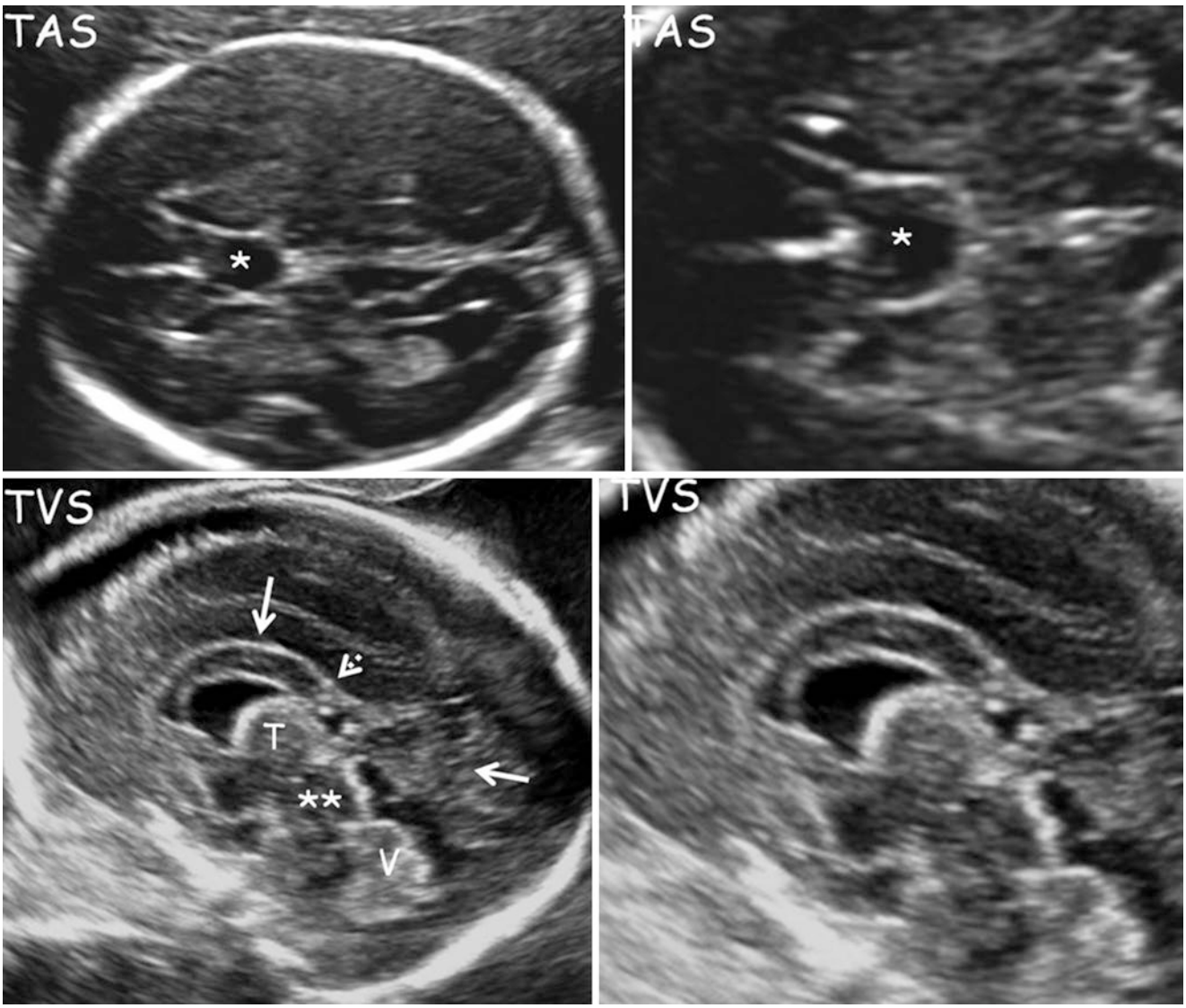

Fig. 2.22 23 weeks (TAS and TVS) *thick corpus callosum with partial agenesis* – axial transventricular, axial magnified (anterior complex), midsagittal and midsagittal magnified sections – CSP is broad and short (*), short CC (solid arrows) does not extend posteriorly (arrowhead) to overlie the quadrigeminal plate (**), rostrum and splenium are absent, thalamus (T), vermis (V)

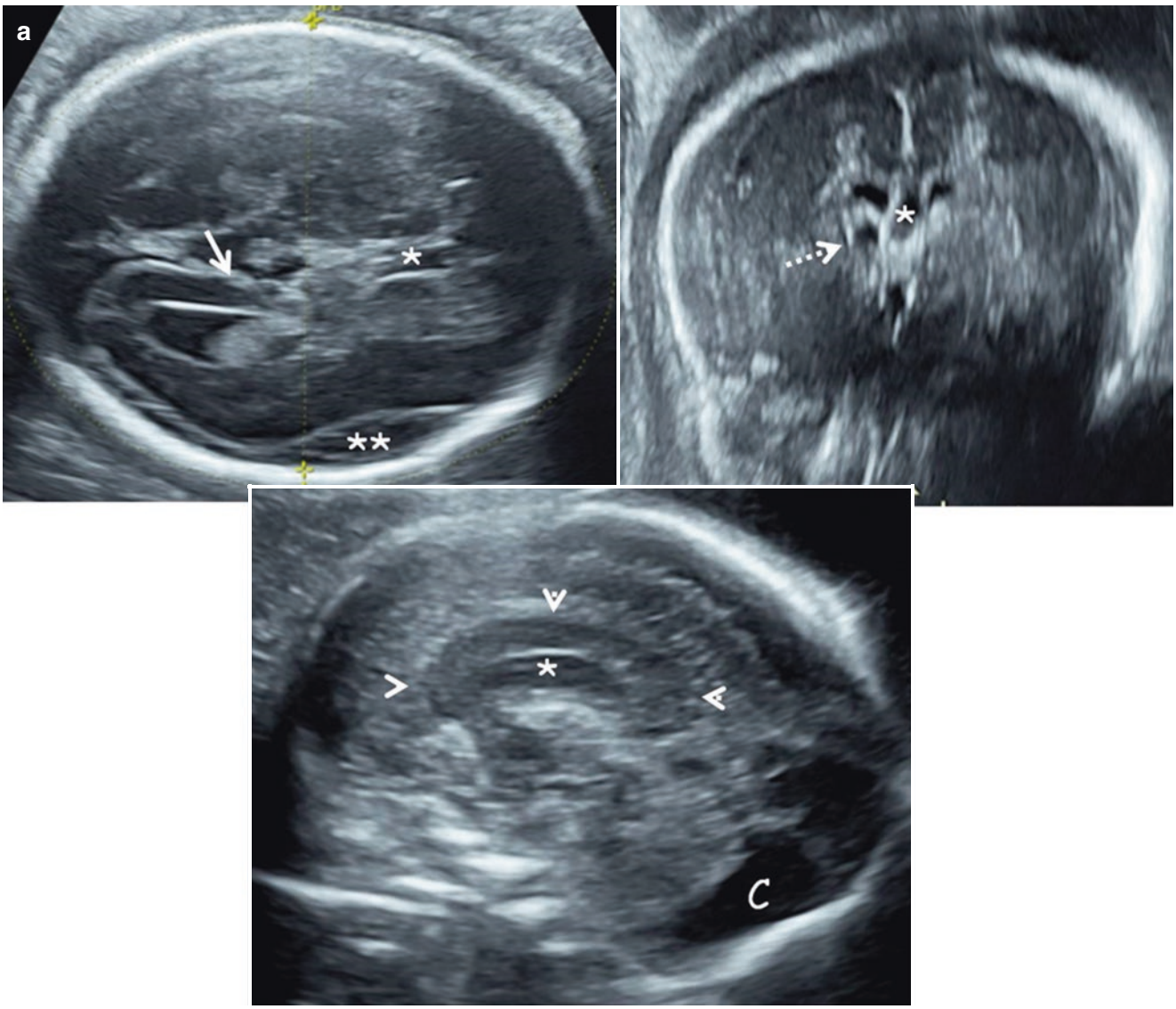

Fig. 2.23 (**a**) 26 weeks (TAS) *thick corpus callosum with lissencephaly* – axial transventricular, coronal transcaudate and midsagittal sections – CSP appears narrow (*), shallow lateral fissure (**), parieto-occipital sulcus not seen (solid arrow), right subependymal cyst (dotted arrow), entire length of CC is present but thick (arrowheads), retrocerebellar cyst (C). (**b**) 26 weeks (TVS) *thick corpus callosum with lissencephaly* – midsagittal, parasagittal, coronal transcaudate and transcerebellar sections – CSP (*), thick CC (solid arrow), retrocerebellar cyst (C), cingulate and calcarine sulci are not seen (dotted arrows). (**c**) 26 weeks (MRI) *thick corpus callosum with lissencephaly* – T2W midsagittal, coronal transcaudate, axial transventricular and transcerebellar and parasagittal sections – thick CC (solid arrows), shallow lateral fissure (arrowheads), subependymal cyst (dotted arrow), retrocerebellar cyst (C)

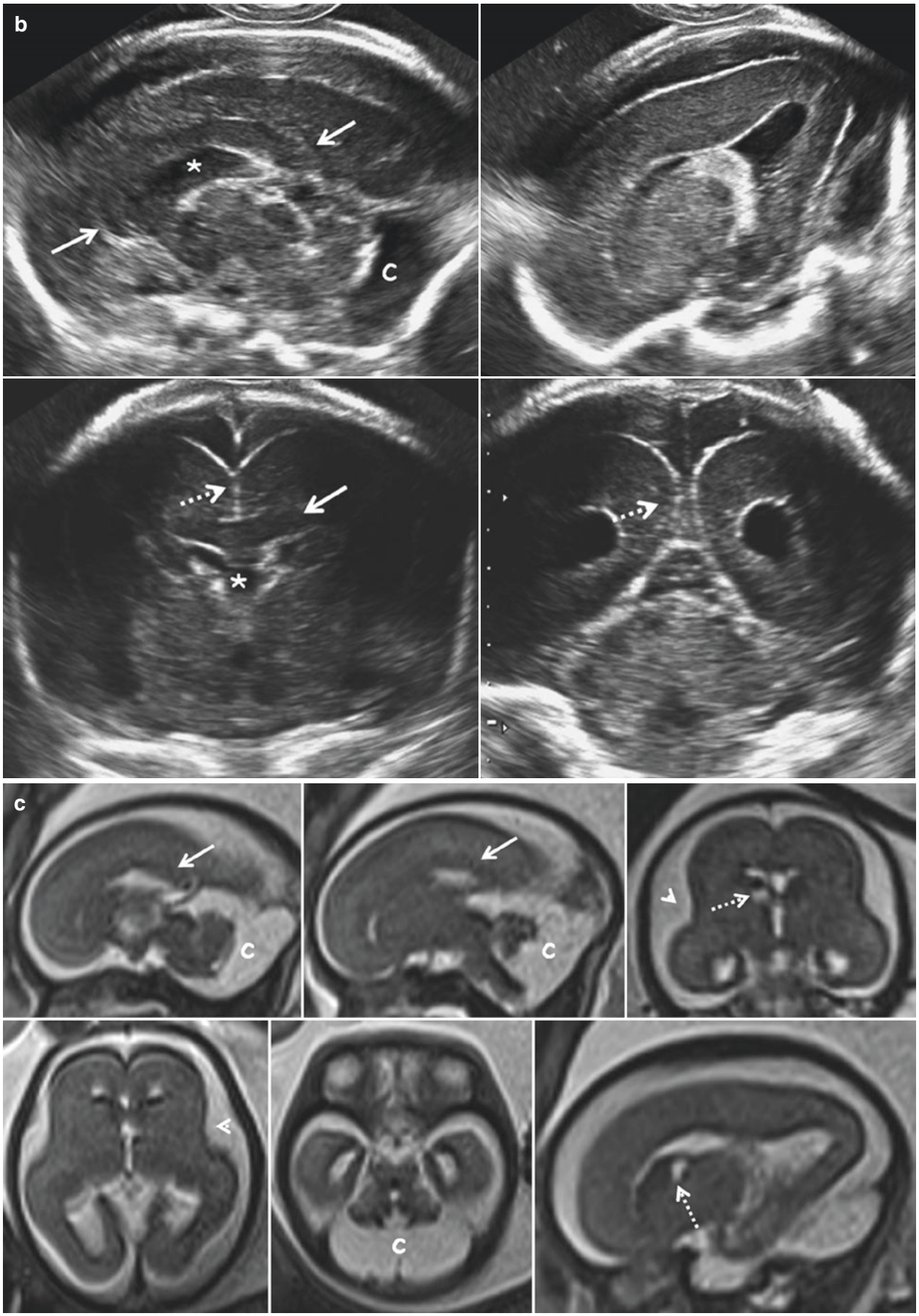

Fig. 2.23 (continued)

2.4 Septal Agenesis

Absence or agenesis of cavum septum pellucidum or septum pellucidum (SP) in the background of normal prosencephalic cleavage is termed septal agenesis. Septal agenesis can either occur as an isolated finding or as a part of septo-optic dysplasia. Septo-optic dysplasia is a grave disorder characterised by septal agenesis with optic chiasma and nerve hypoplasia and hypothalamic-pituitary dysfunction.

1. The frontal horns are continuous across the midline as a result of the absence of CSP or septum pellucidum (Fig. 2.24a, b).
2. The IHF and CC are normal (Fig. 2.24a, b).
3. Mild lateral ventriculomegaly may be a finding.
4. The normal optic nerves and chiasma may be demonstrated directly in the axial or coronal sections of the chiasmatic cistern obtained by high-frequency transabdominal or transvaginal ultrasonography especially in the late second or third trimester. Rendered images of coronal or axial 3D volumes help in the imaging of the optic chiasma (Figs. 2.24c and 2.26b). The presence of a normal-sized optic chiasma may help to rule out or decrease the possibility of septo-optic dysplasia. An optic chiasma that is difficult to be seen may indicate hypoplasia and hence the possibility of septo-optic dysplasia. MRI helps to assess the status of the optic chiasma (Fig. 2.25a, b). Fetal blood sampling for investigation of pituitary function confirms hypopituitarism.
5. Septal agenesis may be associated with malformations of cortical development, schizencephaly, holoprosencephaly, callosal abnormalities or facial clefting (Fig. 2.26a, b).

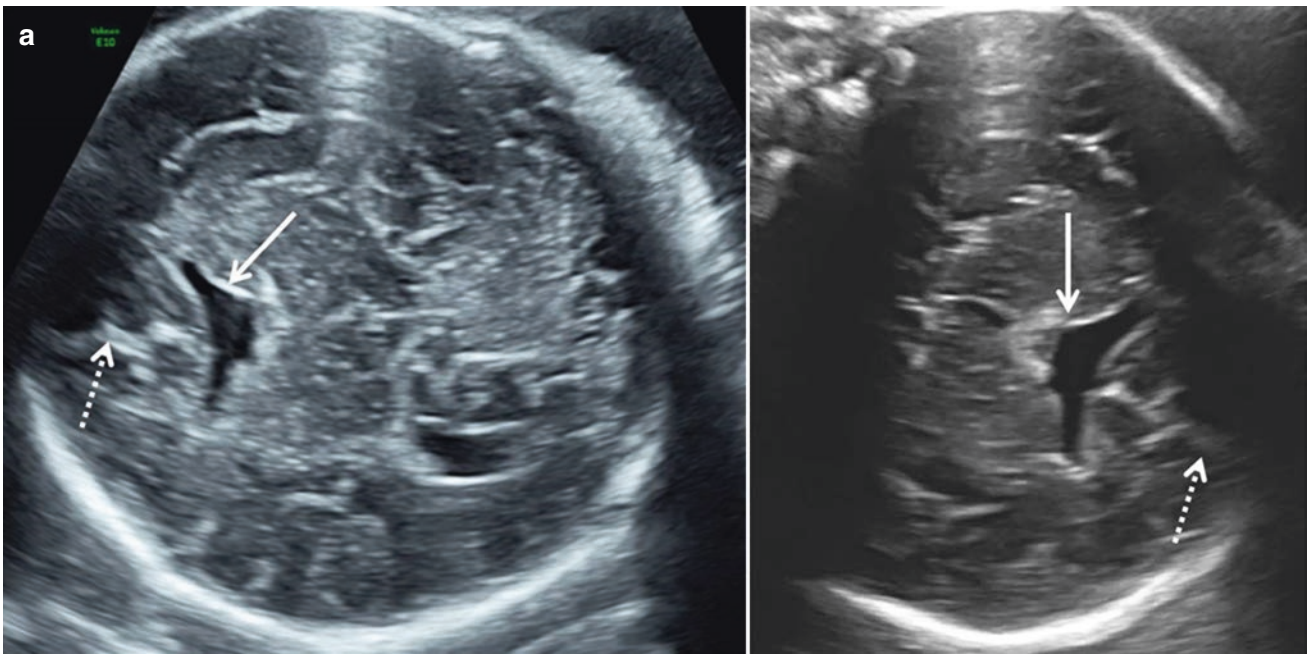

Fig. 2.24 (**a**) 32 weeks (TAS) *isolated septal agenesis* – axial transventricular and coronal transcaudate sections – anterior horns continuous across the midline with absence of CSP (solid arrows), IHF (dotted arrow) is normal. (**b**) 32 weeks (TVS) *isolated septal agenesis* – coronal transcaudate, midsagittal and midsagittal magnified sections – anterior horns continuous with each other across the midline due to absence of CSP (solid arrows), optic chiasma (dotted arrows), CC (arrowheads), vermis (V). (**c**) 32 weeks (TVS) *isolated septal agenesis* – coronal transcaudate (sepia, magnified) – anterior horns continuous with each other across the midline due to absence of CSP (solid arrow), optic chiasma (dotted arrows), CC (arrowheads), temporal lobes (t), head of caudate nucleus (c)

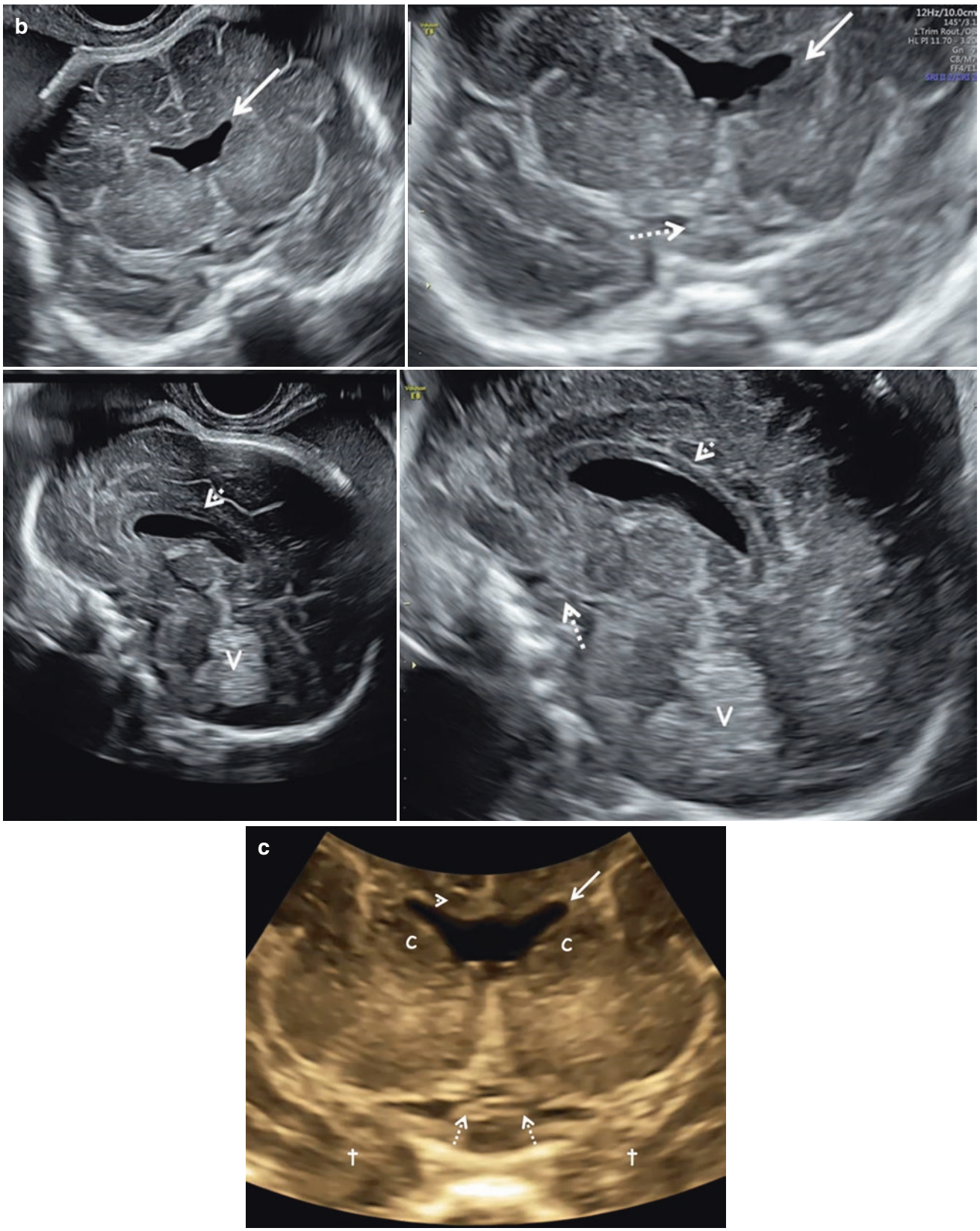

Fig. 2.24 (continued)

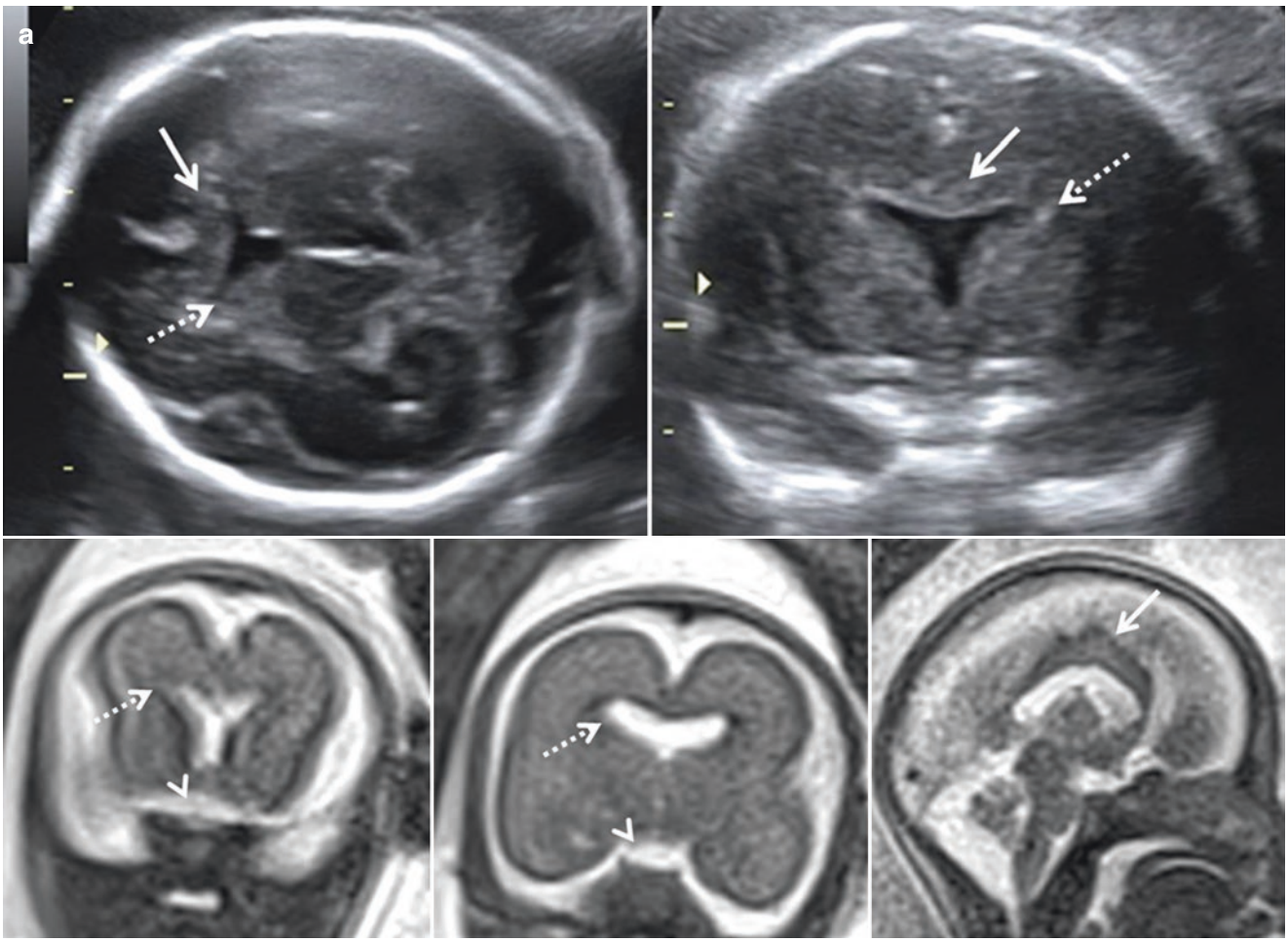

Fig. 2.25 (**a**) 22 weeks (TAS and MRI) *septo-optic dysplasia* – axial transventricular and coronal transthalamic US sections, T2W coronal transcaudate, coronal transthalamic and midsagittal sections – CC (solid arrows), anterior horns continuous across the midline with absence of CSP (dotted arrows), chiasma not seen in the chiasmatic cistern (arrowheads). (**b**) 22 weeks (Autopsy) *septo-optic dysplasia* – basal view of brain, coronal section, the globes and optic nerves – optic tracts (arrowheads) are smaller than the oculomotor nerves (white dotted arrows), anterior horns are continuous with each other across the midline due to absence of CSP (black dotted arrow), optic nerves are thin (black solid arrow)

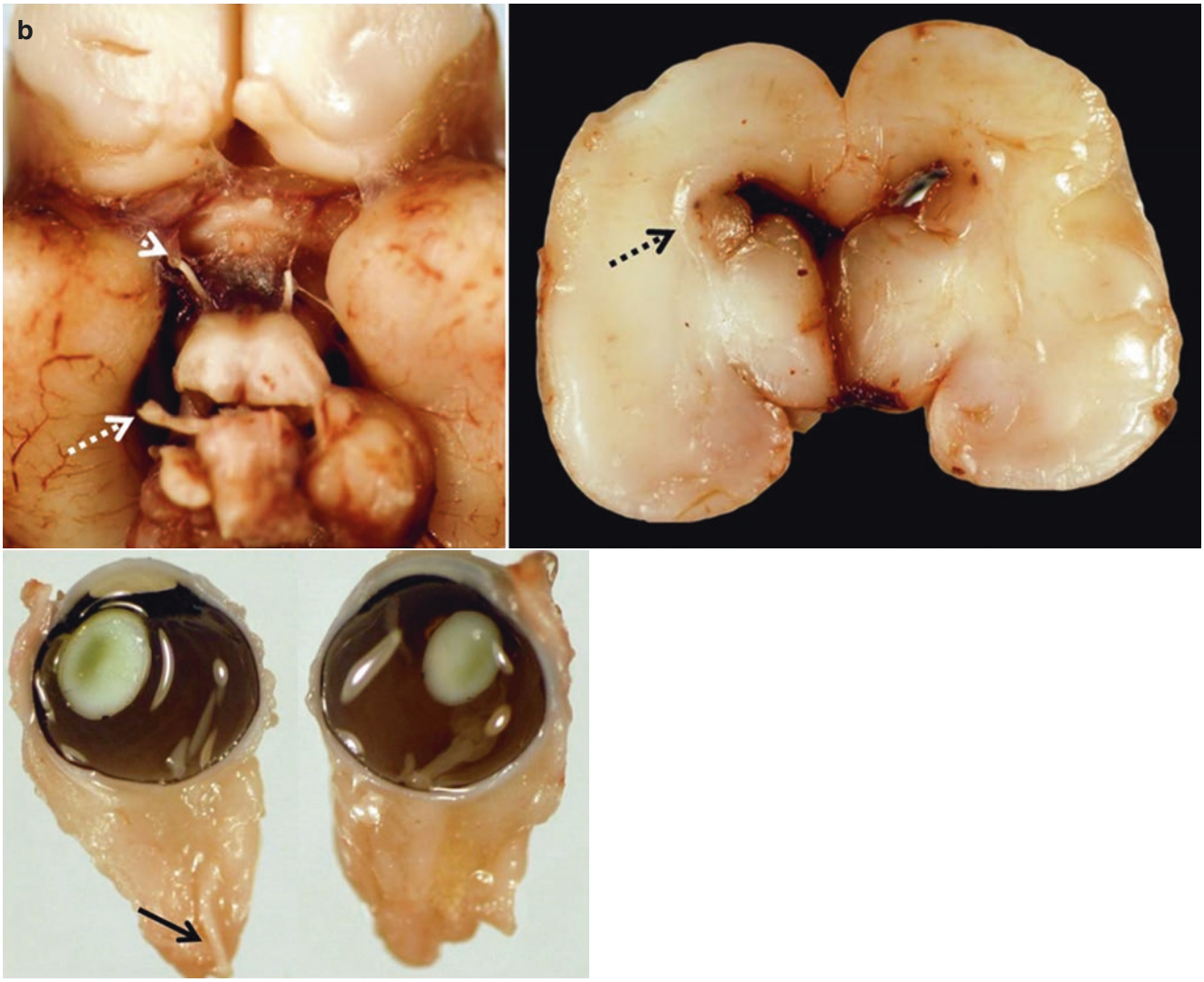

Fig. 2.25 (continued)

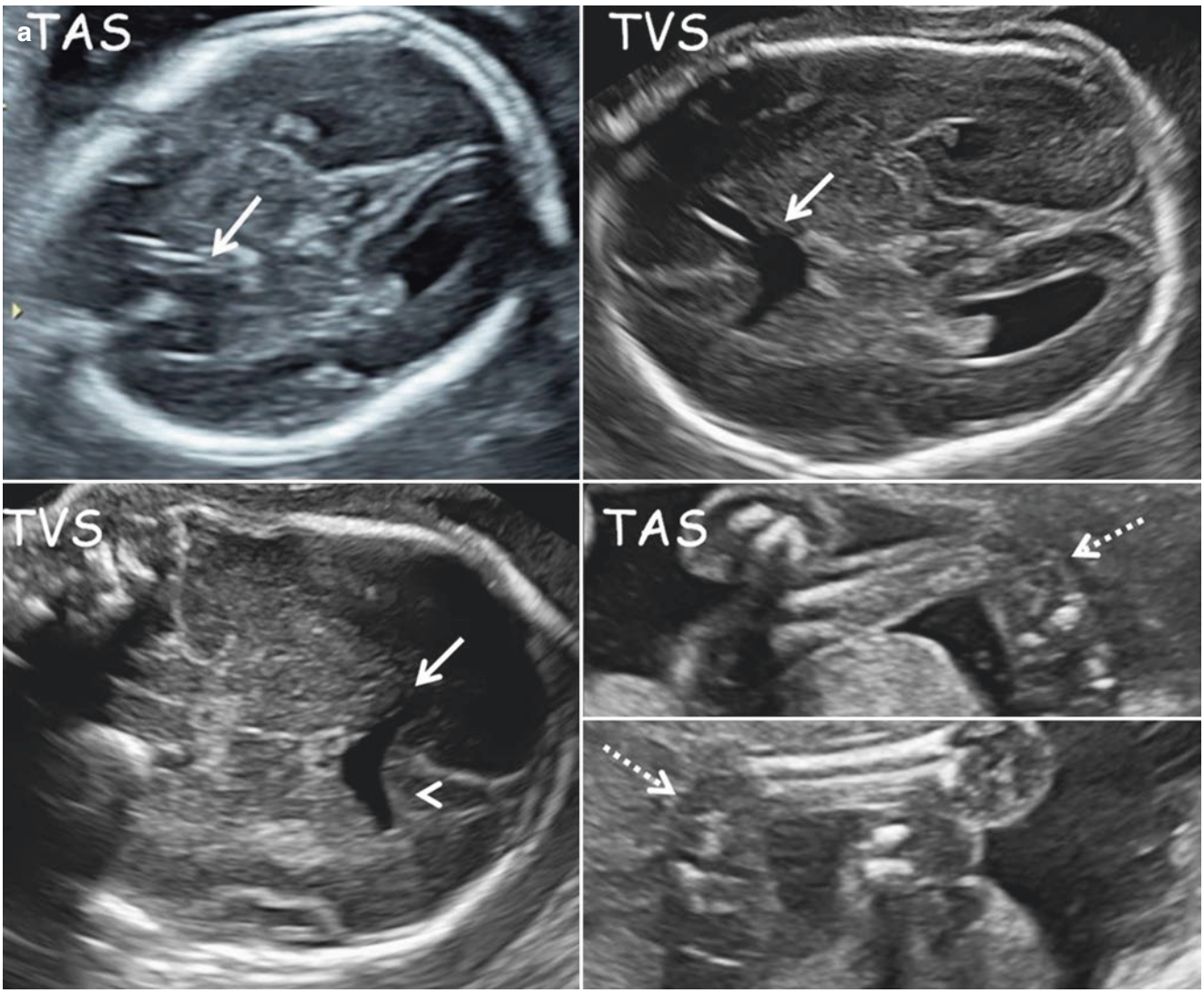

Fig. 2.26 (**a**) 22 weeks (TAS and TVS) *septal agenesis with bilateral congenital talipes equinovarus* – axial transventricular and coronal transcaudate of the cranium and coronal sections of right and left legs – anterior horns continuous with each other across the midline due to absence of CSP (solid arrows), CC (arrowhead), bilateral talipes equinovarus (dotted arrows). (**b**) 22 weeks (TVS) *septal agenesis with bilateral congenital talipes equinovarus* – axial and coronal sections through chiasmatic cistern, 3D axial rendered images of the chiasmatic cistern with edit light option – optic chiasma (H shaped in the 3D rendered mode) (solid arrows) anterior (a), posterior (p)

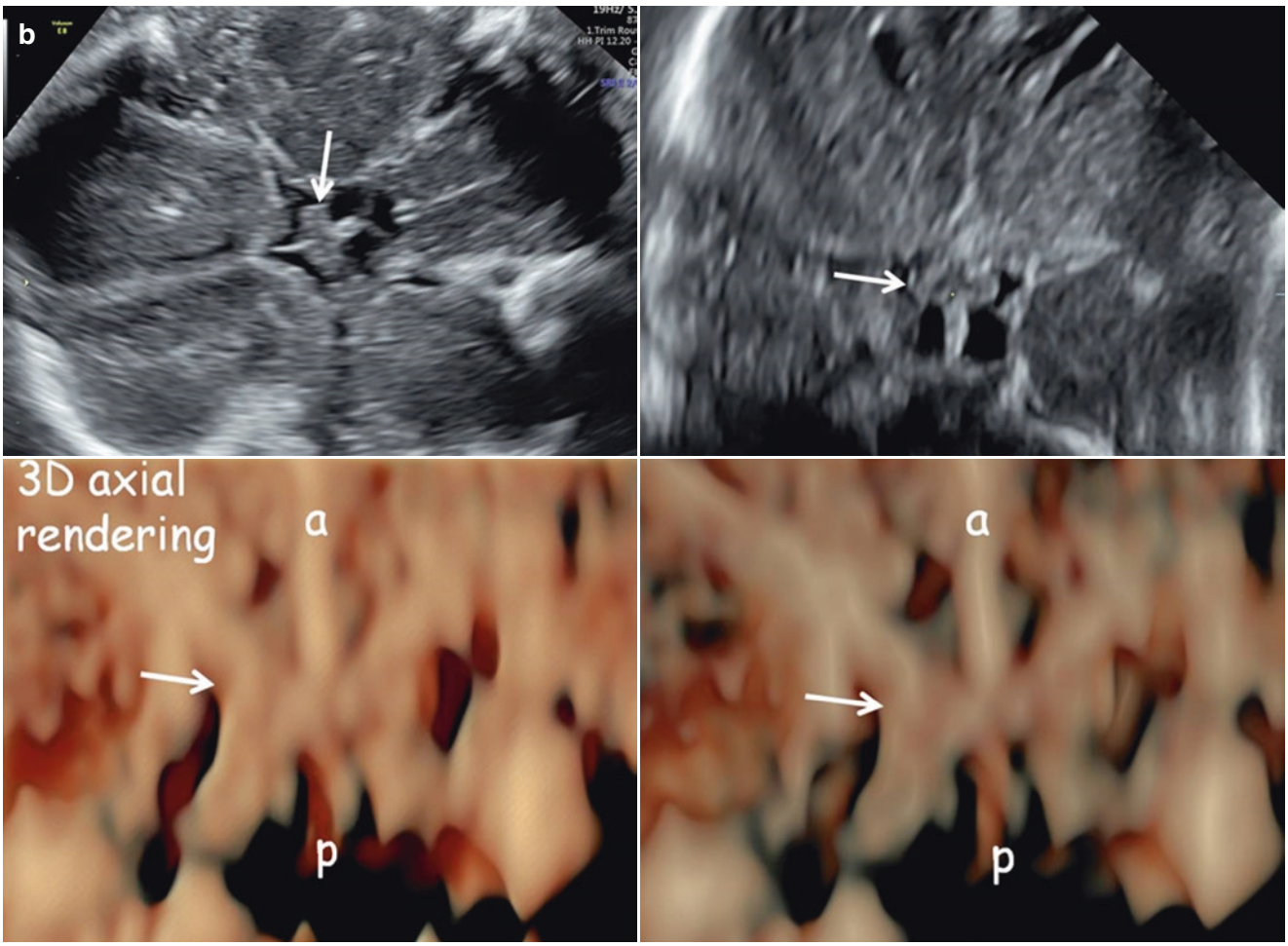

Fig. 2.26 (continued)

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